HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

(98-75)

HEARINGS

BEFORE THE

SUBCOMMITTEE ON
INVESTIGATIONS AND OVERSIGHT
OF THE
COMMITTEE ON
PUBLIC WORKS AND TRANSPORTATION
HOUSE OF REPRESENTATIVES
NINETY-EIGHTH CONGRESS
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HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

WEDNESDAY, JULY 20, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 10:08 a.m., in room 2167, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Also present: Mr. Young of Missouri.

Mr. Levitas. The subcommittee will please come to order.

This morning the Subcommittee on Investigations and Oversight of the Committee on Public Works and Transportation continues with the second phase of its inquiry into the matter of hazardous waste contamination of the Nation's water resources.

As I mentioned, as we started this second phase of our hearing several weeks ago in the aftermath of the confrontation with the executive branch and Environmental Protection Agency over the documents that had been denied this committee, those documents having now been made available and rather substantial changes made in personnel at EPA, we resume with the assistance of the documents that were originally denied an investigation that was contemplated well over a year ago into the program itself.

We view these hearings as partaking of a Janus-like quality, the Roman god of two heads, both looking to the past to see what has happened; but more importantly, looking to the future to see what can be done as we deal with this matter to protect the people, the citizens of this Nation from the impact of and consequences of hazardous waste.

Today's session, and those scheduled for tomorrow and Tuesday and Wednesday of next week, will focus on the health and economic damages experienced by people exposed to toxic chemical waste and problems associated with their efforts to obtain full compensation for those damages, the so-called victim compensation issue.

The issue of victim compensation when reduced to its basic components is also a question of risk assessment: What level of risk do we, as a Nation, believe is appropriate when it comes to exposing human populations to the uncertainties of hazardous waste, or for that matter any toxic chemical? When that level of risk is exceeded, what mechanisms or remedies should be available to compensate those who have been injured as a result of that exposure?

The Superfund law, when enacted in 1980, answered that question so far as it concerned the cleanup of an abandoned hazardous waste site. All that is required at a minimum is, and I quote, a "substantial threat which may present an imminent and substantial danger to the public health and welfare." Similarly, the law provides for assessment of damages against the responsible party, again quoting, "for injury to, destruction of, or loss of natural resources." But the law does not provide for such compensation for injury or damages to individuals, whether such damage is to their health or their economic well-being.

Those who believe that such a provision was necessary did not prevail during the debates that led to the enactment of the Superfund statute and, in large part, this was due to the controversy and uncertainties as to the need for such a provision. Some people have argued and continue to argue there is not enough evidence to justify changes in Federal law. That there is not enough evidence to show with absolute certainty that exposure to hazardous waste cause health or other adverse effects. Yet there would seem to be considerable evidence to the contrary offered by others.

Over the past several years we have seen national health and financial problems faced by residents of Love Canal were not unique, nor were they an aberration. One has simply to look at the problems faced by those exposed to hundreds of tons of DDT in Triana, AL, or those who unknowingly made their homes on the asbestos tailings of Globe, AZ, or the events following the disclosure of high levels of dioxin at Times Beach, MO, a community we will hear more about today.

As my colleague, Congressman Molinari, has frequently pointed out, almost every day when we pick up a newspaper we find the disclosure of another hazardous wastesite that is jeopardizing the health, or potentially jeopardizing the health of the American people. I suppose it is ironic that this morning when I received a copy of the Atlanta Constitution, the morning paper in Atlanta, the banner headline of today's newspaper reads "EPA Seals Site of Atlanta Plant," and the article goes on to discuss the discovery yesterday of a toxic wastesite, an abandoned hazardous wastesite near a school, near a residential area, near a city gymnasium, and two churches.

It is the same story, different names, different places. I think it is doubtful that these hearings we begin today will resolve the questions that surround the uncertainties medical experts and scientists have about the acute and chronic toxicity to human populations. Nor do I believe this hearing will answer all of the questions that arise in attempting to resolve the issue of whether there is a need for change in Federal law to provide for victims of hazardous waste and other pollution.

Such certainty may never exist, but the absence of total certainty does not necessarily mean we must do nothing but wait for a body count before we act. Indeed we look to learn from what we will hear today and in the days ahead and hopefully we will take the steps necessary to ensure that we as a Nation are providing for the protection of the health and well being of the American people in every way that we reasonably can.

I think these hearings today are particularly important because we are going to be hearing from real life human beings who have experienced the impact of hazardous waste. The fact is that too often the general public and, indeed, even the Congress only find out about what it means where a problem of this sort exists through the cold gray print of a newspaper or a 30-second film clip on television. But we are not talking about plastic people. We are talking about human beings and the effect that this problem has had on their lives.

I think it is exceptionally important and especially needful that we in the Congress hear directly from the people who have been victimized by this problem. Not the people who created it, not the people who condoned it, but the people who have been victimized by it, so that we can get a better insight of just what the dimension of this problem is. We are nowhere near seeing the end of the problem, we are really seeing only the tip of the iceberg.

I think we have an unusual responsibility to find out what it is that needs to be done, what the consequences are if we do nothing. That is what we will be looking for today and that is the reason that as chairman of the subcommittee I express my profound appreciation to the individuals who will be sharing with us their story of misfortune.

Before we proceed I would like to inquire whether my colleague, Mr. Molinari, has an opening statement.

Mr. **Molinari**. Thank you, Mr. Chairman.

I do have a prepared statement, but I think a lot of what I was going to say has been covered by you in your own opening statement.

We have had an awful lot of hearings concerning toxic contamination. I suspect, as we proceed this week to hear from those who have been exposed, that indeed we are going to witness firsthand the kind of problems that are encountered throughout this country. I know I have a constituent here today who will be testifying about the problems she and her family and neighbors have had. It is a sad, tragic story.

When we talk about victims compensation, I ask myself the question, if in fact when Congress enacted the Superfund law, if it had provided for victim compensation, if chemical manufacturers were held responsible with open-ended liability for what would result from the chemicals that they manufacture and sell, would the distribution process be what it is today? If the people that distribute them, and those that have licenses to take the toxic chemicals and bring them to legal dumps were held accountable and legally responsible for the health consequences and property damages sustained by the residents of the communities, would they be as lax as they have been in the past?

I suspect not. And I believe when we conclude these hearings we will be in a better position to help determine the steps that we as Members of Congress should take.

I think, Mr. Chairman, with that, we should proceed to our hearing panel. I think this is the time to hear from them rather than myself.

Thank you very much.

[The statement of Mr. Molinari follows:]

Opening Statement of Hon. Guy V. Molinari

Thank you, Mr. Chairman. I join you in welcoming the witnesses who will be testifying before the subcommittee today, particularly Mrs. Rita Jones, a constituent of mine, about whom I will have more to say later when she is called to the witness table.

Mr. Chairman, the so-called issue of "victim compensation"—seeing that persons who experience health or economic injury from their exposure to toxic chemical wastes are adquately and justly paid for the damages they suffer—is a problem of many dimensions viewed in many different ways, depending upon whom you talk with.

In this subcommittee's series of oversight hearings on the Superfund law, we have repeatedly heard from local and State health authorities and EPA officials about the criteria they employ in deciding whether or not to order the closure or cleanup of a particular landfill or waste disposal site. This "risk assessment" process, although often guided by statutory or regulatory thresholds such as "substantial threat to human health and safety," we have found, I believe, to be extremely loose and judgmental in application. The result: Hundreds, if not thousands, of toxic waste disposal sites across the country continue to pose health threats to nearby residents.

Perhaps the real experts we should be listening to are those who are exposed daily to the human health threats associated with drinking water and breathing air contaminated by toxics emanating from waste dump sites. I personally know of such people: Rita Jones is one; other residents of my congressional district are forced to live under the same conditions; the landfill workers for the city of New York, who have recently approached me about their health problems, are others.

What better evidence of threats to human health can be gathered than to talk with and observe persons who suffer chronic respiratory difficulties, higher than normal rates of cancers and birth defects, bladder and kidney disorders, and other medical problems? These are the real experts! They don't need to be told about "threats to human health;" they live with these discomforts and tragedies on a daily basis, while wondering what it takes to convince unresponsive local, State, and Federal Government authorities that there is a problem which needs to be corrected.

I am not unmindful of the medical, scientific, and legal uncertainties involved in this entire issue. The truth of the matter is that there are people out there who are suffering from illnesses of mysterious or unknown origins, who are experiencing real economic hardship and real fears that their current health problems may be but precursors to greater difficulties some time in the future. To them, I suspect, the administration of the Superfund law hasn't been so "super" after all.

One key point which I think the members should keep in mind as we go through this series of hearings: We should remind ourselves of the root cause—toxic chemical contamination—of the victim compensation issue we're addressing. While we do so, consider these alternatives: (1) If we do nothing on victim compensation, leave the legal system alone such that deserving persons face an almost impossible task in recovering monetary damages from toxic chemical generators, have we done anything to encourage chemical manufacturers to clean up their operations by reducing or eliminating their wastes? or, (2) if Congress does enact a victim compensation statute, under which manufacturers could face enormous potential liabilities, have we provided the very type of financial incentive needed to eliminate the root causes of the victim compensation problem?

Our job, aided by the testimony we will hear during these hearings, is to identify the appropriate means by which we can justly and adequately address the victim compensation problem. To that end, I look forward to some extremely productive sessions.

Thank you, Mr. Chairman.

Mr. Levitas. Thank you for your comments.

Dr. Rowland.

Mr. Rowland. No opening statement, Mr. Chairman.

Mr. Levitas. Thank you.

Before proceeding I want to mention two other things. First, as you know, the subcommittee has been continuing its investigation of EPA's handling of two site cleanup efforts, one of which to the

Seymour Recycling site in Seymour, IN, and various actions by EPA officials pertaining to the settlement on that case last fall. I now believe that we will be able to schedule hearings on that part of our inquiry either during or shortly after the August recess. We will make an announcement about that hopefully next week.

Also, with respect to the matter of how the Department of Defense is handling its hazardous waste disposal problems, I believe we may be able to schedule a day of hearings on that either prior to the August break or during the August recess. Hopefully next week we will be able to announce a specific hearing time. There will be one or two other matters added to our agenda as well with respect to some followup on matters that were raised during the first phase of our hearings.

One announcement I want to make to all of the witnesses today. This committee is an investigation and oversight committee. Accordingly, all testimony taken before this subcommittee is taken under oath. I don't want you to think there is any special procedure today in that regard.

And so in the interest of time, if all of the witnesses here today who are scheduled to testify will at this point raise your right hands, I will administer the oath to all of the witnesses at one time.

[Witnesses sworn.]

Mr. LEVITAS. Thank you very much.

We are very pleased to have with us today to introduce the first witness not only a colleague of ours, but a distinguished member of this committee, a subcommittee chairman of this committee, and someone who has been extremely active and interested in this problem for quite some time. Indeed, Congressman Young has on frequent occasions called to this subcommittee's attention specific matters and asked for our assistance and cooperation with particular matters relating to the situation in Missouri as well as the entire problem.

And so it is with that background and with appreciation that I recognize our colleague, Congressman Bob Young, for purposes of introducing the first witness. And upon conclusion of that, I will invite Congressman Young to join us on the panel if he so desires.

Mr. Young.

Mr. YOUNG. Thank you, Mr. Chairman.

I want to take this opportunity to thank you and the other members of the subcommittee for giving me the opportunity to appear here this morning as these very important hearings open. I want to commend you, Mr. Chairman, and the ranking minority member, Mr. Molinari, and the members for your continued oversight and determined work in an area of what has become our Nation's most pressing and immediate environmental concern, the proper management and cleanup of hazardous waste materials.

I appear before you today to introduce a woman, and a public servant, who perhaps better than anyone in the Nation, can provide these hearings with a firsthand impression of the hardship, loss, turmoil and tragedy that toxic waste contamination can bring to a community, and to individual lives. She is Marilyn Leistner, mayor of Times Beach, MO, the small town in my congressional district that has become a modern day ghost town due to the presence of high levels of dioxin and the unusually severe flooding along the Meramec River last December.

Because of the severity and suddenness of the dangerous situation in Times Beach, it has served as a kind of testing ground for EPA responsibility and action under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, also known as Superfund.

Mr. Chairman, after dioxin was discovered throughout the town of Times Beach and the town was virtually destroyed by floods, it was clear to me that some action had to be taken to eliminate the so-called double whammy effect that plagued this community. It had become apparent to me by January 4 of this year that a complete buyout of the town constituted the only way to treat the citizens of Times Beach fairly. The majority of homes and businesses had been demolished from the floods and it made little sense to let these people come back into town and try to rebuild their lives with the threat of dioxin contamination everywhere. In fact, the Center for Disease Control had issued health warnings for the residents not to return to Times Beach for this very reason.

Only after wrestling with redtape, and coordinating all the levels of Government with responsibility for toxic wastes and disaster assistance we were able to bring into effect the offer of the Federal Government to buy out the town and relocate the residents from harm's way. The contracts for the buyout were signed by all parties last month, and the process of appraising the property of Times Beach residents is going on today. Without a doubt, this buyout is absolutely essential as compensation to the innocent victims of the Times Beach disaster, anything less would have been an injustice to the citizens of that troubled community.

Mayor Leistner has seen her friends, her relatives and the people she represents, forced to abandon their homes and businesses. She has worked tirelessly with my staff, EPA, FEMA, the State and local governments in trying to settle on what is proper compensation for the uprooting of these people's lives. Hers has been the monumental task of holding together a community that dioxin has done so much to tear apart. For these reasons, I am sure the subcommittee will benefit greatly from her testimony.

Mr. Chairman, in conclusion, I would respectfully urge the committee to consider how the former residents and businesses are coping in their attempts to rebuild their lives, assess the true economic loss of businesses in Times Beach and whether there is a Federal role in compensation for those businesses, and finally, evaluate the prospects that the victims will ever be fully compensated for their injuries through legal actions against those parties who generate, dispose of, or operate disposal facilities for toxic chemical wastes.

These are only a few of the many issues which your committee must investigate, and I pledge to you I will do all that I can to assist you in your efforts. Mr. Chairman, it is my understanding that the mayor has Mr. Laine Jumper and Mr. Charles Stone here to assist in any questions the committee may have. Mr. Jumper was appointed by the President on the Dioxin Task Force and Mr. Stone is a private businessman.

I want to thank you very much for your attention to this matter. I am sure this subcommittee with its history of trying to get to the bottom of problems will do everything it can to rectify these very bad situations.

Thank you very much, Mr. Chairman. I appreciate the opportunity to come up with the committee.

Mr. LEVITAS. We are pleased to have you with us.

Mr. YOUNG. Mr. Chairman, I don't know, I asked Mr. Jumper and them to put up their hand to be sworn in. Would you want them at the front table with the mayor?

Mr. LEVITAS. I think it would be a good idea.

Madam Mayor, and others from Times Beach, we are pleased to have you with us.

TESTIMONY OF MARILYN LEISTNER, MAYOR, CITY OF TIMES BEACH, MO, ACCOMPANIED BY CHARLES STONE, MELBA PERSCHBACHER, AND LAINE JUMPER

Mrs. LEISTNER. Thank you, Mr. Chairman.

At this time, I would like to take the opportunity to thank Congressman Young for his very dedicated efforts on behalf of the people of Times Beach.

Thank you very much, Congressman Young.

Mr. Chairman, members of the committee and staff, I first want to thank you for the invitation to speak to you about the trials and trauma of Times Beach, MO.

The personal interest that you and our Congress are showing prompts me to speak from my heart.

My name is Marilyn Leistner, mayor of Times Beach, MO, formerly alderwoman, chairman of the board, and acting mayor. I have lived in Times Beach since 1956 and have raised 4 children there. Times Beach is about 1 square mile in size and housed 800 families, about 2,200 people.

It was first subdivided before 1910 into 20 by 100 lots which were given away by the St. Louis Star-Times Newspaper in an advertising program. One free lot with a 3-year subscription to the newspaper. Being located directly on the Meramec River, with its sandy bank, it became known as Times Beach and began being used as a weekend resort and fishing area.

When the depression hit, some people moved into their weekend cottages permanently to ride out the hard times. The town grew slowly and in 1956 it voted to become a village and in 1957 it incorporated as a fourth-class city in order to maintain home rule. Improvement and growth were slow but steady until the recession of the early seventies. Construction of two mobile home parks nearly doubled the population and tight money and credit made the relatively inexpensive land in the Beach quite attractive to young do-it-yourselfers to buy a house or lot and build or fix up themselves. This transformation continued until the late seventies when many new homes were built and many old ones totally rehabbed.

When the dioxin flood disasters hit in December 1982, Times Beach was nearing completion of a metamorphosis from a rundown poor river town to a lower middle class community. Massive cleanup campaigns of abandoned buildings and junk were ongoing and showing success. Progress in the face of economic recession was a source of pride for nearly everyone.

As mayor of Times Beach, I have seen our community transformed from a growing working small class town on a scenic Ozark river to a national symbol of something unclean and uninhabitable. I have heard the talk about Times Beach change from gossip at the park benefit barbecues to nightly jokes by Johnny Carson on the "Tonight Show."

At this time I would like to show you the type of jokes I am referring to. This is the River Front Times weekly newspaper, St. Louis. On the front it says, "Eternal Bliss: Missouri's Most Famous Waste Hauling Family Keeps on Trucking. What do Charles Kuralt, Willie Nelson, and the Grateful Dead all have in common? They are all on the road again."

These are the types of jokes our community has been subjected to since the announcement in November of the dioxin on the streets.

I have felt the pride of accomplishment of a young couple, who had just completed the building of their own home with their own hands, melt into despair and fear as their hard-won dream disappeared into a contaminated ruin. I have shared the joy of an elderly woman who found some small treasure thought lost in the mud and debris and have cried along with a 23-year-old girl terrified of having children for fear her babies will be born deformed. I have seen the very best come out in some people and the very worst in others.

The one true constant seen in all the people, however, is the simple fact that everyone's life has been brutally and irrevocably changed. Gone forever are the friends and neighbors of 5, 20, and 30 years standing. Gone forever are those people who were your babysitters and lawn cutters, your pinochle partners and bowling buddies, your schoolmates and favorite teachers.

When I ran for this office 2½ years ago, if I had known I would wind up in Washington, I would have probably hidden in my closet. You will have to forgive me if I appear a little nervous. I am not really a public person.

Gone is the guy down the street who would trade a tuneup on your truck for a paint job on his porch. Gone is the lady you used to go "garage saling" with. Gone is the local watering hole where you stopped on Wednesday night to have a beer with the people you knew and enjoyed. Gone is the church where you first prayed or said "I do" or simply worshipped on Sunday.

Gone is the cop you called Mike who watched your house while you were down at the lake. Gone are the town meetings where you hollered about potholes in the roads and blamed the aldermen for some things and the mayor for everything. Gone are the smalltown politics, the smalltown problems, and the fierce smalltown pride. Gone is the reverend who read your water meter, and the Indian who graded your yard. Gone forever is everything that made the beach your home, the one place on Earth that gave you the sense of belonging and security and love that you could obtain nowhere else.

The news media has been filled recently with an abundance of shows, articles, specials, and reports about Times Beach and contamination. But the media has yet to communicate what the disasters have actually done to our people, what the reality of our lives truly is, and what the future holds for us.

No one in our immediate area was familiar with dioxin or the possible effects of exposure to this chemical. The fear of the people was all the greater because of their limited knowledge. Children were avoided by their classmates. Mothers from adjoining communities advised their children to avoid contact with Times Beach children. In one instance, an old business associate refused to shake hands with a Times Beach businessman and cited possible dioxin contamination as the reason.

Cleaning establishments refused to accept the clothing of Times Beach residents because of possible contamination and in one very bizarre happening, one man entered a McDonald hamburger restaurant, was recognized from being on television, pointed at, someone said, "He's from Times Beach," and the entire McDonald's Restaurant emptied. Even simple things like trading an automobile was made difficult because the dealers were afraid of dust contamination on the used car since it had been in Times Beach. In innumerable ways, the flood, coupled with the dioxin disaster, has caused untold misery to the people of Times Beach.

Much has been made and said about the Federal buyout of Times Beach. I want to take this opportunity to thank the Federal Government for the aid and comfort extended to us under the various disaster relief programs. I am proud and grateful to live in a country where such aid is a matter of law and course. The immediate Federal flood relief was swift and comprehensive. Emergency shelters were made available and the Red Cross and Salvation Army did a magnificent job in responding to our need for food, clothing, and temporary shelter.

This brings up a most important point about the whole Times Beach situation. If our only problem had been the floods, we would have been 95 percent recovered by now, the end of July. Massive efforts were underway, even before the waters receded to collect debris and organize work teams to help restore our homes. Assistance and volunteers in the best of American tradition came pouring in from surrounding areas.

All these efforts, donations, and work, however, came to an abrupt and incredible halt on December 23 when barricades were placed on roads leading into the town, and the Center for Disease Control in Atlanta issued a strong warning to the effect that if you were in Times Beach, get out; if you were out of Times Beach, stay out.

The State of Missouri subsequently declared the entire town an imminent health hazard. For people who owned or rented homes in Times Beach there were small Federal and State grants for the repurchasing of furniture and other personal items if qualifications were met. Many could not meet the qualifications. Federal and State authorities also made some provisions for temporary housing, in apartments, mobile homes, motels, wherever emergency temporary housing was available.

City employees lost not only their homes, but their livelihood and thus experienced a double hardship. I was not mayor at the time the problems exploded, but was a member of the board of aldermen. The pressures on the Times Beach mayor were so great that two successive mayors resigned for personal reasons.

Hopes for a new Times Beach soared when the Army Corps of Engineers proposed rebuilding the city on unused Federal park ground directly adjacent to the city of Times Beach. This was also the recommendation of the Federal emergency flood hazard mitigation report prepared in response to President Reagan's disaster declaration of December 10, 1982. This proposal is one of the specific alternatives defined in the Superfund law. When defining "remedy" or "remedial action," the term includes the costs of permanent relocation of residents and businesses and community facilities where it is determined that relocation is more cost effective than cleanup.

This was the answer to virtually all of our problems but we quickly learned that this was not to be. Under this program, the present problems of the business people would not have developed.

A plan to purchase only the privately owned real estate was announced by EPA Administrator Anne Burford on February 22, 1983. We, city officials and concerned citizens, began an intensive letter writing and lobbying campaign to convince our State legislature to appropriate the necessary matching funds; which indeed occurred. Terms of the State appropriation bill prevented the State from holding title to the ground; so the city of Times Beach was made a party to the Superfund contract to act as interim titleholder.

Throughout this process, numerous questions were put to FEMA asking about various facets of the buyout; concern was intense as to whether or not people would be able to relocate, whether ruined businesses would be compensated to ever reopen elsewhere.

On March 19, 1983, FEMA called a meeting with the residents, and announced the general terms of the buyout. In question and answer form, FEMA made statements such as: "Businesses are generally eligible for similar acquisition and relocation benefits," and "The intent of the program is to make each resident 'whole' again, as they were on November 30, 1982." We, the city officials, were convinced that our people would receive compensation sufficient for them to start over somewhere else. That the businesses would be made "whole" again as "free of defect or impairment, intact. Whole implies that nothing has been omitted, ignored, abated, or taken away." We were told by FEMA that the purpose of the buyout was to make everyone whole again as they were before the flood and announcement of the contamination.

We are not whole unless the economic injury we have suffered is recognized and compensation to cover that injury is provided.

According to the terms of the relocation brochure distributed businesses are eligible to receive up to a maximum $10,000 relocation expense payment. You are disqualified from even that, if you have a similar business elsewhere. This $10,000 sounds like a lot until you analyze the circumstances of some businesses in Times Beach. I will hit the highlights of some particular instances. Detailed sheets for each are attached.

The owner of a gas station/liquor store known as Jim's Fina—Jim and Leon's Corp.—has invested $50,000 in inventory and purchase of the business. Not owning the real estate, he will receive at best the $10,000, but probably only the minimum $2,500 because he has no inventory left to move. He not only lost his $20,000 year income, but all of his business investment. Ineligible for SBA disaster loans, he has nothing to look forward to except many years of paying off his business loan and probable bankruptcy.

Steve and Marianne Hrin moved their family owned auto body shop business to Times Beach in 1979. Times Beach's prime location on a major interstate seemed ideal. The flood cost them only 3 weeks. The dioxin cost them their business of 29 years. The $10,000 won't even move one frame alignment rack, much less 40 tons of other equipment. The purchase of their real estate will not even begin to compensate them for the loss of a thriving business. Laying off their employees, has resulted in a huge jump in their unemployment taxes and insurance costs. Mrs. Hrin wrote, "We were a classic example of the American dream come true; work hard, invest wisely and save regularly." The failure of the Superfund law to provide business-oriented compensation again has shattered forever another American dream.

The largest business in Times Beach, the two mobile home parks, is owned by Mr. Charles Stone. Included in his business was a used home brokerage firm and a laundromat. With all 337 mobile home pads empty of paying customers, he has to borrow to pay his $17,000 per month interest payments. Active in the buyout negotiations, he repeatedly was assured by FEMA that he would be "made whole again" by midsummer.

The reality of his buyout is that he will be forced deeply into debt to finance the tripled cost of a new park. The 8-month delay in solving the dilemma of Times Beach is causing the accumulated interest to eat up a substantial part of his buyout proceeds and working capital. Without compensation, Mr. Stone will lose most of his equity and will basically be starting a $4 million business in a very uncertain economic climate and in a much less desirable location.

J.C. and Doris Walters, owners of KOA Kampground, face virtually unsurmountable relocation odds. They have lost the 1983 season and will lose the 1984 season because it is already too late to advertise in the 1984 nationwide directories that bring them 80 percent of their business. Their new campground is not yet under construction as they are awaiting the buyout funds which will enable them to start construction of their new campground. Being the closest to St. Louis and near the Six Flags Amusement Park on I-44 was a prime location. This campground was located approximately 2 miles from Six Flags Over MidAmerica.

There is simply no good ground available with the perfect highway access that KOA had. The Walters are ineligible for the $10,000 relocation allowance because they own a small campground elsewhere.

I could go on about Robert Fitts, who had nine rental mobile homes. These have turned into nine overdue payments each month. Or, Melba Perschbacher, who has two commercial properties with 10-year leases. She no longer has her assured retirement income but she still has the burdensome debt of her mortgage payments.

I could describe 30 more owner-operated concerns who face the loss of everything without help. How many of these will ever be able to reopen? How much unemployment will result? Additional hardship cases are attached.

However large the financial toll has proven to be, the human toll remains. The fact is that we have been the rope in this tug of war. So much controversy has surrounded dioxin, as one doctor has said, that dioxin is the most studied chemical today and still no serious harmful effects are known. However, the Missouri Department of Natural Resources in a letter to Alden Shields, dated August 17, 1982, states "Dioxin acts as a toxin, carcinogen, mutagen, and fetal toxin * * *".

At this time I would like to quote a manual I have used very much since the day it was sent to me. On page 223 of the EPA Manual, it says several deaths related to the TCDD have been recorded. Some due to liver damage and others to chronic exposure to the chemical.

Additionally symptoms such as chloracne can be passed by an exposed person to close associates, such as family members through holding hands or other close contact.

The concern of serious health effects is a real one for the people of Times Beach, but it is more in the nature of a long range concern for most of us. Others are not so fortunate.

At an intersection of "hot streets" lived four families. Mr. and Mrs. Lewis Bierman moved to Times Beach in 1975. In February 1978, Linda suffered a miscarriage. In March 1979 she gave birth to Melissa, who had congenital neuroblastoma, a rare cancer in infants. Melissa died 4 months later. After having been assured by doctors that having another child was a one in a 300,000 chance, Linda bore Jessica in December 1980.

Tragically, Jessica, too, has neuroblastoma cancer and undergoes chemotherapy and radiation treatments every 5 weeks. Her prognosis is very poor. Their tremendous financial burden is made much worse by the cutbacks in insurance coverage by Mr. Bierman's employer. By the way, he is a Federal employee and in 1979 his insurance was cut back by 15 percent. Future health insurance may become unobtainable as companies exclude people who have lived in known contaminated areas.

There are other people I have here who have had health problems. They do not want their names to be used because they do not want to be harassed by the media. And in Times Beach, MO, you had all the press, TV stations and media practically living with you since December 5 of last year.

Across the street lived Mr. and Mrs. "A". After living there about 2 years, Mrs. "A" died mysteriously in childbirth from a massive infection that overwhelmed a depressed immune system. Three years later the baby died with a similar disorder.

Next door to Mr. "A" lived Mr. and Mrs. Laine Jumper. A baby born in 1971 died 10 months later from a heart defect. Mr. Jumper suffered an acute neuroestenia in 1978 resulting in 8 weeks hospitalization and 6 months lost work. Consulted neurologists couldn't explain the depressed nerve functions and personality problems but did say that it could be environmentally related. Somewhat recovered, Mr. Jumper has never returned to the physical condition necessary to his business and medical insurance is unaffordably expensive.

Mr. Chairman, at this time I would like to tell a story. I have not been a public person about the health of my children, but since 1977 three of my four children have developed severe health problems. I have a 19-year-old daughter who in 1977 developed a very serious seizure disorder. In March of this year she attempted suicide because of the fact that she felt with all the publicity, she had no future, and she felt that the dioxin was very harmful and that she wasn't going to be able to have children or lead a normal life.

You will have to forgive me for getting emotional. I am sorry.

Another daughter, Tammy, 23 years old, has been treated by a doctor for over a year. Three years ago we almost lost her with a hyperthyroid disorder. She also has a hypoglycemia disorder. But she has been treated for the last year for what the doctors are terming precancerous stages of the cervix.

My 24-year-old daughter has a very serious disorder, some type of allergy, possibly environmentally related. Her nose, her lips, her eyes swell shut. Her lip swells with her nose. She has been seeing a dermatologist once and twice a month for almost 2 years.

The Bodimer family moved to Times Beach in early 1972. In 1972 their street was heavily sprayed with the contaminated oil. The father started to have heart attacks and problems with diabetes in 1979. By the way, this man, since the December 5 flood is a complete invalid. He even refuses to get out of bed. He feels there is no reason for him to live any more. Both the twin boys in the family after going through the screen held by the Missouri Division of Health Center for Disease Control in Kirkwood, MO, were advised to see a dermatologist. Both teenage boys have now been diagnosed as having chloracne.

The mother, Shirley, has been seeing a dermatologist for the past 3 years. She has a recurring skin rash which cannot be diagnosed as to what is causing it or what it is.

Across town is another neighborhood where seizure disorders are the rule rather than the exception. In a grouping of 15 homes, there are seven families with one or more members suffering a seizure disorder. Directly across the street a small girl 5 years has a serious nose bleed. She wakes up in the morning and her pillow is just covered with blood. This small child also has a seizure disorder. A year ago the mother developed a seizure disorder. They are both on dilantin and phenobarbitol. Behind them a family lived. They had a baby, 18 months old died December 17 from a hydroencephalis, which is water on the brain. These represent only those who were willing to talk in this area.

Two doors down the street from me on the right, a year and a half ago a baby was born with the kidneys on the outside of the body. Perhaps there are others who exist but we have talked about health problems with a lot of people who are unwilling even to discuss their problems.

Telephone repair men assigned to Times Beach 10 years developed severe rashes on their hands and feet that subsided only after transferring to other areas. One of these telephone men I had the opportunity to carry on a conversation with in the Kroger store at Eureka, MO. The rash is very bad on his arms. He hasn't been able to find anything to treat the rash. I referred him to St. Joseph's Hospital for the health screening.

There have been numerous young women with breast cancer; some in their teens. A city employee had a daughter develop seizures after moving into Times Beach. One incredible irony involves a former mayor who received a medical retirement after 14 years in the Marine Corps. He had been sprayed in Vietnam with agent orange and developed chronic bone marrow disease, resultant hemophilis, and severe chest pains. His discharge lists an unknown toxin agent as the cause—Dioxin in the agent orange?—how sad to note that he moved to dioxin contaminated Times Beach where his daughter developed severely swollen salivary glands and 2 weeks ago had surgery.

While the human temptation exists to blame anything and everything on dioxin, I have related only problems that have defied diagnosis as to their cause from all facets of the medical community. I have ignored many other normal type problems existing in these people. I truly believe that many severe health problems in Times Beach will be linked to the dioxin; yet, there is no program of compensation for health benefits or insurance or liability clauses for same in the Superfund law as it stands today. There are no provisions for people oriented compensation from environmental diseases. The Superfund law requires polluters to reimburse the fund for cleanup costs, but not for the human costs. Polluters should stand liable under this law for damages to people without the delaying and evasive and costly necessity of civil lawsuits.

There is no compensation for the anguish, stress, and resultant mental health problems suffered by our people. At least four fatal stress related heart attacks have occurred since January. Are they victims of bad hearts or pressure from all of this? At least six complete nervous breakdowns and innumerable near collapses were definitely caused by the problems.

Other uncompensated costs involved the transportation of children to school; an additional 10, 15, and in some instances 80 miles daily while living in temporary housing awaiting the buyout money. We went to the Rockwood School District and asked that the children be allowed to stay in the school district. They had already lost their homes, their community. We felt it would be very tragic for them to be moved from the school district where they had been going to another school district. In September of this year they will have to start in schools in the areas where they are now living.

Most families will be unable to permanently relocate in time to get their children registered for school. If they do get them registered they may have to move again possibly in October, November, or December. There is a shortage of affordable housing in the area, thus forcing many to move farther from work, which raises transportation costs.

What of the future? When the floods came I heard many who had lost everything say, "Well, at least we still have our health." Now, who is to know what our health will be? Lifetime followup of people exposed to dangerous chemicals should be mandatory in order that the toxic timebombs of cancer and pollutant induced disease will be attributed to the proper cause. A fund should be set aside from the Superfund tax on chemicals to provide necessary health care for affected persons. Fund subsidized life and health insurance is another alternative.

There is no provision for compensation for personal property. I appeared on cable news network with Dr. Carnou. His advice to the people of Times Beach was, move out if you are still living there. If you are planning on moving back, don't go back, and don't take absolutely anything with you.

Many people at that time destroyed everything that they tried to wash or clean up from their homes, thus leaving them with no bedsheets, no clothing, nothing. When the CDC issued it's health warning on December 23, 1982, it advised us to cease all nonemergency cleanup and leave everything undisturbed because of the possibility of contamination. Most of us heeded that warning and did not attempt cleanup of clothes, furniture, tools, and other personal property from the flood. A lot of people never returned to their homes from the December 5 flood.

It wasn't until February 23, 1983, that the EPA concluded that we could go ahead and remove our things, with proper cleaning. What vandals and thieves didn't destroy, 2½ months of rotting in mud and debris did. The buyout will not compensate anyone for any losses of this nature. Small Federal grants to us of $5,000 only go a small way to replace a lifetime's accumulation and most did not even qualify for the grant. Businesses were told that SBA loans were available. Almost all applied and almost all were refused. There was no ready money for the business people and they have suffered accordingly.

Trying to summarize the agonizing death of our city is difficult. This experience has been an emotional roller coaster ride for 2,000 people. Peaks of hope one day to be followed by a plunge into despair the next.

We suffered the worst flood in our recorded history on December 5, 1982. Were that the only problem we would have prevailed without Federal aid, but this monster called dioxin has removed any clear cut goals from our lives and left nothing but questions, doubt, and an underlying knowledge that things will never be the same again. Should there ever be another Times Beach in another location, at some future date we would suggest the following:

For business people:

First, adequate replacement value and relocation.

Second, where a business cannot be relocated fairly then just compensation for the value of the business enterprise.

Third, no resident or business can relocate without knowing: The amount of money available for rebuilding; and a definite date the money will be available.

Fourth, equal grants for business people who have lost income as for residents who have lost shelter.

Fifth, consideration for the ongoing expenses of the business community, such as mortgages, insurance, inventory, debt, et cetera.

Sixth, speed. The need to make the above needed information available as rapidly as possible cannot be overemphasized.

For the entire community:

First, compensation for personal property declared contaminated.

Second, compensation from polluters for health problems with strict liability clauses.

Third, lifetime followup of exposed persons.

Fourth, detailed Federal guidelines to provide consistent, equitable treatment for residences, businesses, and local government.

Fifth, mandatory uniform State Superfund laws to guarantee State government cooperation as well as necessary matching funds.

Sixth, implementation of extraordinary remedies where such measures are warranted as opposed to taking the cheapest way out.

These are our recommendations to you. It has come to our attention that Senators Danforth and Eagleton have sponsored legislation which will correct some of these inequities. However, it appears this legislation will not be passed in time for it to help the people of Times Beach. Our people need to come under the provisions you are making for others. We ask that you please amend your bill to include us.

The laws need to be changed. Provisions for extraordinary response, such as relocating entire communities as described in section 101, PPH 24, should be implemented where it is warranted, uniform mandatory State Superfund laws should be required nationwide, to guarantee necessary State matching funds and a swift response from State officials.

Victim compensation must be included for residents and businesses that suffer financial and health losses through no fault of their own. Federal response time must be shortened. Some of these cannot be applied to us; in other areas by amending the Superfund law and making it retroactive to include Times Beach, some of our serious problems can be alleviated.

I have presented here the unexaggerated truth. I feel that the intent of Congress in passing the Superfund law was not in the areas of response to and compensation in the Federal effort at Times Beach. I ask that you, the committee, consider these facts and make appropriate recommendations to the full Congress. I thank you sincerely for myself and the people of Times Beach for your interest. In calling home last night I have learned that in the State of Missouri there are now 165 potential sites, 31 confirmed at this time. The possibility does exist now that anyone who permanently relocates from Times Beach may possibly move to another contaminated site.

Thank you very much and God bless you all.

[Attachments to the Leistner statement follow:]

STATEMENT OF MARILYN LEISTNER, MAYOR OF TIMES BEACH, MO

Mr. Chairman, Members of the Committee, and Staff: I, first, want to thank you for the invitation to speak to you about the trials and trauma of Times Beach, Missouri.

The personal interest that you, our Congress, are showing prompts me to speak from my heart. My name is Marilyn Leistner, Mayor of Times Beach, Missouri; formerly Alderwoman, Chairman of the Board, and Acting Mayor. I have lived in Times Beach since 1956 and have raised four children there. Times Beach is about one square mile in size and housed 800 families, about 2,200 people. It was first subdivided before 1910 in 20′ x 100′ lots which were given away by the St. Louis Star-Times Newspaper in an advertising program. One free lot with a three year subscription to the newspaper. Being located directly on the Meramec River, with its sandy bank, it became known as Times Beach and began being used as a weekend resort and fishing area. When the depression hit some people moved into their "weekend" cottages permanently to ride out the hard times. The town grew slowly and in 1956 it voted to become a "village" and in 1957 it incorporated as a fourth class city in order to maintain "home rule." Improvement and growth were slow but steady until the recession of the early seventies. Construction of two mobile home parks nearly doubled the population and tight money and credit made the relatively inexpensive land in the Beach quite attractive to young "do-it-yourselfers" to buy a house or lot and build or "fix-up" themselves. This transformation continued until the late seventies when many new homes were built and many old ones totally "rehabbed". When the dioxin-flood disasters hit in December 1982 Times Beach was nearing completion of a metamorphosis from a run-down poor river town to a lower middle class community. Massive clean up campaigns of abandoned buildings and junk were on going and showing success. Progress in the face of economic recession was a source of pride for nearly everyone.

As Mayor of Times Beach, I have seen our community transformed from a growing working class small town on a scenic Ozark river to a national symbol of something unclean and uninhabitable. I have heard the talk about Times Beach change from gossip at the park benefit barbecues to nightly jokes by Johnny Carson on the Tonight Show. I have felt the pride of accomplishment of a young couple, who had just completed the building of their own home with their own hands, melt into despair and fear as their hard won dream disappeared into a contaminated ruin. I have shared the joy of an elderly woman who found some small treasure thought lost the mud and debris and have cried along with a 23 year old girl terrified of having children for fear her babies will be born deformed. I have seen the very best come out in some people and the very worst in others. The one true constant seen in all the people, however, is the simple fact that everyone's life has been brutally and irrevocably changed. Gone forever are the friends and neighbors of 5, 20, and 30 years standing. Gone forever are those people who were your babysitters and lawncutters, your pinochle partners and bowling buddies, your schoolmates and favorite teachers. Gone is the guy down the street who would trade a tune-up on your truck for a paint job on his porch. Gone is the lady you used to go "garage sale-ing" with. Gone is the local watering hole where you stopped on Wednesday night to have a beer with the people you knew and enjoyed. Gone is the church where you first prayed or said "I do" or simply worshiped on Sunday. Gone is the cop you called "Mike" who watched your house while you were down at the lake. Gone are the town meetings where you hollered about potholes in the roads and blamed the aldermen for some things and the mayor for everything. Gone are the small town politics, the small town problems and the fierce small town pride. Gone is the Reverend who read your water meter, and the Indian who graded your yard. Gone forever is everything that made "The Beach" your home—the one place on earth that gave you the sense of belonging and security and love that you could obtain nowhere else.

The news media has been filled recently with an abundance of shows, articles, specials and reports about Times Beach and contamination. But the media has yet to communicate what the disasters have actually done to our people, what the reality of our lives truly is and what the future holds for us.

No one in our immediate area was familiar with dioxin or the possible effects of exposure to this chemical. The fear of the people was all the greater because of their limited knowledge. Children were avoided by their classmates. Mothers from adjoining communities advised their children to avoid contact with Times Beach children. In one instance an old business associate refused to shake hands with a Times Beach businessman and cited possible dioxin contamination as the reason. Cleaning establishments refused to accept the clothing of Times Beach residents because of possible contamination and in one very bizarre happening one man entered a McDonald hamburger restaurant, was recognized from being on television, pointed at; someone said "He's from Times Beach" and the entire McDonald's Restaurant emptied. Even simple things like trading an automobile was made difficult because the dealers were afraid of dust contamination on the used car since it had been in Times Beach. In innumerable ways the flood, coupled with the dioxin disaster, has caused untold misery to the people of Times Beach.

Much has been made and said about the Federal buyout of Times Beach. I want to take this opportunity to thank the Federal Government for the aid and comfort extended to us under the various disaster relief programs. I am proud and grateful to live in a country where such aid is a matter of law and course. The immediate Federal flood relief was swift and comprehensive. Emergency shelters were made available and the Red Cross and Salvation Army did a magnificent job in responding to our need for food, clothing, and temporary shelter. This brings up a most important point about the whole Times Beach situation. If our only problem had been the floods we would have been 95% recovered by now, the end of July. Massive efforts were under way, even before the waters receded to collect debris and organize work teams to help restore our homes. Assistance and volunteers in the best of American tradition came pouring in from surrounding areas. All these efforts, donations, and work, however, came to an abrupt and incredible halt on December 23rd when barricades were place on roads leading into the town, and the Center for Disease Control, Atlanta issued a strong warning to the effect that if you were in Times Beach—get out; if you were out of Times Beach—stay out. The State of Missouri subsequently declared the entire town an "imminent health hazard". For people who owned or rented homes in Times Beach there were small Federal and State grants for the repurchasing of furniture and other personal items if qualifications were met. Many could not meet the qualifications. Federal and State authorities also made some provisions for temporary housing, in apartments, mobile homes, motels, wherever emergency temporary housing was available. City employees lost not only their homes, but their livelihood and thus experienced a double-hardship. I was not mayor at the time the problems exploded, but was a member of the Board of Aldermen. The pressures on the Times Beach mayor were so great that two successive mayors resigned for personal reasons.

Hopes for a new Times Beach soared when the Army Corps of Engineers proposed rebuilding the city on unused Federal park ground directly adjacent to the city of Times Beach. This was also the recommendation of the Federal Emergency Flood Hazard Mitigation Report prepared in response to President Reagan's disaster declaration of 10 December '82. This proposal is one of the *Specific Alternatives* defined in the Superfund Law. When defining "Remedy" or "Remedial Action", "the term includes the costs of permanent relocation of residents and businesses and community facilities where it is determined that relocation is more cost effective then clean up." This was the answer to virtually all of our problems but we quickly learned that this was not be. Under this program the present problems of the business people would not have developed.

A plan to purchase only the privately owned real estate was announced by E.P.A. administrator, Anne Burford, on February 22, 1983. We (city officials and concerned citizens) began an intensive letter writing and lobbying campaign to convince our State Legislature to appropriate the necessary matching funds; which indeed occurred. Terms of the State Appropriation Bill prevented the State from holding title to the ground; so the City of Times Beach was made a party to the Superfund Contract to act as interim titleholder. Throughout this process numerous questions were put to F.E.M.A. asking about various facets of the buyout; concern was intense as to whether or not people would be able to relocate, whether ruined businesses would be compensated to ever reopen elsewhere. On March 19, 1983, F.E.M.A. called a meeting with the residents, and announced the general "terms" of the buyout. In question and answer form F.E.M.A. made statements such as: "Businesses are generally eligible for (similar) acquisition and relocation benefits", and "The intent of the program is to make each resident 'whole' again, as they were on November 30, 1982." We, the City Officials, were convinced that our people would receive compensation sufficient for them to start over somewhere else; that the businesses would be made "whole" again and recover. Webster's Dictionary defines "whole" as "free of defect or impairment, intact;" "whole implies that nothing has been omitted, ignored, abated, or taken away". We are not "whole" unless the economic injury we have suffered is recognized and compensation to cover that injury is provided.

According to the terms of the "Relocation Brochure" distributed businesses are eligible to receive up to maximum $10,000 relocation expense payment. You are disqualified from even that, if you have s similar business elsewhere. This $10,000 sounds like a lot until you analyze the circumstances of some businesses in Times Beach. I'll hit the highlights of some particular instances—detailed sheets for each are attached.

The owner of a gas station/liquor store known as Jim's Fina (Jim and Leon's Corporation) had invested $50,000 in inventory and purchase of the business. Not owning the real estate, he will receive at best the $10,000; but probably only the minimum $2,500 because he has no inventory left to move. He not only lost his $20,000 yearly income, but all of his business investment. Ineligible for S.B.A. disaster loans, he has nothing to look forward to except many years of paying off his business loan and probable bankruptcy.

Steve and Marianne Hrin moved their family owned auto-body shop business to Times Beach in 1979. Times Beach's prime location on a major interstate seemed ideal. The flood cost them only three weeks—the dioxin cost them their business of 29 years. The $10,000 won't even move one frame alignment rack, much less 40 tons of other equipment. The purchase of their real estate will not even begin to compensate them for the loss of a thriving business. Laying off their employees, has resulted in a huge jump in their unemployment taxes and insurance costs. Mrs. Hrin wrote, "We were a classic example of the American Dream come true; work hard, invest wisely, and save regularly". The failure of the Superfund law to provide business oriented compensation again has shattered forever another "American Dream".

The largest business in Times Beach, the two mobile home parks, is owned by Mr. Charles Stone. Included in his business was a used home brokerage firm and a laundromat. With all 337 mobile home pads empty of paying customers, he has to borrow to pay his $17,000 per month interest payments. Active in the buyout negotiations, he repeatedly was assured by F.E.M.A. that he would be "made whole again" by midsummer. The reality of his buyout is that he will be forced deeply into debt to finance the tripled cost of a new park. The eight month delay in solving the dilemma of Times Beach is causing the accumulated interest to eat up a sustantial part of his buyout proceeds and working capital. Without compensation, Mr. Stone, will lose most of his equity and will basically be starting a four million dollar business in a very uncertain economic climate and in a much less desirable location.

J.C. and Doris Walters, owners of KOA Kampground face virtually unsurmountable relocation odds. They have lost the 1983 season and will lose the 1984 season because it is already too late to advertise in the 1984 nationwide directories that bring them 80% of their business. Their new kampground is not yet under construction as they are awaiting the buyout funds which will enable them to start construction of their new campground. Being the closest campground to St. Louis and near the Six Flags Amusement Park on I–44 was a prime location. There is simply no good ground available with the perfect highway access that KOA had. The Walters are ineligible for the $10,000 relocation allowance because they own a small campground elsewhere.

I could go on about Robert Fitts, who had nine rental mobile homes. These have turned into nine overdue payments each month. Or, Melba Perschbacher who has two commercial properties with 10 year leases. She no longer has her assured retirement income but she still has the burdensome debt of her mortgage payments.

I could describe thirty more owner-operated concerns who face the loss of everything without help. How many of these will ever be able to reopen? How much unemployment will result? Additional hardship cases are attached.

However large the financial toll has proven to be, the human toll remains. The fact is that we have been the rope in this tug-of-war. So much controversy has surrounded dioxin, as one doctor has said, that dioxin is the most studied chemical today and still no serious harmful effects are known. However, the Missouri Department of Natural Resources in a letter to Alden Shields, dated August 17, 1982 states "Dioxin acts as a toxin, carcinogen, mutagen, and fetal toxin . . . ."

The concern of serious health effects is a real one for the people to Times Beach, but it is more in the nature of a long range concern for most of us. Others are not so fortunate.

At one intersection of "hot streets" lived four families. Mr. and Mrs. Lewis Bierman moved to Times Beach in 1975. In February, 1978, Linda suffered a miscarriage. In March, 1979 she gave birth to Melissa, who had congenital neuroblastoma, a rare cancer in infants. Melissa died four months later. After having been assured by doctors that having another such child was a 1 in 300,000 chance, Linda bore Jessica in December, 1980. Tragically, Jessica, too, has neuroblastoma cancer and undergoes chemotherapy and radiation treatments every five weeks. Her prognosis is very poor. Their tremendous financial burden is made much worse by the cutbacks in insurance coverage by Mr. Bierman's employer. Future health insurance may become unobtainable as companies exclude people who have lived in known contaminated areas.

Across the street lived Mr. and Mrs. "A". After living there about two years, Mrs. "A" died mysteriously in childbirth from a massive infection that overwhelmed a depressed immune system. Three years later the baby died with a similar disorder.

Next door to Mr. "A" lived Mr. and Mrs. Laine Jumper. A baby born in 1971 died ten months later from a heart defect. Mr. Jumper suffered an acute neuroestenia (loss of energy) in 1978 resulting in eight weeks hospitalization and six months lost work. Consulted neurologists couldn't explain the depressed nerve functions and personality problems but did say that it could be environmentally related. Somewhat recovered Mr. Jumper has never returned to the physical condition necessary to his business and medical insurance is unaffordably expensive.

Opposite Mr. "A" was Mr. and Mrs. "L" living relatively healthy lives until the time of the oil spraying. Three of Mrs. "L's" four children developed severe problems. One daughter became sterile and has been diagnosed with a pre-cancerous cervix. Another daughter experienced undiagnosed low energy and painful extremities for years. She developed a seizure disorder in her early teens and has resulting emotional and personality problems. A third daughter has a disfiguring and incurable skin disorder over 70% of her body. Mrs. "L", herself, began suffering severe menstrual problems and underwent a total hysterectomy in 1974. The surgeon described the uterine tumors as "something that I, or my collegues had never seen before and still have no diagnoses." She has arthritic joint pain with no arthritis, ringing ears, and neural pain localized in her arms; all undiagnosable by various doctors. Mr. "L" has had the acne and blackheads around the eyes characteristic of chloracne, elevated cholestrol and triglycerides, lower extremity cramps, hyper-irritability, loss of libido and personality changes.

Mr. and Mrs. "C" living behind Mr. and Mrs. "J" have demonstrated undiagnosed medical problems. Mrs. "C" has a degenerative nerve and disc problem in her spine, migraines, and severe female disorders. Their eight year old daughter has monthly kidney and bladder bleeding, as well as personality problems. Mr. "C" is hyper-irritable and experiences fluctuating emotional disturbances and hypertension.

The Bodimer family moved to Times Beach in early 1972. In 1972 their street was heavily sprayed with the contaminated oil.

The father started to have heart attacks and problems with diabetes in 1975.

The twin boys in the family have both been diagnosed as having chloracne.

The mother, Shirley, has been seeing a dermatologist for the past three years. She has a recurring skin rash which cannot be diagnosed as to what is causing it or what it is.

Across town is another neighborhood where seizure disorders are the rule rather than the exception. In a grouping of fifteen homes, there are seven families with one or more members suffering a seizure disorder. One family has a baby born with its kidneys on the outside of its body. Behind them was a baby with a hydroencephalic brain (water on the brain). These represent only those who were willing to talk in this area. Perhaps, others exist who do not discuss health problems. Telephone repairmen assigned to Times Beach ten years developed severe rashes on their hands and feet that subsided only after transfering to other areas.

There has been numerous young women with breast cancer; some in their teens. A City employee had a daughter develop seizures after moving into Times Beach. One incredible irony involves a former major who received a medical retirement after fourteen years in the Marine Corps. He had been sprayed in Vietnam with Agent Orange and developed chronic bone marrow disease, resultant hemophillia and severe chest pains. His discharge lists "an unknown toxin agent" as the cause (dioxin in the agent Orange?). How sad to note that he moved to dioxin contaminated Times Beach where his daughter developed severely swollen salivary glands requiring surgery.

While the human temptation exists to blame anything and everything on dioxin, I have related only problems that have defied diagnosis as to their cause from all facets of the medical community. I have ignored many other "normal type" problems existing in these people. I truly believe that many severe health problems in Times Beach will be linked to the dioxin; yet, there is no program of compensation for health benefits or insurance or liability clauses for same in the Superfund Law as it stands today. There are no provisions for "people oriented" compensation from environmental disasters. The Superfund Law requires polluters to reimburse the fund for cleanup costs; but not for the human costs. Polluters should stand liable under this law for damages to people without the delaying and evasive and costly necessity of civil lawsuits.

There is no compensation for the anguish, stress, and resultant mental health problems suffered by our people. At least four fatal stress related heart attacks have occurred since January—are they victims of bad hearts or pressure from all this? At least six complete nervous breakdowns and innumerable near collapses were definitely caused by the problems. Other uncompensated costs involve the transportation of children to school; an additional 10, 15 and in some instances 80 miles daily while living in "temporary housing" awaiting the buyout money. Most families will be unable to permanently relocate in time to get their children registered for school. Our kids will be forced into new schools away from friends and familiar surroundings. There is a shortage of affordable housing in the area, thus forcing many to move farther from work, which raises transportation costs.

What of the future? When the floods came I heard many who had lost everything say, "Well, at least we still have our health". Now, who's to know what our health will be? Lifetime follow-up of people exposed to dangerous chemicals should be mandatory in order that the "toxic time bombs" of cancer and pollutant induced disease will be attributed to the proper cause. A fund should be set aside from the Superfund tax on chemicals to provide necessary health care for affected persons. Fund subsidized life and health insurance is another alternative.

There is no provision for compensation for personal property. When the C.D.C. issued its health warning on December 23, 1982 it advised us to cease all non-emergency cleanup and leave everything undisturbed because of possible contamination. Most of us heeded that warning and did not attempt clean up of clothes, furniture, tools, and other personal property from the flood. It wasn't until February 23, 1983 that the E.P.A. concluded that we could go ahead and remove our things, "with proper cleaning". What vandals and thieves didn't destroy, two and one-half months of rotting in mud and debris did. The buyout will not compensate anyone for any losses of this nature. Small Federal grants to us of $5,000 only go a small way to replace a lifetime's accumulation and most did not even qualify for the grant. Businesses were told that S.B.A. loans were available. Almost all applied and almost all were refused. There was no "ready" money for the business people and they have suffered accordingly.

Trying to summarize the agonizing death of our city is difficult. This experience has been an emotional roller coaster ride for 2,000 people. Peaks of hope one day to be followed by a plunge into despair the next.

We suffered the worst flood in our recorded history on December 5, 1982. Were that the only problem we would have prevailed without Federal aid but this monster called "dioxin" has removed any clear cut goals from our lives and left nothing but questions, doubt, and an underlying knowledge that things will never be the same again. Should there ever be another "Times Beach" in another location, at some future date we would suggest the following:

1. For the business people:

a. Adequate "replacement value" and "relocation compensation"

b. Where a business cannot be relocated fairly then just compensation for the value of the "business enterprise".

c. No resident or business can relocate without knowing: (1) The amount of money available for rebuilding. (2) A definite date the money will be available.

d. Equal grants for business people who have lost income as for residents who have lost shelter.

e. Consideration for the on-going expenses of the business community, such as mortgages, insurance, inventory, debt, etc.

f. Speed. The need to make the above needed information available as rapidly as possible cannot be overemphasized.

2. For the entire community:

a. Compensation for personal property declared contaminated.

b. Compensation from polluters for health problems with strict liability clauses.

c. Lifetime followup of exposed persons. (National registry?)

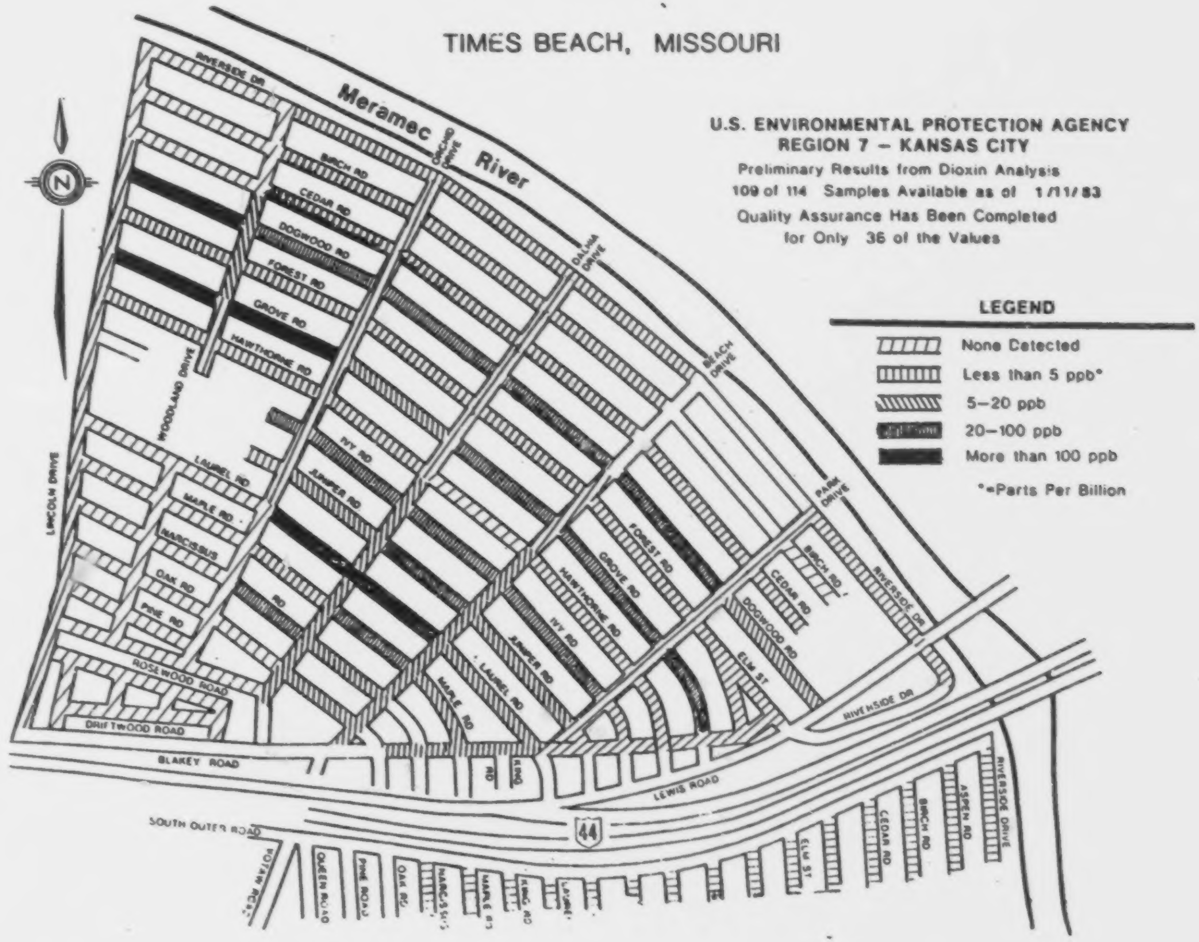
d. Detailed Federal guidelines to provide consistent, equitable treatment for residences, businesses and local government.

e. Mandatory uniform State Superfund laws to guarantee State government cooperation as well as necessary matching funds.

f. Implementation of extraordinary remedies where such measures are warranted as opposed to taking the "cheapest way out".

These are our recommendations to you. It has come to our attention that Senators Danforth and Eagleton have sponsored legislation which will correct some of these inequities, however, it appears this legislation will not be passed in time for it to help the people of Times Beach. Our people need to come under the provisions you are making for others. We ask that you please amend your bill to include us. The laws need to be changed. Provisions for extraordinary response (such as relocating entire communities as described in Sec. 101, PPH 24) should be implemented where it is warranted, uniform mandatory State Superfund laws should be required nationwide to guarantee necessary State matching funds and a swift response from State officials. Victim compensation must be included for residents and businesses that suffer financial and health losses through no fault of their own. Federal response time must be shortened. Some of these cannot be applied to us; in other areas by amending the Superfund law and making it retroactive to include Times Beach some of our very serious problems can be alleviated.

I have presented here the unexaggerated truth. I feel that the intent of Congress in passing the Superfund Law was not in the areas of response to and compensation in the Federal effort at Times Beach. I ask that you, the committee, consider these facts and make appropriate recommendations to the full Congress. I thank you sincerely for myself and the people of Times Beach for your interest. May God bless each of you.


Meramec River
RIVERSIDE DR
BIRCH RD
CEDAR RD
DOGWOOD RD
FOREST RD
GROVE RD
HAWTHORNE RD
WOODLAND DRIVE
ORCHID DRIVE
DALHIA DRIVE
BEACH DRIVE
PARK DRIVE
IVY RD
JUNIPER RD
LAUREL RD
MAPLE RD
NARCISSUS
OAK RD
PINE RD
RD
ROSEWOOD ROAD
DRIFTWOOD ROAD
LINCOLN DRIVE
BLAKEY ROAD
KING RD
ELM ST
RIVERSIDE DR
LEWIS ROAD
44
SOUTH OUTER ROAD
POTAW ROAD
QUEEN ROAD
PINE ROAD
OAK RD
NARCISSUS
MAPLE RD
KING RD
LAUREL
ELM ST
CEDAR RD
BIRCH RD
ASPEN RD
RIVERSIDE DRIVE
N

TIMES BEACH, MISSOURI

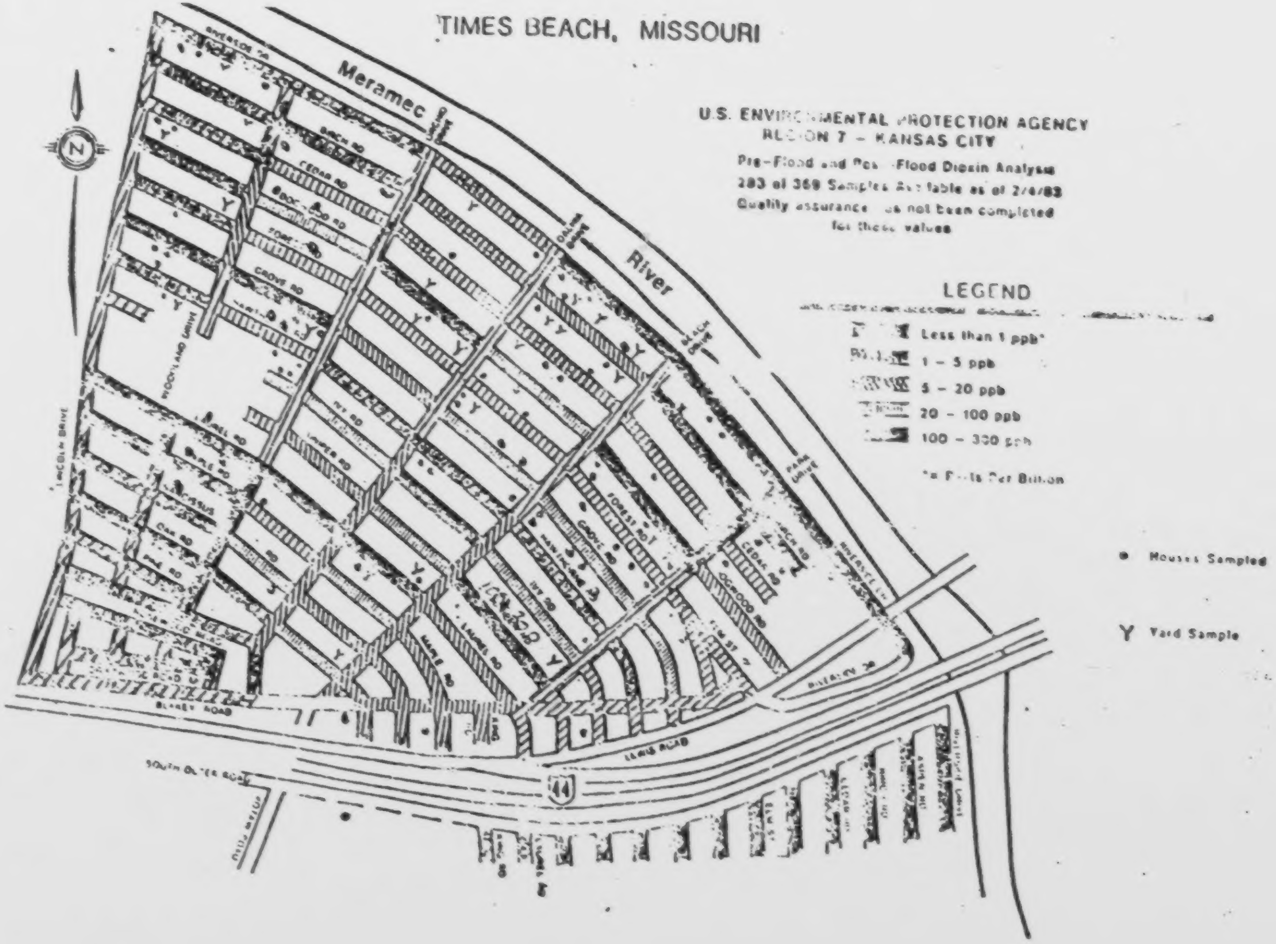
U.S. ENVIRONMENTAL PROTECTION AGENCY
REGION 7 – KANSAS CITY
Preliminary Results from Dioxin Analysis
109 of 114 Samples Available as of 1/11/83
Quality Assurance Has Been Completed
for Only 36 of the Values

LEGEND

▨	None Detected
▥	Less than 5 ppb*
▧	5–20 ppb
▩	20–100 ppb
■	More than 100 ppb

*=Parts Per Billion

TIMES BEACH, MISSOURI



U.S. ENVIRONMENTAL PROTECTION AGENCY
REGION 7 - KANSAS CITY

Pre-Flood and Post-Flood Dioxin Analysis
283 of 369 Samples Available as of 2/4/83
Quality assurance has not been completed
for these values

LEGEND
Less than 1 ppb*
1 - 5 ppb
5 - 20 ppb
20 - 100 ppb
100 - 300 ppb
* = Parts Per Billion
● Houses Sampled
Y Yard Sample
Meramec
River
N
GROVE RD
FOREST RD
CEDAR RD
OGWOOD RD
LAUREL RD
MAPLE RD
IVY RD
WOODLAND DRIVE
LINCOLN DRIVE
BLAKEY ROAD
LEWIS ROAD
SOUTH OUTER ROAD
PARK DRIVE
44

Department of Social Services
MISSOURI DIVISION OF HEALTH

RECOMMENDED PUBLIC HEALTH PRECAUTIONS AT TIMES BEACH, MISSOURI
Centers for Disease Control

On December 16, 1982, a private contract laboratory released results of analysis of soil samples which had been collected at Times Beach before the recent flooding had occurred. These results showed two samples with elevated TCDD dioxin levels of 10 and 16 ppb However, today 12-23-82, preliminary data from Environmental Protection Agency contract laboratories have become available for samples which were also collected from Times Beach before the week of the flooding. The data are very preliminary and EPA has not yet had the opportunity to quality assure and verify these data. However, while most results remain in the range of 1 to 20 ppb, two of the first 38 samples showed TCDD levels of greater than 100 ppb. At this point we do not know the extent of areas with these high levels or what has happened to the soil from these more highly contaminated areas as a result of the flooding. It is possible that much of the most highly contaminated soil has been diluted or spread about by the flood waters. It is also possible that isolated highly contaminated areas still exist or have been shifted to new areas. Because of the high degree of present uncertainties, that can only be resolved by further extensive post flooding sampling, the following public health recommendations are made:

1. Non-emergency cleanup activities in Times Beach area should cease. In the unlikely event that emergency activities are necessary, they should be done with full protective gear and only after notification of and consultation with the appropriate federal and state agencies.
2. Residents who have been temporarily relocated are discouraged from moving back into the area.
3. Residents who have already begun to move back into the area are encouraged to leave.

It is recommended that these measures be observed until more extensive post-flood soil sampling and analysis are completed. EPA plans to accomplish these activities within the most rapid time frame possible. For individuals who choose to remain despite these recommendations, the following minimal precautions should be taken:

A. Individuals should limit direct contact with the soil and should not eat, drink or smoke while working in these areas.
In addition, they should wash their hands before any activity which would lead to similar exposures. Also activities which are likely to produce significant amounts of air-borne soil particles should be held to a minimum.

B. Individuals should wear boots which should be thoroughly rinsed off before leaving the area.

C. Coveralls should be worn on-site. If during the course of their activity these individuals should become drenched, the coveralls should be changed immediately. In all cases, used coveralls should be kept in plastic bags apart from all other items to be laundered. Then, all similarly soiled clothing should be dry-cleaned (preferably separate from all other cleaning loads).

D. When direct contact with soil is likely, gloves should be worn. These items should then be cleaned as above or packaged for proper incineration.

E. At this time the use of respirators does not appear to be warranted. However, the use of dust filter masks is encouraged.

F. Individuals should take care to shower and thoroughly wash off adhered dirt after each day's activities with special attention to areas where dirt might accumulate, (e.g., under fingernails).

THE ABOVE STATEMENT RECEIVED BY TELEPHONE FROM CDC AT 2:00 p.m.
THURSDAY, December 23, 1982.

MO Division of Health

INTERAGENCY FLOOD HAZARD MITIGATION REPORT

in response to the December 10, 198

Disaster Declaration

(FEMA-672-DR-MO)

Covering

Bollinger, Carter, Cape Girardeau, Franklin, Iron, Jefferson, Lincoln, **Perry, Phelps,** Pulaski, Ripley, Ste. Genevieve, St. Charles, St. Louis, **and Wayne** Counties, Missouri.

This report was prepared by the Region VII Interagency Hazard Mitigation **Team** which consisted of the Following Federal and State Agencies:

←Federal Emergency Management Agency
Department of the Army, Corps of Engineers
Department of Commerce
Department of Housing and Urban Development
Department of Agriculture
Department of Transportation
Department of the Interior
Environmental Protection Agency
Small Business Administration
←State of Missouri, Department of Natural Resources

EXECUTIVE SUMMARY

Purpose of Report

The report and recommendations of the team are intended to provide the framework for Flood Hazard Mitigation during the recovery process and reduce the potential for future flood losses.

Description of the Disaster

On December 6, 1982, the Governor of Missouri requested that a Major Disaster be declared in the State of Missouri, resulting from heavy rains and flash flooding. On December 10, 1982, the President of the United States determined that flood damages within the State of Missouri were of sufficient magnitude and severity to warrant a major disaster declaration under PL 93-288.

The intense early winter storms moved across the State over a devastating five-day period (December 1-5, 1982), causing severe weather, tornadoes, damaging winds, intense rainfall and widespread flooding of major rivers and their tributaries throughout the central and eastern portions of the State. Subsequently, fifteen counties were declared eligible for Individual Assistance: Bollinger, Cape Girardeau, Carter, Franklin, Iron, Jefferson, Lincoln, Perry, Phelps, Pulaski, Ripley, Ste. Genevieve, St. Charles, St. Louis and Wayne Counties. Several counties were also declared for Public Assistance along with individual cities and towns throughout the disaster area.

The most severe damages and hardship occurred in various communities along the routes of the major rivers and streams, such as the Mississippi, Missouri, Meramec and numerous other tributaries. Estimates indicate that over 3000 residences were severely damaged and over 700 were completely destroyed. Early estimates indicated flood crests were equal to or greater than the 100-year flood event, establishing record stages in numerous communities and urban areas.

Six lives were reported lost as a result of these storms, two of which were persons attempting rescue operations for stranded flood victims. Total damage estimates for this disaster are not available, but losses are expected to exceed $100 million.

Preparation of the Report

The Region VII Interagency Hazard Mitigation Team was activated by the Regional Director on December 14, 1982, and a letter of confirmation was sent on December 14 (Appendix 1). On December 16, 1982, the Team Leader requested and the Regional Director concurred to extend the deadline for the submission of this report to 30 days from the date of declaration. This action was needed in order to adequately involve the vast geographical area and the large number of communities in the disaster area.

Recommendations

The major recommendations in this report are for the development of a sound floodplain management program within the State of Missouri, fully staffed and managed at the State level; consideration of a multi-agency approach to acquisition of properties in Times Beach; public awareness campaigns on the severity of flash flood watches/warnings, procedures to follow when they are received, and better dissemination of information to protect the citizens.

Other recommendations of importance are the possibility of a Section 1362 Acquisition Project; allocation goals for funding Section 1362 on a Regional basis; and funding of the U.S. Army Corps of Engineers Lower Meramec River flood control project in St. Louis and Jefferson Counties, Missouri.

Recommendations of significant importance are to finalize "constructive total loss" (CTL) provisions of the National Flood Insurance Program (NFIP); State of Missouri to enact enabling legislation for all counties to join the NFIP; and modification to existing ordinaces for the placing and replacing of mobile homes on pads located in designated floodways when destroyed or removed.

Many recommendations are of an investigative nature, an approach that should allow for a complete analysis of the options and should result in a more comprehensive and reasonable effort for flood hazard mitigation.

Implementation

The lead agency or agencies associated with each recommendation is identified and will be pursued by the team leader for a status accounting for the programs of implementation. FEMA will be responsible for monitoring each recommendation and for completing the 90-day post-flood progress report provides training and other kinds of assistance to local emergency preparedness officials. Local agency dissemination of received warnings, and interpretations of those warnings, are vital to the protection of lives and property. (For example, when a flood crest at 34 fett on the Eureka gage is forecast, what does that mean to a resident of a particular street in Eureka, and to any evacuation route?)

Lead Agency: State Emergency Management Agency (SEMA).

Financing: Minimal.

Schedule: Immediately. Incorporate in State 406 Plan.

7. **Work Element:** Investigate alternative means of inter-agency communication via single-sideband radios and assigned frequences.

Background: During flooding, it is imperative that agencies such as the NWS, the USGS, and the Corps of Engineers share information to keep abreast of flooding developments and flood crest forecasts. During the latest flooding events, the NWS telephone lines were saturated with calls from the public, the news media, and intra-NWS office coordination. The Corps and the USGS experienced difficulty in relaying pertinent data to the NWS and in obtaining the latest flood crest forecasts from the NWS. The Corps is presently equipped with single-sideband radios and assigned frequencies for communicating with their operational projects and field offices.

Lead Agency: NWS, USGS, and Corps of Engineers (COE).

Financing: Purchase of base and mobile single-sideband radios for the USGS, NWS, and COE.

Schedule: FY84 and FY85.

8. **Work Element:** Automate and enhance the rainfall and river stage reporting network to improve the disaster warning capability and maximize lead times.

Background: The NWS issued warnings and statements as promptly and efficiently as was physically possible. [illegible] and enhancement will provide [illegible] time [illegible] to the public.

Lead Agency: NWS, USGS, and COE.

Financing: Ongoing.

Schedule: Immediately.

9. **Work Element:** The Federal Coordinating Officer (FCO) of FEMA, in conjunction with appropriate federal and state agencies, (including the U.S. Army, Corps of Engineers; the Environmental Protection Agency; the Small Business Administration; the Department of Housing and Urban Development; and the State of Missouri, Department of Natural Resources and State Emergency Management Agency) should identify a potential comprehensive plan, programs and sources of funding to acquire and relocate properties of Times Beach, Missouri area. This relocation/acquisition is to [illegible] sound floodplain management practices.

Background: St. Louis County (HUD Entitlement Community) and City of Times Beach should be invited to attend planning and discussion sessions. Precedents for multi-agency cooperation in relocating flood-prone communities in other states exist. The possibility that there are multiple hazards may enhance opportunities for mitigation.

Lead Agency: FEMA Regional Office.

Financing: FEMA as host, providing minutes, etc.

Schedule: As soon as possible.

Federal Emergency Management Agency

Region VII 911 Walnut Street Kansas City, Missouri 64106

DISASTER FIELD OFFICE
8400 WATSON ROAD
ST. LOUIS, MISSOURI 63119

MARCH 19, 1983

GENERAL QUESTIONS AND ANSWERS
TIMES BEACH, MISSOURI, RELOCATION PLAN

1. Q. WHAT IS THE FEDERAL GOVERNMENT GOING TO DO WITH TIMES BEACH?

A. AS THE ENVIRONMENTAL PROTECTION AGENCY SAID ON FEBRUARY 22, 1983, FEMA IS GOING TO BUY OUT THE PROPERTY OF THOSE RESIDENTS WHO WANT TO MOVE.

2. Q. HOW WILL FEMA DO THIS?

A. FEMA INTENDS TO SEND ALL PEOPLE WHO OWN REAL PROPERTY, INCLUDING MOBILE HOMES, IN TIMES BEACH A LETTER ADVISING THEM OF THE PROPOSED BUY OUT AND ASKING THEM TO TELL US IF THEY ARE INTERESTED IN SELLING THEIR PROPERTY.

3. Q. HOW MUCH WILL FEMA PAY FOR THE PROPERTY?

A. FEMA WILL PAY WHAT IS KNOWN AS "FAIR MARKET VALUE" FOR THE PROPERTIES AS THEY WERE ON NOVEMBER 30, 1982, PRIOR TO THE FLOOD AND WITHOUT LESSENING THE VALUE DUE TO DIOXIN.

4. Q. WHAT IF I CAN'T AFFORD TO MOVE?

A. AS PART OF THE RELOCATION PLAN, FEMA IS PREPARED TO OFFER ELIGIBLE HOMEOWNERS AND BUSINESSES RELOCATION ASSISTANCE, SUCH AS REASONABLE MOVING EXPENSES, AND HELP IN FINDING A COMPARABLE HOME OR BUSINESS. IN ADDITION, UP TO A TOTAL COST OF $15,000 FOR OWNER/OCCUPANTS FOR ASSISTANCE IN 1) OFFSETTING NEW HOME SETTLEMENT COST, 2) ANY DIFFERENCE IN MORTGAGE RATES, 3) AS WELL AS HELP IN MEETING MINIMUM DIFFERENCES IN COSTS BETWEEN HOMES WHICH WILL BE DECENT, SAFE, AND SANITARY. [SEE QUESTION SEVEN FOR ELIGIBILITY.]

5. Q. WHO WILL BE BOUGHT OUT?

A. GENERALLY, THOSE INDIVIDUALS THAT OWN PROPERTY WITHIN THE CORPORATE LIMITS OF TIMES BEACH, MISSOURI, WILL BE CONSIDERED FOR POTENTIAL BUY OUT.

6. Q. WHO WILL BE RELOCATED?

A. GENERALLY, THOSE PERSONS THAT HAVE BEEN BOUGHT OUT, AS WELL AS THOSE RENTING PROPERTY IN TIMES BEACH, INCLUDING THOSE TIMES BEACH RESIDENTS NOW IN "TEMPORARY HOUSING".

7. Q. WHAT ARE FEMA'S ELIGIBILITY TIME STANDARDS?

A. APPLICANTS CAN APPLY FOR TWO TYPES OF ASSISTANCE:

1)OWNER/OCCUPANTS COULD BE ELIGIBLE FOR BOTH A BUY OUT AND RELOCATION TO COMPARABLE DECENT, SAFE, AND SANITARY HOUSING. TO BE ELIGIBLE FOR RELOCATION ASSISTANCE, THE OWNER/OCCUPANT MUST HAVE OWNED/OCCUPIED THE HOUSE CONTINUOUSLY FROM AUGUST 26, 1982, TO FEBRUARY 22, 1983, OR UNTIL FORCED TO LEAVE DUE TO CONDITIONS BEYOND HIS/HER CONTROL, BUT NOT PRIOR TO DECEMBER 1, 1982.

2)RENTERS GENERALLY WILL BE ELIGIBLE ONLY FOR RELOCATION ASSISTANCE IF THEY OCCUPIED A UNIT UNDER A VALID LEASE IN EFFECT ON NOVEMBER 24, 1982, THAT CONTINUED IN EFFECT UNTIL FORCED TO LEAVE DUE TO CONDITIONS BEYOND HIS/HER CONTROL, BUT NOT PRIOR TO DECEMBER 1, 1982.

8. Q. IF I DONT'T AGREE WITH WHAT YOUR OFFER IS, WHAT ARE MY CHOICES?

A. AS HAS BEEN SAID, THE PROGRAM IS VOLUNTARY SO YOU CAN ALWAYS REFUSE THE OFFER. YOU CAN ALSO ASK TO MEET WITH THE APPRAISER AND DISCUSS YOUR INDIVIDUAL CONCERNS. ANOTHER OPTION IS TO HIRE YOUR OWN APPRAISER TO PERFORM A SECOND APPRAISAL, THEN MEET WITH OUR APPRAISER TO DISCUSS THE DIFFERENCES.

9. Q. HOW WILL ANY BACK TAXES, LIENS, ETC., BE HANDLED?

A. AS WITH ANY NORMAL REAL ESTATE TRANSACTION, THE SELLER IS RESPONSIBLE FOR CLEARING ALL DEBTS ON THE PROPERTY. ALL LIENS AND TAXES WILL HAVE TO BE TAKEN CARE OF PRIOR TO THE TRANSFER OF TITLE.

10. Q. DO BUSINESSES QUALIFY TO BE BOUGHT AND RELOCATED?

A. YES, BUSINESSES ARE GENERALLY ELIGIBLE FOR SIMILAR ACQUISITION AND RELOCATION BENEFITS. THE CRITERIA FOR THE APPRAISAL IS SOMEWHAT DIFFERENT, BUT WE BELIEVE IT IS FAIR AND EQUITABLE.

11. Q. UNDER THE PLAN, WILL YOU PAY FOR THE REPLACEMENT OF PERSONAL PROPERTY?

A. WE WILL GENERALLY NOT PAY FOR PERSONAL PROPERTY INASMUCH AS THE ADVISORY FROM THE CENTERS FOR DISEASE CONTROL AND THE STATE DEPARTMENT OF HEALTH HAVE INDICATED THAT WITH PROPER CLEANING YOU MAY REMOVE YOUR PERSONAL PROPERTY.

12. Q. WILL YOU BUY MY MOBILE HOME?

A. GENERALLY, YES, WE WILL PURCHASE YOUR MOBILE HOME BASED UPON THE FAIR MARKET VALUE, IF YOU MEET THE CRITERIA.

13. Q. SOME MOBILE HOME OWNERS HAVE RECEIVED INSURANCE CLAIMS ON DAMAGE FROM THE FLOOD. IF FEMA BUYS THEM OUT WILL THEY RECEIVE A WINDFALL?

A. NO, THE INTENT OF THE PROGRAM IS TO MAKE EACH RESIDENT WHOLE AGAIN, AS THEY WERE ON NOVEMBER 30, 1982, BUT NOT TO ALLOW ANY WINDFALL PROFITS. ANY INSURANCE PROCEEDS OR SALVAGE AMOUNTS WILL BE DEDUCTED FROM THE FAIR MARKET VALUE PRIOR TO SETTLEMENT.

14. Q. WHAT IF THE OWNER/RENTER OF A POSSIBLE "BUY OUT" IS UNABLE TO PERSONALLY NEGOTIATE THIS OFFER, (i.e., HANDICAPPED, HE'S IN ANOTHER STATE, OR SHE'S AN ABSENTEE OWNER)?

A. FEMA WILL NEGOTIATE ONLY WITH OWNERS AND RENTERS OF GOOD TITLE. HOWEVER, IF SUCH OWNERS SIGN A LEGAL SPECIFIC POWER OF ATTORNEY TO SOMEONE, IT WILL BE RECOGNIZED.

15. Q. WHAT ELSE MUST BE DONE BEFORE FEMA CAN PROCEED WITH THE RELOCATION?

A. PRIOR TO OFFICIALLY BEGINNING THE BUY OUT, FEMA MUST OBTAIN A WRITTEN COMMITMENT FROM THE STATE OF MISSOURI TO TAKE TITLE TO THE LAND, AND EPA MUST PROVIDE SOME ASSURANCES TO THE STATE OF MISSOURI RELATIVE TO THE CLEAN UP. WE ARE HOPEFUL THAT THESE ISSUES WILL BE RESOLVED.

16. Q. HOW SOON WILL WE BE ABLE TO SELL AND MOVE?

A. BUYING AND MOVING A CITY OF APPROXIMATELY 800 FAMILIES IS NOT AN EASY TASK. THIS SUMMER, WE PLAN ON BEING ABLE TO BEGIN MAKING OFFERS TO BUY, AND DEPENDING UPON YOUR RESPONSE, MOVING CAN BEGIN ANYTIME AFTER THAT. FROM NOW UNTIL JULY WE INTEND TO BE STAFFING UP, DOING ALL OF THE REQUIRED TITLE WORK, APPRAISALS, ETC. WE ARE DOING ALL OF THIS FIRST SO WHEN WE START MAKING OFFERS, IT WILL MOVE REASONABLY FAST.

17. Q. WHAT HAPPENS IF I DECIDE NOT TO SELL AND WISH TO REMAIN IN TIMES BEACH?

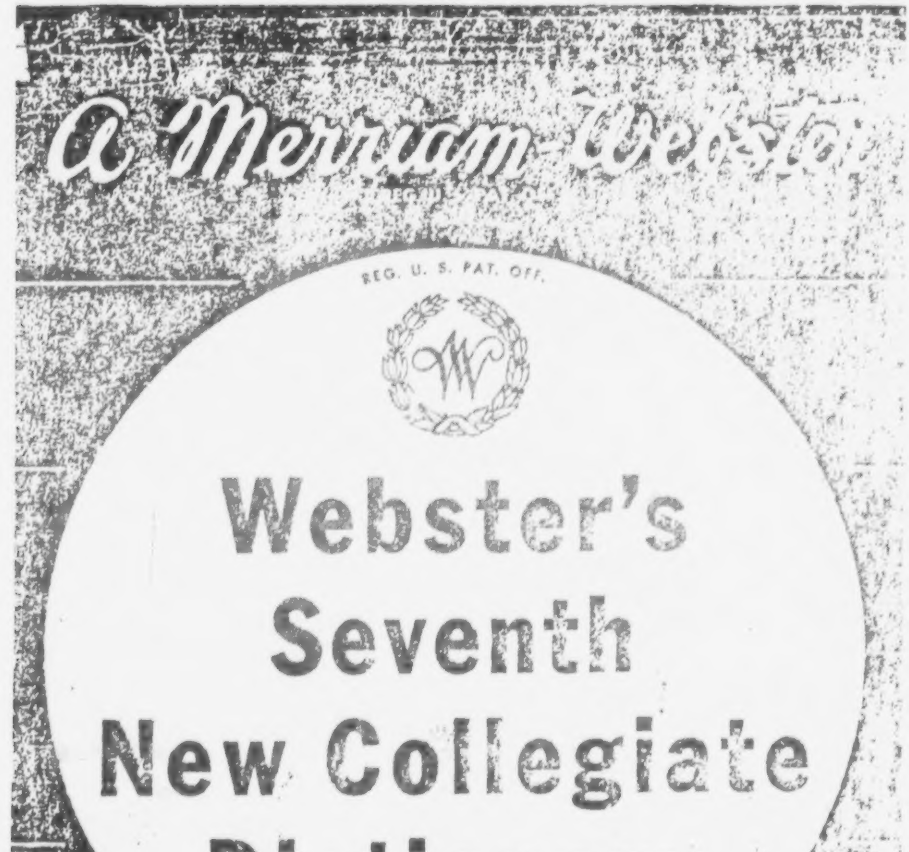
A. FOLLOWING THE BUY OUT, THE EPA AND THE STATE OF MISSOURI WILL DETERMINE WHAT ACTIONS ARE NECESSARY TO DEAL WITH THE DIOXIN CONTAMINATION IN TIMES BEACH. AT A MINIMUM, YOU MAY BE REQUIRED TO RELOCATE FROM TIMES BEACH TEMPORARILY WHILE CLEAN UP OCCURS. WHAT HAPPENS BEYOND THAT DEPENDS UPON THE LONG TERM USE THE STATE MAKES OF TIMES BEACH. DUE TO THE FLOOD PROBLEM, MOST CONSTRUCTION OF RESIDENTIAL AND COMMERCIAL/INDUSTRIAL BUILDINGS WILL BE PROHIBITED.

18. Q. WILL THE UNUSED MONEY BE DIVIDED EQUALLY AMONG THE REMAINING RESIDENTS?

A. NO, ANY UNUSED FUNDS WILL REMAIN IN THE U.S. TREASURY.

19. Q. HOW MAY I CONTACT FEMA SO THAT ADDITIONAL QUESTIONS MAY BE ANSWERED?

A. FEMA HAS INSTALLED A HOT-LINE "800" AREA CODE NUMBER FOR YOU TO USE IN SEEKING ANSWERS TO YOUR QUESTIONS. THE NUMBER FOR MISSOURI IS 1-800-392-1932. NORMAL OPERATIONS ARE MONDAY THROUGH FRIDAY, 8:00 A.M. TO 5:00 P.M.



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COMPLETELY NEW EDITION

^{2}**whittle** *vb* **whit·tling** \ˈhwit-liŋ, -ᵊl-iŋ, ˈwit-\ *vt* **1 a** : to pare or cut off chips from the surface of (wood) with a knife **b** : to shape or form by so paring or cutting **2** : to reduce, remove, or destroy gradually as if by cutting off bits with a knife : PARE — usu. used with an adverb ⟨~ down expenses⟩ ~ *vi* **1** : to cut or shape wood by slowly paring it with a knife **2** : to wear oneself or another out with fretting — **whit·tler** \-lər, -ᵊl-ər\ *n*

whit·tling *n* : a piece cut away in whittling

whit·tret \ˈhwi-trət, ˈwi-\ *n* [ME *whitrat*, fr. *white*, *whit* white + *rat*] *chiefly Scot* : WEASEL

whity *or* **whit·ey** \ˈhwīt-ē, ˈwīt-\ *adj* : WHITISH — usu. used in combination

^{1}**whiz** *or* **whizz** \ˈhwiz, ˈwiz\ *vb* **whizzed**; **whiz·zing**; **whiz·zes** [imit.] *vi* **1** : to hum, whir, or hiss like a speeding object (as an arrow or ball) passing through air **2** : to fly or move swiftly with a whiz ~ *vt* : to cause to whiz; *esp* : to rotate very rapidly

^{2}**whiz** *or* **whizz** *n, pl* **whiz·zes** **1** : a hissing, buzzing, or whirring sound **2** : a movement or passage of something accompanied by a whizzing sound

^{3}**whiz** *n, pl* **whiz·zes** [prob. by shortening & alter.] : WIZARD 3

whiz-bang *or* **whizz-bang** \ˈhwiz-ˌbaŋ, ˈwiz-, -ˈbaŋ\ *n* **1 a** : a small shell of such high velocity that the sound it makes in passing through the air is almost simultaneous with its explosion **b** : a firecracker or similar firework resembling such a shell **c** : ROBOT BOMB **2** : someone or something conspicuous for noise, speed, or startling effect

whiz–bang *adj* : EXCELLENT, NOTABLE

whiz·zer \ˈhwiz-ər, ˈwiz-\ *n* : one that whizzes: as **a** : BULL-ROARER **b** : a centrifugal machine for drying something (as grain, sugar, or nitrated cotton)

who \(ˈ)hü, ü\ *pron* [ME, fr. OE *hwā*; akin to OHG *hwer*, interrog. pron., who, L *quis*, Gk *tis*, L *qui* rel. pron., who] **1** : what or which person or persons — used as an interrogative ⟨~ was elected president⟩ ⟨find out ~ they are⟩; used by speakers on all educational levels and by many reputable writers, though disapproved by some grammarians, as the object of a verb or a following preposition ⟨~ did I see but a Spanish lady —Padraic Colum⟩ ⟨do not know ~ the message is from —G.K.Chesterton⟩ **2** : the person or persons that : WHOEVER **3** — used as a function word to introduce a relative clause; used esp. in reference to persons ⟨my father, ~ was a lawyer⟩ but also in reference to groups ⟨a generation ~ had known nothing but war —R.B.West⟩ or to animals ⟨dogs ~ . . . fawn all over tramps —Nigel Balchin⟩ or to inanimate objects esp. with the implication that the reference is really to a person ⟨earlier sources ~ maintain a Davidic ancestry —F.M.Cross⟩ — **as who** *archaic* : as one that : as if someone — **as who should say** *archaic* : so to speak — **who's who** : the identity of or the noteworthy facts about each of a number of persons

whoa \ˈwō, ˈhō, ˈhwō\ *v imper* [ME *whoo*, *who*] — a command to a draft animal to stand still

who·dun·it \hü-ˈdən-ət\ *n* [substandard *who done it?*] : a detective story or mystery story presented as a novel, play, or motion picture

who·ev·er \hü-ˈev-ər\ *pron* : whatever person : no matter who — used in any grammatical relation except that of a possessive

^{1}**whole** \ˈhōl\ *adj* [ME *hool* healthy, unhurt, entire, fr. OE *hāl*; akin to OHG *heil* healthy, unhurt, ON *heill*, OSlav *cělŭ*] **1 a** (1) : free of wound or injury : UNHURT (2) : recovered from a wound or injury : RESTORED (3) : HEALED **b** : free of defect or impairment : INTACT, UNBROKEN **c** : physically sound and healthy : free of disease or deformity **2** : having all its proper parts or components : ENTIRE, UNMODIFIED ⟨~ milk⟩ **3 a** : constituting the total sum or undiminished entirety of : INTEGRAL **b** : each or all of the **4 a** : constituting an undivided unit : UNBROKEN, UNCUT ⟨a ~ roast suckling pig⟩ **b** : directed to one end : not scattered or dispersed : CONCENTRATED ⟨promised to give it his ~ attention⟩ **5** : seemingly complete or total **6** : having the same father and mother ⟨~ brother⟩

syn WHOLE, ENTIRE, TOTAL, ALL, GROSS mean including everything or everyone without exception. WHOLE implies that nothing has been omitted, ignored, abated, or taken away; ENTIRE may suggest a being completed or perfected; TOTAL implies that everything has been counted, weighed, measured, or considered; ALL may equal WHOLE, ENTIRE, or TOTAL; GROSS implies that customary or expected deductions have not been made **syn** see in addition PERFECT

^{2}**whole** *n* **1** : a complete amount or sum : a number, aggregate, or totality lacking no part, member, or element **2** : something constituting a complex unity : a coherent system or organization of parts fitting or working together as one **syn** see SUM — **on the whole** **1** : in view of all the circumstances or conditions : all things considered **2** : in general : in most instances : TYPICALLY

whole gale *n* : wind having a speed of 55 to 63 miles per hour

whole·heart·ed \ˈhōl-ˈhärt-əd\ *adj* : undivided in purpose, enthusiasm, or will : HEARTY, ZESTFUL ⟨~ effort⟩ **syn** see SINCERE — **whole·heart·ed·ly** *adv* — **whole·heart·ed·ness** *n*

whole hog *n* : the whole way or farthest limit : ALL — used esp. in the phrase *go the whole hog*

whole·ness \ˈhōl-nəs\ *n* : the quality or state of being whole

whole note *n* : a musical note equal in value to four quarter notes or two half notes

whole number *n* : INTEGER

^{1}**whole·sale** \ˈhōl-ˌsāl\ *n* **1** : the sale of commodities in quantity usu. for resale (as by a retail merchant) **2** : a large scale or indiscriminate proceeding — used esp. in the phrase *by wholesale*

^{2}**wholesale** *adj* **1** : of, relating to, or engaged in the sale of commodities in quantity for resale ⟨a ~ grocer⟩ **2** : performed on a large scale esp. without discrimination ⟨~ slaughter⟩ **syn** see INDISCRIMINATE

^{3}**wholesale** *adv* : in a wholesale manner

^{4}**wholesale** *vb* : to sell at wholesale

whole·sal·er \-ˌsā-lər\ *n* : a merchant middleman who sells chiefly to retailers, other merchants, or industrial, institutional, and commercial users mainly for resale or business use

whole·some \ˈhōl-səm\ *adj* **1** : promoting health or well-being of mind or spirit **2** : promoting health of body **3 a** : sound in body, mind, or morals **b** : having the simple health or vigor of normal domesticity **4 a** : based on well-grounded fear : PRUDENT **b** : SAFE **syn** see HEALTHY — **whole·some·ly** *adv* — **whole·some·ness** *n*

whole–souled \-ˈsōld\ *adj* : moved by ardent enthusiasm or single-minded devotion : WHOLEHEARTED

whole step *n* : a musical interval comprising two half steps (as C–D or F♯–G♯) — called also *whole tone*

whole wheat *adj* : made of ground entire wheat kernels

whole wheat flour *n* : [illegible]

whol·ly \ˈhō(l)-lē\ *adv* [ME *hoolly*, fr. *hool* whole] **1** : to the full or entire extent : COMPLETELY **2** : to the exclusion of other things : SOLELY

whom \(ˈ)hüm, üm\ *pron*, *objective case of* WHO — used as an interrogative or relative; used as object of a verb or a preceding preposition ⟨to know for ~ the bell tolls —John Donne⟩ or less frequently as the object of a following preposition ⟨the man ~ you wrote to⟩ though now often considered stilted esp. as an interrogative and esp. in oral use; occas. used as predicate nominative with a copulative verb or as subject of a verb esp. in the vicinity of a preposition or a verb of which it might mistakenly be considered the object ⟨~ say ye that I am —Mt 16:15 (AV)⟩ ⟨people . . . ~ you never thought would sympathize —Shea Murphy⟩

whom·ev·er \hü-ˈmev-ər\ *pron*, *objective case of* WHOEVER

^{1}**whomp** \ˈhwämp, ˈhwómp, ˈwämp, ˈwómp\ *n* [imit.] : a loud slap, crash, or crunch

^{2}**whomp** *vb* : BANG, SLAP

whomp up *vt* **1** : to stir up : AROUSE **2** : to put together esp. hastily

whom·so \ˈhüm-(ˌ)sō\ *objective case of* WHOSO

whom·so·ev·er \ˌhüm-sə-ˈwev-ər\ *objective case of* WHOSOEVER

^{1}**whoop** \ˈh(w)üp, ˈh(w)up, *least frequently for vi 3* ˈhüp [illegible] *vb* [ME *whopen*, fr. MF *houpper*, of imit. origin] *vi* **1** : to utter a whoop in expression of eagerness, enthusiasm, or enjoyment : SHOUT **2** : to utter the cry of an owl : HOOT **3** : to make the characteristic whoop of whooping cough **4** : to go or pass with a loud noise ~ *vt* **1 a** : to utter or express with a whoop **b** : to urge, drive, or cheer on with a whoop **2** : to agitate in behalf of **3** : BOOST, RAISE — **whoop it up** **1** : to celebrate riotously : CAROUSE **2** : to stir up enthusiasm

^{2}**whoop** \ˈh(w)üp, ˈh(w)up, *least frequently for 3* ˈhüp [illegible] **1 a** : a loud yell expressive of eagerness, exuberance, or jubilation — often used interjectionally **b** : a shout of hunters or of men in battle or pursuit **2** : the cry of an owl : HOOT **3** : the crowing intake of breath following a paroxysm in whooping cough **4** : the smallest bit ⟨not worth a ~⟩

whoop–de–do *or* **whoop–de–doo** \ˌh(w)üp-dē-ˈdü, ˌh(w)up-, ˌwüp-, ˌwup-\ *n* [prob. irreg. fr. ^{1}*whoop*] **1** : noisy and conspicuous activity esp. in entertainment, politics, or commercial promotion **2** : agitated public discussion

^{1}**whoop·ee** \ˈ(h)wup-(ˌ)ē, ˈ(h)wü-(ˌ)pē; (h)wü-ˈpē, (h)wü-\ *interj* [irreg. fr. ^{1}*whoop*] — used to express exuberant delight

^{2}**whoop·ee** \ˈ(h)wup-(ˌ)ē, ˈ(h)wü-(ˌ)pē\ *n* **1** : the feverish participation in alcohol-and-sex orgies first widely conspicuous during the U.S. prohibition era — often used in the phrase *make whoopee* **2** : boisterous convivial fun

whooping cough *n* : an infectious disease esp. of children caused by a bacterium (*Bordetella pertussis*) and marked by a convulsive spasmodic cough sometimes followed by a crowing intake of breath — called also *pertussis*

whoop·la \ˈh(w)ü-ˌplä, ˈh(w)üp-ˌlä\ *n* [alter. of *hoopla*] **1** : a noisy commotion **2** : boisterous merrymaking

^{1}**whoosh** \ˈhwüsh, ˈwüsh, ˈ(h)wush\ *vb* [imit.] : to move with an explosive or sibilant rush

^{2}**whoosh** *n* : a swift or explosive rush

^{1}**whop** \ˈhwäp, ˈwäp\ *vt* **whopped**; **whop·ping** [ME *whappen*, alter. of *wappen* to throw violently] **1** : to pull or whip out **2 a** : BEAT, STRIKE **b** : to defeat totally

^{2}**whop** *n* : a heavy blow : THUMP

whop·per \ˈhwäp-ər, ˈwäp-\ *n* [^{1}*whop*] **1** : something unusually large or extreme of its kind **2** : a monstrous lie

whop·ping \ˈhwäp-iŋ, ˈwäp-\ *adj* : extremely large

^{1}**whore** \ˈhō(ə)r, ˈhó(ə)r\ *n* [ME *hore*, fr. OE *hōre*; akin to ON *hōra* whore, *hōrr* adulterer, L *carus* dear — more at CHARITY] : a woman who practices unlawful sexual intercourse esp. for hire : PROSTITUTE

^{2}**whore** *vi* **1** : to have unlawful sexual intercourse as or with a whore **2** : to pursue a faithless, unworthy, or idolatrous desire ~ *vt*, *obs* : to corrupt by lewd intercourse

whore·dom \ˈhōrd-əm, ˈhórd-\ *n* [ME *hordom* sexual immorality, idolatrous practices, fr. ON *hōrdōmr* adultery, fr. *hōrr*] **1** : the practice of whoring **2** : faithless, unworthy, or idolatrous practice or pursuits

whore·house \ˈhō(ə)r-ˌhau̇s, ˈhó(ə)r-\ *n* : a building in which prostitutes are available

whore·mas·ter \-ˌmas-tər\ *n* : a man consorting with whores or given to lechery

whore·mon·ger \-ˌməŋ-gər, -ˌmäŋ-\ *n*, *archaic* : WHOREMASTER

whore·son \ˈhōrs-ᵊn, ˈhórs-\ *n*, *often attrib* **1** : BASTARD **2** : coarse fellow — used as a generalized term of abuse

whor·ish \ˈhōr-ish, ˈhór-\ *adj* : of or resembling a whore : LEWD

whorl \ˈhwȯr(ə)l, ˈwȯr(ə)l, ˈ(h)wər(-ə)l\ *n* [ME *whorle*, *wharle*, prob. alter. of *whirle*, fr. *whirlen* to whirl] **1** : a drum-shaped section on the lower part of a spindle in spinning or weaving machinery serving as a pulley for the tape drive that rotates the spindle **2** : an arrangement of similar anatomical parts (as leaves) in a circle around a point on an axis **3** : something that whirls, coils, or spirals or whose form suggests such movement **4** : one of the turns of a univalve shell **5** : a fingerprint in which the central papillary ridges turn through at least one complete circle

whorled \ˈhwȯr(ə)ld, ˈwȯr(ə)ld, ˈ(h)wər(-ə)ld\ *adj* : having or arranged in whorls; *esp* : VERTICILLATE ⟨~ leaves⟩

whort \ˈhwərt, ˈwərt\ *or* **whor·tle** \ˈhwərt-ᵊl, ˈwərt-\ *n* : WHORTLEBERRY 1

whor·tle·ber·ry \ˈhwərt-ᵊl-ˌber-ē, ˈwərt-\ *n* [alter. of earlier *hurtleberry*, fr. ME *hurtilberye*, irreg. fr. OE *horte* whortleberry + ME *berye* berry] **1** : a European blueberry (*Vaccinium myrtillus*); *also* : its glaucous blackish edible berry **2** : BLUEBERRY 1

^{1}**whose** \(ˈ)hüz, üz\ *adj* [ME *whos*, gen. of *who*, *what*] : of or relating to

STATEMENT OF FIXED, MONTHLY EXPENSES
OF JAMES KELL, OWNER OF REAL PROPERTY AND IMPROVEMENTS
KNOWN AS GALLEY WEST CENTRE & MI K STO I
FOR PERIOD BEGINNING 11/30/82

Pmt. 1st Deed of Trust Note (interest only) (Calculated on est. average of 12%)	$2,400.00
Pmt. 2nd. Deed of Trust Note	1,906.87
Insurance:	
Through 2/22/83 (excluding finance charge)	1,207.50
Beginning 2/23/83	900.00
Prorated Real Estate Taxes	NOT AVAILABLE
Union Electric Line Extension Charges	1,447.00
Equipment/Fixtures Lease	2,516.55
Prorated Depreciation:	
Building	3,850.67
Fixtures and Equipment	172.00
Leasehold Improvements	116.42
Security System Lease & Monitoring Charges	30.00
Utilities	78.00
Telephone	172.00
Total Monthly Fixed Expenses	$13,589.51

STATEMENT OF FIXED, MONTHLY EXPENSES OF KELL INTERNATIONAL, INC., LESSEE, OF REAL PROPERTY IMPROVEMENTS KNOWN AS MILK STOP I FOR PERIOD BEGINNING 11/30/82

Rent	$650.00
Security System Lease & Monitoring Charges	30.00
Utilities	230.00
Telephone	68.00
Total Monthly Fixed Expenses	$978.00

South Town Auto Body West, Inc.
905 BLAKEY
TIMES BEACH, MO. 63025

938-5400

July 15, 1983

TO THE MEMBERS OF THE CONGRESS OF THE UNITED STATES

Ladies and Gentlemen:

I think the time has come for government agencies to consider how it's actions affect people. I refer to the quarantine of Times Beach after high levels of dioxin contamination were discovered there.

Our family business was in Times Beach. We started our business in 1959, in a building we leased, in south St. Louis. For twenty years, the income from that business provided us with a nice home, good education for our three children, a comfortable life style, and even a week-end home at a nearby lake. We were a classic example of the American Dream come true! Work hard, invest wisely, and save regularly.

But as the boys grew into men and entered the business, we decided that we needed something better for them. So, in 1979, we obtained a sizable loan, in order to build a modern shop, on a main thoroughfare (I-44) in Times Beach.

The first year was tough! A new location and a failing economy forced us to put $32,000.00 of our savings into the business. But, by August of 1981, we were able to start repaying our loan to the company at the rate of $250.00 a week. And 1982 was our best year since before 1973. Then, on December 5, 1982, a record-breaking flood filled our beautiful building with 8½ feet of water! We thought we were finished, but with the help of good friends and family, we re-opened, just 3 weeks later.

Three days later, the barricades to the city went up, warning people not to enter Times Beach. Our business was ended. 23 years of hard work, careful planning, and our legacy to our children was wiped out. We had no income, our sons had to find employment elsewhere. But the bills still kept coming. Loan payments on the building in Times Beach and on our home, utility bills, living expenses had to be paid. We've been using our savings just to stay alive! There's not much left. My husband is 52 and I am 50. How do we start over again? Our enthusiasm for life is gone. We feel that our lives have been destroyed.

I hope this letter will move you sufficiently to consider amending the Superfund Law, so that we can be reimbursed for the financial losses we have suffered, through no fault of our own. The last seven months have been a never-ending nightmare!

Sincerely,

Marianne Hrin

Marianne Hrin, Sec'y.-Treas.

Mr. James Curtis is the minister at the Full Gospel Tabernacle and also served as city water commissioner. He wrote us as follows:

Dear Sirs:

The following account is given to show some of the additional expenses that have been incurred since our 'voluntary decision' was made to move from Times Beach into government provided housing.

We are aware that dioxin is one of the most controversial issues of recent months and its effect on humans has been the subject of many debates. We are also aware that our only knowledge of dioxin comes from "experts" and therefore we must base our decisions on what the "experts" advise.

In this case the "experts" are the Centers for Disease Control, the Environmental Protection Agency, the Federal Emergency Management Agency, the Department of Natural Resources, etc., et al. Their advice was to move from our homes, not to even attempt to repair them from the flood of December 5, 1982. We aim to show that following that advice has proven to be quite a financial burden.

Some of these expenses are as follows:

A storage unit was rented to store some items we could not fit in the trailers cost $50.00 per month.

Since we have a large family we were provided with two trailers. These trailers were provided with a furnace for heat in winter but we had to provide our own air conditioning for summer. Installation required removal of a whole window unit, and the cost for both air conditioning units was $1200.

Having two trailers requires two billings on utilities. The following figures represent only the amount above a bill paid in Times Beach: Electric $60.00 per month. Water: $18.00 per month. Gas: $50.00 per month and phone $22.50 per month. (Service and equipment only.)

For the six months we have been in temporary housing these figures represent an additional cost of $2253.00.

Following the advice of "experts" to protect our health has not been cheap. I might add that all my income prior to the flood came from within Times Beach. Income from such business ventures as rental property, construction work, Times Beach water commissioner, etc., amounted to about $1200.00 per week. My income now is $105.00 per week from unemployment insurance. Losing my job with the City meant losing health insurance for my family.

A book on the story of our lives would never be salable to the public; the account is too unreal, too fabricated. But the horror of our day-to-day existence is very real.

Remarks made following the flood of December were centered around this thought, "In spite of what we have lost we still have our health".

Seven months later our thoughts echo that we have lost our homes, we have lost our friends of many years, we have lost our church, we have lost our jobs, we have lost our health insurance, we have lost our town, and we wonder if we have lost our health which will agonizingly reveal itself in the next few years or perhaps in the bodies of our children.

My personal goal was to retire this year and reap the benefits of my business. Now, my goal is simply: Make it 'till tomorrow.

Sincerely

(signed) James Curtis

Sherwood Glen Mobile Home Court and Easy Living Mobile Manor represents a total of 337 mobile home spaces and are owned by Mr. Charles Stone.

At the present time there are no lots rented in either park. Mr. Stone also operated a mobile home brokerage firm and a laundromat. Since the closing of the roads he has had no customers for the parks, the laundry or the brokerage firm which dealt in used mobile homes. Taxes, insurance and interest payments continue on as before. The interest on all these enterprizes amounts to over $11,000 per month. Each month he borrows this money.

Based on the fact that an announcement was made that Times Beach people were to be made "whole" again and also that checks would be in the mail by late June or early July, Mr. Stone has closed on property to be used for a new mobile home park. His interest payments are now an additional $6,000 per month.

The cost of building a new mobile home park vs. the cost of his former parks are incomplete at this time but it appears that costs have at least tripled. Zoning laws are more strict now than formerly. Land is not as available, nor is it as level. Escavation in the new location will cost about $250,000. Mr. Stone does not even qualify for the relocation expense for one of these parks.

Melba Perschbacher is the owner of two pieces of commercial property in Times Beach. Both properties were leased. Ten year leases with two 5 year options. Fair market value does not compensate for these leases which represented assured income for the future.

Again, mortgage payments must be made.

As one piece of property is a convenience store located across the street from the mobile home court it is the intent of the owner to relocate at the new mobile home court which is to be built, but it is unlikely that the same interest rate will apply to any new loan and the owner fears that the new buildings "fair market value" will not be the same as "replacement costs".

I would like to emphasize the need for speed in handling any future situations like the Times Beach buy out. The Times Beach buyout was to have been completed by late June. Mrs. Perschbacher's husband, Bill, died on May 13. He had been told by his doctor to avoid stress, yet he lived with the stress of Times Beach for over 6 months. Perhaps if more rapid progress had been made in the buy out, he, Al Kampen, and some of the other heart patients might still be with us. We cannot over-emphasize the strain under which we have all lived and the need to have this stress alleviated as speedily as possible.

Gerald J. Hatcher is the owner of Ill-Mo Bob Cat, an equipment house, renting and selling Bob Cat loaders and Yanmar tractors. The owner of this business advised us that he had received no aid of any kind from any agency. This includes government agencies as well as charitable organizations including the Red Cross and the Salvation Army. Mr. Hatcher says: "The authorities stated on December 23rd 'If you are in the city, get out; if you are out of the city, stay out'".

I moved my business out of the city and the expense of moving and locating in another area was paid by me. At this point the expense is well cover $27,000. The Relocation Brochure for Times Beach gives as top figure of $10,000 for relocation expenses. Mr. Hatcher further stated: "I am at present suffering financial injury because I was forced to move to another location. I am not now as accessible or as visable from the highways in my new location. My customers have not yet found me and some of those who have found me are afraid I will contaminate them".

Laine Jumper is a building contractor who lived in Times Beach. He received a $5,000 grant for replacement of personal belongings. This is the only assistance he has received.

Mr. Jumper has 6 building sites in Times Beach for which he will probably be offered $2000 each, yet the cheapest comparable lots in St. Louis County are in the $13,000 range.

Delay has cost 1½ years at a minimum. He cannot service old customers that are relocating from Times Beach as he has no capital. Small Business Administration loan was refused because he has no business and he cannot have a business without an SBA loan. He had a $30,000 yearly income from rental property and developing real estate. This was the result of working 14 hours per day for the last 5 years. He now feels he must start over and the favorable opportunities he enjoyed 5 years ago no longer exist. Times Beach was unique in that a skilled builder could do all his own wiring, plumbing, masonry, concrete working and all facets of building himself. Times Beach no longer exists. All other municipalities require licensed bonded tradesmen and this adds an additional $15,000 to $20,000 to the cost of a house.

Mr. Jumper has devoted a great deal of time to working and pushing for the buy out. He lobbied for this on State and Federal levels and emphasized that the buy-out include businesses and residences. Now that it is here they are buying out only real estate. He feels the bottom line is that promises made at State and Federal levels are being ignored and forgotten and that is a price most of us cannot afford to pay.

South Town Auto Body West, Inc.
905 BLAKEY
TIMES BEACH, MO. 63025

938-5400

July 15, 1983

TO THE MEMBERS OF THE CONGRESS OF THE UNITED STATES

Landies and Gentlemen:

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Sincerely,

(signed) Marianne Hrin
Marianne Hrin, Sec'y.-Treas.

Robert Fitts owned and rented 9 mobile homes. Only one of the mobile homes was sufficiently covered by insurance that it was paid off after the insurance settlement was received. Mr. Fitts was in the process of making necessary repairs and renovating the remaining eight mobile homes when the roads into Times Beach were barricaded. Now he has no renters for his homes although he has a mortgage payment to make on each of them. In some instances the lending agency is insisting on payments on principal and interest, others are offering some assistance by only requiring payment of interest.

This condition has existed for the past eight months. This is the usual situation. No help for the businessman. Some recognition of this type of hardship is needed.

Jim's Liquor Store is owned by Gary Waltman.

Under present Superfund Laws Mr. Waltman fails to qualify for assistance in all but one category.

Mr. Waltman purchased the liquor store for $50,000. He leased the building and he supported his family from the operation of this store until December 1982. When the streets and roads of Times Beach were sealed off and dioxin contamination caused the people of Times Beach to move elsewhere he had no customers. He then closed his business.

He has been told that the only Superfund or F.E.M.A. category he falls under is a relocation allowance. At best this appears to be $10,000. It can be as little as $2,500. These are the figures quoted in the Relocation Brochure for Times Beach, distributed on June 17, 1983.

Interest on the outstanding indebtedness of this business has continued for the past 8 months and he apparently does not qualify for any other part of the buyout. He has no other livelihood. Even if he gets the top limit of a $10,000 relocation allowance he is still out $40,000.

He had some money which he used as a downpayment on the Times Beach business, however, after the buyout he will not have enough money to buy into another business at someother location.

Twin Flags KOA
1380 South Outer Road
Times Beach, Missouri
July 15, 1983

Marilyn Leistner, Mayor
City of Times Beach
Times Beach, Missouri

Dear Mayor Leistner:

You are aware of the dilemma the businesses of Times Beach are in due to the health advisory issued by the Center for Disease Control and Environmental Protection Agency on December 23, 1982 because of the presence of dioxin along the shoulders of the city streets. We would like to share with you how this has affected the small business we own and our lives.

Both of us are former school teachers who invested our entire life savings in a campground franchise of Kampgrounds of America. We were greatly relieved upon the announcement of the buy-out of the residences and businesses of Times Beach because we were facing financial ruin. However, since the buy-out involves buying real estate only and not the business and provides no compensation for economic injury, we will have no income for 1983, therefore no return on our investment for at least one full year. Our income for 1984 and future years will also be reduced until our customers learn where we have relocated.

In addition to loss of income, we have had considerable expense for which there is no compensation. After the flood in December and before the health advisory to vacate the town was issued, we spent approximately $8,000.00 to clean up our campground for reopening. We have spent hundreds of dollars for storage of our equipment and trips to Jefferson City to attend meetings with state officials concerning the Times Beach situation.

In the five years since we bought the campground, after meeting operating expenses, we had reinvested all the income in improvements. We were owner-operators who spent twelve to eighteen hours a day, seven days a week in the operation of our campground. We had developed a very successful campground that operated at capacity for about ninety days a year. Because of this, we had purchased approximately one acre of adjacent land in September 1982 to expand the number of campsites by about twenty per cent. We have lost the potential of this increased income through no fault of our own.

It is impossible to describe the emotional trauma we have experienced in losing the business we have both worked so hard to develop, were so proud of and enjoyed so much. Much of our business was from repeat customers with whom we had developed friendships.

The slowness of the actions of the government agencies in responding to this crisis has added to our financial loss and also greatly increased the emotional stress we have had to endure.

President Reagan has on numerous occasions indicated his support of small business in the United States. We hope that with your urging, the Reagan administration will exhibit their sincerity in this support and compensate us for our loss of business and not just our real estate

We thank you for the time and effort you have spent on behalf of all the citizens of Times Beach and especially the business community.

Attached you will find a statement from our accountant listing our gross income on a monthly basis for the year 1982.

Sincerely,

J.C. Walters
Doris J. Walters

J. C. Walters
Doris J. Walters

Gary Const. Company
Residential • Farm • Commercial

General Contractor
Gary Overton
938-5407 - 587-6690
Rt. 2, Box 354
Eureka, MO 63025

June 21, 1983

Mr Ronald Reagan
1600 Pennsylvania Ave.,
Washington, D C 20460

Dear Mr President:

We are in a dilemma. We have been told that the businesses and business men would be compensated for our losses, and now we have found out that this is not the fact. Where do we turn now? We have suffered a great loss both monetary and mentally. The government has done well up until now and we give many thanks.

Please re-write the contract with the Federal, State, County, and City Goverments to include us and give some consideration to our needs which are:

Immediate assistance in relocating our businesses and in most instances the relocation allowances will not cover actual costs.

$10,000 cannot possibly compensate for the loss of 6 months or more income. In many instances the businessmen hired employees. Now unemployment compensation charges will be levied against the employer because he laid off his employees. His business no longer operates profitably because of a government ruling. The only acceptable solution to this problem is for the FEMA [illegible] to equitably compensate for the [illegible] of the governments actions.

Yours very truly,

Gary Overton

cc: Larry Zinsinger - FEMA - D C
Paul Ward - FEMA - St Louis
Senator John Danforth
Senator Thomas Eagleton
Senator Richard Gephardt
Congressman Robt Young
Governor Kit Bond

MISSOURI DEPARTMENT OF NATURAL RESOURCES
P.O. Box 176 Jefferson City, Missouri 65102 (314) 751-3332

MEMORANDUM

Date: August 17, 1982
To: Alden Shields
From: Fred A. Lafser
Subject:

Attached is a map indicating locations in Missouri where dioxin has been found. This is the result of a followup investigation that EPA has been doing in reference to the Verona dioxin situation. EPA, in tracking dioxin generated at the NEPACCO facility in Verona, estimates that approximately 153 pounds were generated. 110 pounds have been accounted for.

In 1971-72 Russell Bliss contracted with Nepacco to haul waste oil from their location. During that time period Russell Bliss (who also had contracts with stable owners) would take this waste oil from NEPACCO and transport to his contracted dusting operations and spray this on horse arenas and other dusty areas. This resulted in contamination of four horse arenas. That information will be released tomorrow morning. EPA has just received the documentation of the levels of contamination.

Material was sprayed during 1971 which applied 6 pounds of dioxin at the Shenandoah Stables. Soon after the spraying horses, cats, birds, and mice died. Personnel that were in contact with the material developed signs of dioxin poisoning. CDC recommended removal of the contaminated dirt. That contaminated dirt was removed and located at a fill on Highway 61 adjacent to the stables. The material is covered by Highway 61 and was unable to be sampled. After the original clear up occurred, organic odors were still noticed in the stable. A second clean up activity, again on the Shenandoah properties, took place. Samples of that material indicated 1700 parts per billion (ppb) 2,3,7,8 TCDD (dioxin). Sampling inside the existing horse arena showed levels in the soil surface of 75 to 110 ppb. Dust samples collected on the rafters and bleachers inside the stables indicated 100 ppb.

Another area where dioxin was sprayed was at Bubbling Springs Ranch near Fenton, Missouri. Again illnesses occurred and horses died. Again CDC recommended the material be removed. Unfortunately the materially hauled by

Christopher S. Bond Governor
Fred A. Lafser Director

truck loads at a fill area. This material is currently located at the Minker Residence and fill adjacent to the residence has washed out into Romaine Creek. Levels of dioxin found within ten feet of the Minker residence contained 660 ppb. Levels located in the washed out area between the house and going to Romaine Creek are 900 ppb. Another area that material was dumped from Bubbling Springs Ranch was Stout Trailer near Fenton. Soil sampled at depths of 30" indicated approximately 25 ppb.

The other areas that exist in the state are the Timberline Stables in New Bloomfield which again was strayed and again cleaned up by contractors. Samples of the residual soil showed 53 ppb in the soil and 43 ppb in the dust inside the arena. The contaminated Timberline soil was removed and deposited in the old Jefferson City Landfill. Samples did not show any new detectable amounts in runoff from the landfill.

Bliss Farm in Rosati is also another area where Bliss decreased his load after being overweight. His farm road has shown concentrations of 50 ppb.

Some of this information will be released tomorrow by the EPA. EPA is currently getting some calls on the status of the investigation. We had a meeting with EPA yesterday. Present was Dr. Renate Kimbrough, a dioxin expert, and her assessment of the situation is that any level of dioxin above 1 ppb in these types of situations need to be addressed. She also indicated a significant concern for the accessiblity of these sites to the general public and felt it would be appropriate to limit accessibility if at all possible. There are no standards that can be identified for making this health judgement, and we feel we have to go on the recommendations of experts from the CDC.

EPA will be releasing some information tomorrow indicating the fact that they did find the dioxin and are recommending that these sites have limited access. They are currently looking into the legal options on how they may proceed in taking control of these sites and to clean up (if possible).

DNR is currently acting in a support role in supplying EPA with technical information pertaining to their activities and assisting them in coordinating cleaning up these sites.

In reality, the dioxin announcement will ruin any business that exists at the Timberline and Shenandoah stables and will, in effect, limit public access. EPA and the state's recommendation on the Minker residence is to take as fast action as possible to cap the material and prevent erosion until a disposal option can be developed. Pat Phillips of the Division of Health and Scott Richie of the EPA will be personally meeting with the residents around the Minker site today and informing them of the data and interpreting that data as to how it may affect them. They will also be recommending that they avoid the contaminated area.

To put this into perspective, the levels that were sampled of 25 to 900 ppb should be compared to 37 ppb that existed at Sturgeon where millions of dollars of lawsuits are being won by the workers that were exposed in the cleanup operation; 10 ppb at Seveso, Italy where 730 residents were evacuated and 50 ppt (parts per trillion) in fish where CDC recommends non-consumption of those fish.

Dioxin is a very long lived chemical and will not go away in the near future. There have not been any deaths to our knowledge - but we would not expect such. Dioxin acts as a toxin, carcinogen, mutagen, and fetal toxin and we would not expect to see that many immediate effects.

cc; Chip Casteel

FOR OFFICIAL USE ONLY

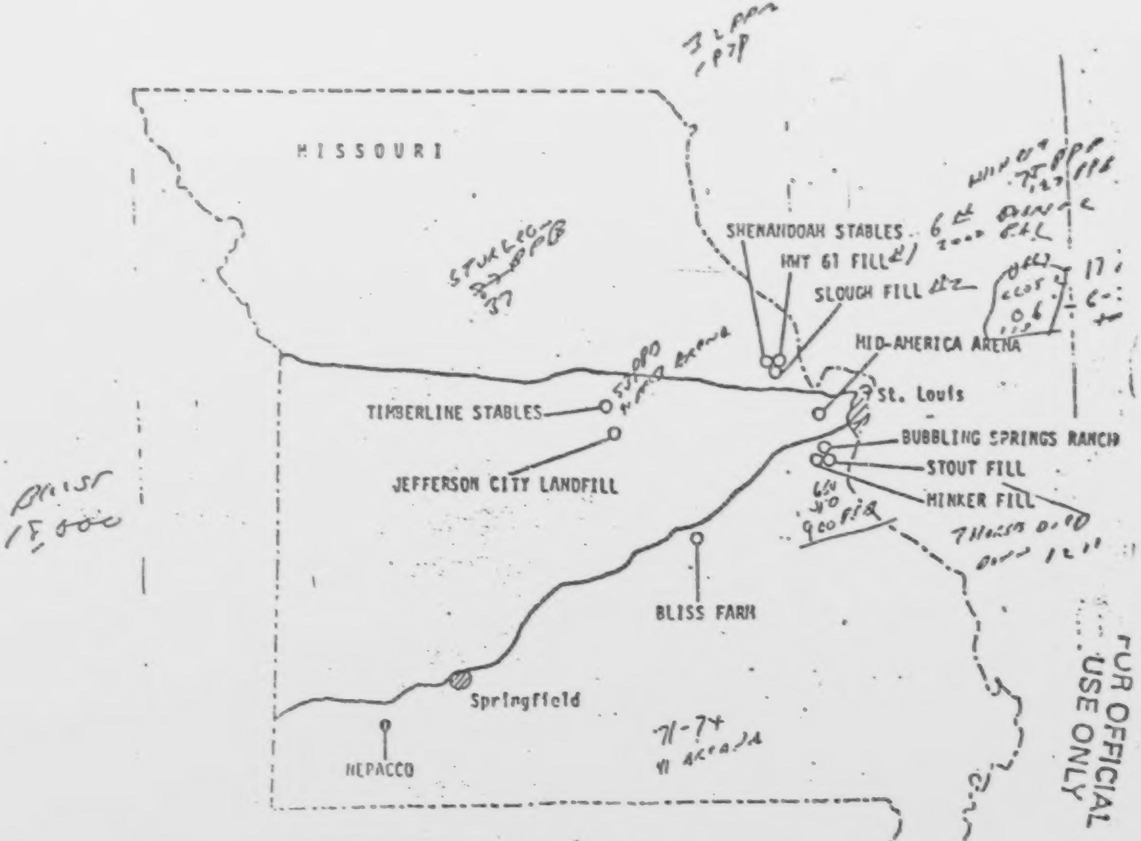

MISSOURI
SHENANDOAH STABLES
HWY 61 FILL
SLOUGH FILL
MID-AMERICA ARENA
St. Louis
BUBBLING SPRINGS RANCH
STOUT FILL
MINKER FILL
TIMBERLINE STABLES
JEFFERSON CITY LANDFILL
BLISS FARM
Springfield
NEPACCO
71-74
15,000

Figure 1 - Location Map, Horse Arenas and Secondary Sites

E. A. MUNYAN, M.D.
17281 EAST 23rd STREET
MEDICAL PLAZA BUILDING
INDEPENDENCE, MISSOURI 64057

Telephone 373-1122

Jan. 7, 1983

Mr. Dan Maxwell
235 Grove Rd.
Times Beach, MO 63025

Dear Mr. Maxwell:

There has been a considerable amount of reporting on the ABC News Network in the past few months regarding the dangers of the so-called poisonous drug DIOXIN. The United Press Press International (U.P.I.) has been instrumental in bringing these "dangers" to the attention of the general public in the multiple articles it sends to member newspapers. Obviously the ABC is doing the same.

Articles by the news media suggest that Dioxin causes cancer, liver disease, degenerative diseases, miscarriage, birth defects and nerve damage.

The Council on Scientific Affairs of the American Medical Association (AMA), reported on the health effects of Agent Orange and Dioxin contaminants in the October 15, 1982 issue of the JAMA. They stated that Dioxin (TCDD) "is a contaminant", not a "key ingredient" of Agent Orange as reported in the UPI article. A copy of the AMA report is attached.

For 30 years mixtures of chemicals abreviated 2,4-D and 2,4,5-T have been used extensively by home-owners and farmers for ridding lawns and agricultural acreage of unwanted broadleaf vegetation. Similar spray programs have been used in the United States as a means of forestry management. Agent Orange or Herbicide Orange, was the label given by the U.S. military forces to a 50:50 mixture consisting mainly of 2,4-D, 2,4,5-T and varying amounts of the contaminant "Dioxin". These agents were sprayed over certain areas of Vietnam for the express purpose of defoliating the jungle and destroying one the enemy's means of concealment. Also used were Agents Green, Pink, Purple, Herbicide Orange II, Herbicide White, and Herbicide Blue.

Large numbers of persons have been exposed to these herbicides over the past 30 years, in the normal course of employment, in industrial accidents, and of course,in Vietnam. A number of law suits and government regulatory responses are now pending, based on alleged adverse health effects from such exposures. The AMA Council on Scientific Affairs Report states that "There is still little substantive evidence for the many claims that have been made against these compounds".

The most pronounced biological effect of "Dioxin" is its tendency to cause a form of acne called chlor-acne.

The most extensive human experience with the adverse effects of TCDD in man involved the residents of Sevesco, Italy, in July, 1976, when TCDD was accidently released from a chemical plant and a plume of reactor contents rose approximately 60 feet above the factory. More than 37,000 persons were potentially exposed to varying doses.

Two years after the incident occurred, the health effects were assessed. The mild chlor-acne, which occurred mainly in a small group of children, healed quickly. Asymptomatic nerve impairment was reported and there was some liver involvement, but without apparent functional disorder.

The most recent progress report on the residents of the Sevesco area emphasizes the preliminary nature of their findings, but states that except for the skin, no organs or body functions were impaired, no derangement of gestation, no fetal death, no gross malformations, no growth retardation at term, and no cell abnormalities have yet occurred (8 years after the exposure).

So what are the news media, the Environmental Protection Agency (EPA), and the environmentalists so excited about? Is the report of the AMA Council on Scientific Affairs in error regarding the lack of damage that has been done by Dioxin, or are the UPI & ABC reports gross exaggerations of potential dangers? Shouldn't responsible newspapers and radio networks check into this matter? Perhaps you should report to the public what the Council on Scientific Affairs said in its Council Report.

I urge you to look into this matter and check with the AMA Council on Scientific Affairs before any official action is taken in the so-called contaminated areas. Also please try to stop the mis-information.

Very Truly Yours,

E. A. Munyan

E. A. Munyan, M.D.

cc: United Press International
American Broadcasting Company
News Network

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
ATLANTA, GEORGIA 30333
TELEPHONE (404) 633-3311

October 2, 1974

H. Denny Donnell, Jr., M.D.
Division of Health
Missouri Department of
  Health and Welfare
State Office Building
Jefferson City, Missouri 65101

Dear Dr. Donnell:

As you know, the investigation being conducted by the Missouri Department of Health and the Center for Disease Control (CDC) concerning the contamination of horse arena soil by 2,3,7,8-tetrachlorodibenzodioxin (TCDD), has identified a source of distillate residues containing very high concentrations of TCDD. Preliminary toxicologic analyses of these residues here at CDC indicate concentrations of TCDD in the range of 1000 ppm. For the past 3 years, these residues have remained untouched in a storage tank on the premises of Hoffman-Taff of Verona, Missouri, a subsidiary of Syntex Agribusiness, Incorporated. Because of the extreme toxicity of TCDD, we feel that it is important for the public safety that this material be promptly and safely destroyed.

TCDD is a very stable compound which requires incineration at temperatures exceeding 800 degrees centigrade at exposure times exceeding 30 seconds for proper disposal. This incineration should be performed in a properly constructed incinerator capable of achieving these conditions. We recommend utilizing this means of disposal through a reputable service company capable of handling this material in the safest possible manner. Any company contracting for this service should be clearly informed of the nature, concentration, and toxicity of the material in question. Prior to disposal, we would advise that they consult with the Dow Chemical Co., Midland, Michigan, which has had substantial experience in disposing of TCDD-contaminated waste.

Once as much as possible of the tank's contents has been safely and properly removed, we feel that the tank itself should be sealed and deep-buried at a site remote from drainage systems and other potential sources of human exposure and where subsequent unearthing will not occur. Prior to burial, the tank should be carefully sealed and properly marked with respect to its residual contents and toxicity. Other means of disposing of the tank do not preclude the possibility of human exposure either immediately or at some distant point in time. In particular, since TCDD is relatively insoluble and since the residue is a thick, tarry substance, cleaning or decontamination of [illegible]nk does not seem to be a realistic alternative to deep-burial. T[illegible]iate surroundings of the storage tank should be inspected after [illegible]al, and concrete, pipes, or other objects that appear contaminated wit[illegible] esidue should be dealt with in a manner similar to that described f[illegible]k.

[illegible] is not a volatile compound, it can be highly toxic by skin [illegible] Its toxicity cannot be overemphasized, particularly since [illegible]ation in the residue in question appears to be extremely high. [illegible]refore very concerned that all persons involved in the removal of [illegible]aste material be properly informed as to its hazard and that adequate s[illegible] be provided before disposal is attempted. We would appreciate your in[illegible]rming us of any action taken to effect disposal. As you know, the invest[illegible]on in collaboration with your Division is continuing; as in the [illegible]t, we will stay in close contact with you regarding further developments.

Sincerely yours,

Clark W. Heath Jr.

Clark W. Heath, Jr., M.D., Director
Cancer and Birth Defects Division
Bureau of Epidemiology

[illegible] Herbert R. Domke
[illegible]r. Patrick Phillips
Mr. Godfrey J. Moll
Mr. Edwin B. Michaels
Dr. Glen E. Schweitzer
Dr. Gene Wallen
Mr. William F. Barthel
[illegible]r. Philip Landrigan
Renate Kimbrough
[illegible] David Firestone
Dr. Coleman D. Carter
Dr. Philip Brachman

Times Beach Chronology: 2 Months Of News About A Troubled City

By Daniel O'Neill
Journal Staff Writer

It has been nearly two months since news first circulated that Times Beach was one of many sites in Missouri being investigated by the Environmental Protection Agency (EPA) for possible dioxin contamination.

In that time, the city has absorbed a cruel combination of natural and man-made disasters that threaten its very existence and have made it the source of local and national news almost daily.

The news has been sometimes confusing, sometimes repetitive and often conflicting. The following is a chonological look at the recent events that have affected Times Beach as they were reported in the local media.

WEDNESDAY, NOV. 10

The *Journal* reports that a list compiled by the EPA reveals Times Beach is among sites to be investigated for possible dioxin contamination. The list, which was not intended to be publicized, includes Times Beach among areas sprayed by an Ellisville waste oil dealer, Russell Bliss, who is known to have purchased waste-oil laced with dioxin.

When contacted by a Times Beach official, Bliss assures the city that no dioxin was in the oil he used to spray Times Beach streets.

WEDNESDAY NOV. 17

The *Journal* reports that an EPA spokesman has notified Times Beach officials that the town is high on the agency's priority list to be investigated for possible dioxin-contamination.

In making the announcement of the notification, Times Beach Mayor Joe Capstick says, "They don't know exactly who has it, but they have to assume that it's possible we have it."

WEDNESDAY, NOV. 24

Local daily newspapers report that federal health and environmental officials considered steps to seal off dioxin-contaminated areas in the state as early as last summer. Plans included evacuation of residents in areas of Jefferson County.

Dioxin was generated from a pharmaceutical plant in Verona, Mo. as an unwanted byproduct in the making of other chemical products such as disinfectants.

Dr. Renate Kimbrough, a spokesman for the U.S. Centers for Disease Control (CDC) in Atlanta, advises that concentrations of dioxin above 1 part per billion parts of soil are, in her opinion, cause for immediate action.

TUESDAY, NOV. 30

The EPA begins testing streets in Times Beach. It is also reported that more than 100,000 gallons of waste oil that may have been contaminated with dioxin were sprayed on most of the streets in Times Beach by Russell Bliss in 1972.

Some Beach residents remember the spraying and recall such things as streets turning purple, a funny smell like paint thinner and birds leaving the area.

WEDNESDAY, DEC. 1

Residents of Times Beach plan a fund-raising drive to hire Envirodyne Engineers Inc., a private firm, to test for dioxin.

Times Beach resident Laine Jumper said "Residents want to know right now whether the dioxin is here. We appreciate what the EPA is doing but they're not fast enough."

EPA officials say that if dioxin is found in the first tests, more tests will have to be taken.

THURSDAY, DEC. 2

An expert on dioxin says residents of contaminated areas need not worry about health effects unless they develop chloracne, a disfiguring skin ailment. Robert A. Neal, president of the Chemical Industry Institute of Toxicology - an industry financed institute that studies toxicological concerns related to the manufacture, use and disposal of chemicals - tells Beach residents that there is "no evidence that dioxin caused long-term health problems."

"If there is no chloracne," Neal said, "one can be reasonably assured that serious illness from exposure is not resulting."

The same day, Russell Bliss says that half the dioxin-contaminated oil he possessed was sprayed on his farm in Rosati, Mo. and at his three horse arenas in eastern Missouri. Bliss says the remainder of the oil was burned as heating oil. "I hope my soul rots in hell if I'm lying," Bliss adds. "It looked like good oil to me."

Health officials describe the form of dioxin produced by the company in Verona - Northeastern Pharmaceutical Co. - as the most poisonous of the 75 varieties of dioxin. Officials call it 150,000 times more toxic than cyanide.

SATURDAY, DEC. 4

Week-long rains continue and officials begin warning of possible high flood waters. Civil Preparedness advises residents of Times Beach to gather their belongings and evacuate the town before flood waters hit. Most residents don't believe the Meramec River will reach the predicted heights and few pay any attention to the warnings.

SUNDAY, DEC. 5

The Meramec River overflows its banks and rages through the area, completely inundating Times Beach. Waters rise up to four feet in one hour and reach record levels. Most people are caught by surprise in Times Beach and virtually every home is damaged or destroyed.

Jef County Journal 1/13/83

WEDNESDAY, DEC. 15

Preliminary tests done by Envirodyne Inc. show that a section of Times Beach City Park contains at least 10 different toxic-waste chemicals. The test was done after Envirodyne was hired by a citizens' group to test other soil samples in the city. Representatives of the company decided to test the park soil after smelling a curious odor in the area.

FRIDAY, DEC. 17

EPA officials tell residents not to jump to conclusions about how much of the town is contaminated with dioxin. However, Art Spratlin, deputy director of air and hazardous material programs at the EPA regional office in Kansas City, expresses fears that the EPA and the city may face an unprecedented cleanup. Spratlin says the "potential is mind-boggling."

Test results by Envirodyne show levels of dioxin up to 16 parts per billion in Times Beach, exceeding the danger level of one part per billion.

TUESDAY, DEC. 21

The EPA announces that three toxic waste sites in Missouri are on its priority list of sites nationwide. The areas are the Shenandoah Stables at Moscow Mills, the Syntex facility in Verona and the Stout and Minker residential areas near Meramec Heights in Jefferson County.

SUNDAY, DEC. 26

The EPA announces that it plans to begin tests in Times Beach to determine how the recent flooding affected contamination in areas of the community. The agency also recommends that residents in Times Beach, driven from their homes by the flood, stay away and that those who have returned to their homes, leave. The recommendation comes after results of preliminary tests by the agency reveal levels of contamination in excess of 100 parts per billion.

Meanwhile, Mayor Joseph Capstick resigns and Alderman Sidney Hammer is named acting mayor.

TUESDAY, DEC. 28

Former mayor Capstick says he believes the EPA and health officials are overreacting to the problem. Many residents voice similar sentiments as they return to their damaged homes. Some flood victims in the area begin receiving disaster relief checks from the government.

Acting mayor Hammer accuses federal authorities of acting too slowly on the dioxin question. Hammer says, "The EPA must think we're a bunch of jerks and country bumpkins." A spokesman for the EPA indicates his office is working as quickly as possible and promises to "pull out all the stops" to solve the problem.

WEDNESDAY, DEC. 29

Nearly 1,000 people turn out at Eureka Junior High School for a hearing held to discuss health problems. They voice anger and criticism at the way state and federal authorities have handled the situation.

State Rep. Bob Feigenbaum tells the crowd he will introduce a bill that will create a state "Superfund" to help clean up the waste.

The Federal Emergency Management Agency (FEMA) announces it will bring a number of mobile homes to the area for those driven from their homes.

THURSDAY, DEC. 30

Despite the possibility of dioxin contamination, Pat Breheny, regional director of FEMA, says officials have decided to haul flood debris from Times Beach because leaving it strewn about the city poses an immediate hazard.

President Reagan orders federal agencies - EPA and FEMA - to give him a report on the Times Beach situation.

FRIDAY, DE

Results of additional soil tests, taken before the flooding, show more contamination. The EPA releases results indicating dioxin levels in several streets and they announce plans to do more testing. Officials also plan to examine residents for correlations between health problems and dioxin contamination.

Meanwhile, plans to remove flood debris are halted until new tests show whether the debris is contaminated.

A St. Louis paper reports that the county police refuse to patrol Times Beach because of the dioxin. City officials had asked the county for police assistance after having to release four city policemen because there was no insurance to cover them.

SUNDAY, JAN. 2

A spokesman for the White House says the president has been briefed on Times Beach and is "continuing to look at the situation."

The state decides to send mental health workers to the city to help counsel residents and help them cope with the strain of the situaton.

WEDNESDAY, JAN. 5

The U.S. Army Corps of Engineers proposes to move the entire town of Times Beach and build a new town on higher ground. U.S. Rep. Robert Young, whose district includes Times Beach, supports the proposal enthusiastically.

Hammer says, "I think it inks. It's one of the dumbest eas and most ridiculous ideas e ever heard. I'm prepared to ht it every inch of the way."

tate natural resources officials suggest that the flood debris be moved to a sanitary landfill and local authorities meet to decide where to take the waste

THURSDAY, JAN. 6

Hammer announces that he could support a proposal by the Corps and Young to build a new town. "Ninety-nine percent of the people would support it if they give us something comparable to what we had," says Hammer. The acting mayor also indicates that the removal of flood debris will begin the next day. The trash is to be hauled to Bob's Home Service, a toxic-waste landfill in Warren County.

Meanwhile, tests show that the highest concentration of dioxin is in a residential area just west and north of the center of town. Test samples that had shown levels above 100 parts per billion were taken from the area on the northeast corner of the city park.

U.S. Sens. Thomas Eagleton and John Danforth call for federal hearings on the dioxin problem and say the "EPA is still dragging its feet in getting the testing teams assembled and on the job."

The previous night, nearly 500 residents met at Eureka Junior High School and vented their anger at delays in the cleanup and the uncertainty surrounding the dioxin. At the meeting Hammer displayed a large map from the EPA depicting the wide extent of the dioxin contamination. The map, derived from tests taken before the flood, shows that most of the city's road beds are dioxin-contaminated with levels ranging from below five parts per billion to more than 100 parts.

Residents of Wright City, where Bob's Home Service is located, express uneasiness about accepting debris from Times Beach at the dump site.

FRIDAY, JAN. 7

President Ronald Reagan orders the formation of a special task force to coordinate flood and dioxin cleanup in Times Beach. Missouri Gov. Christopher "Kit" Bond sent a letter to Reagan stating "I know of no other community that has experienced this magnitude of simultaneous trauma."

The EPA raises questions about toxic levels in flood debris, forcing the state to delay plans to move the debris to a landfill.

Meanwhile, officials in Warren County threaten to go to court to block dumping of the waste in Wright City.

SUNDAY, JAN. 9

Members of Reagan's task force arrive in Times Beach, along with Bond, spurring the start of the long-awaited debris clean up.

Officials order that the debris be loaded into trucks and transported to the hazardous waste landfill in Wright City. Hammer says, "Finally, something is starting to happen" while cautioning that citizens will "take matters into their own hands if the cleanup doesn't continue."

Hammer also makes a plea for the president to come to Times Beach to view the situation first-hand, adding: "If we can't get help from this country, we'll go to another nation for help."

Officials of Warren County obtain a court order from Circuit Court Judge Edward B. Hodge of Warrenton blocking the transfer of the debris to Wright City.

MONDAY, JAN. 10

Tractor-trailer trucks are loaded with flood debris and await the go-ahead to begin transporting the possibly dioxin-contaminated cargo out of Times Beach. The transfer has been temporarily stopped by Judge Hodge's restraining order. State officials say they will try to dissolve the order.

TUESDAY, JAN. 11

Warren County officials are told to post $75,000 bond by 9 p.m. if they wants to maintain the temporary court order blocking the transfer of debris. The restraining order will expire if the bond is not posted. A dozen trucks, piled with debris, wait in Times Beach.

Residents of Times Beach collect more than 500 signatures on a petition asking the president to affect a federal buy-out of their flood-ravaged, dioxin-contaminated town.

WEDNESDAY, JAN. 12

The restraining order blocking the transfer of flood debris from Times Beach to Bob's Home Service in Wright City is lifted after Warren County residents fail to post the necessary bond to maintain the court, order. Trucks loaded with trash begin rolling out of Times Beach by 1 p.m. Tests results show no harmful levels of dioxin in the flood debris.

THURSDAY, JAN. 13

More than 100 people sign a letter asking foreign countries for help in the crisis. Times Beach alderman Harold Goodmon says, "If it will help, the people will sign anything. We're that desperate. We'll declare ourselves a foreign country." Letter organizers say that copies of the letter will be sent to the governments of Sweden, Great Britain, Japan, Australia, Belgium, Canada, Norway, France and the Netherlands.

SATURDAY, JAN. 15

Residents of Times Beach and Warren and Jefferson counties join forces to protest and draw attention to the dioxin problem with a demonstration on I-44. Organizers expect protestors to number more than 1,000.

Eureka Fire Protection District
Of St. Louis County
131 S. CENTRAL P. O. BOX 97 EUREKA, MISSOURI 63025
(314) 938-5505

February 10, 1983

Mayor Sid Hammer
City of Times Beach
Times Beach, Missouri 63025

Dear Sir:

The following directive has been issued to our personnel:

When answering alarms in Times Beach, Absolutely no personnel will enter or approach any abandoned structure which is on fire. Protection exposures will take priority over the involved structure, unless otherwise instructed by the Duty Officer.

No personnel or equipment will enter any area which has been corded off, due to high hazzard, unless it is a life threatening matter. Protective clothing and breathing apparatus will be worn on all fires and incidents in which a high concentration of dust is present. All equipment will be cleaned thoroughly prior to leaving Times Beach and individuals are instructed to take a shower immediately after returning to quarters.

No equipment will enter Times Beach until advised to do so by the Duty Officer. Staging area for equipment will be advised by Duty Officer, if necessary. Up-dates on this matter will be posted as they are received from the E.P.A..

Chief Joseph P. Trower
Joseph P. Trower
Chief of Department

c.c.: Mrs. Leistner
Connie LaCrone
file

United States Senate
COMMITTEE ON APPROPRIATIONS
WASHINGTON, D.C. 20510

July 13, 1983

Marilyn Leistner
Mayor
City of Times Beach
235 Grove Road
Times Beach, Missouri 63025

Dear Mayor Leistner:

Thank you for writing to Senator Eagleton regarding the problems being experienced by business persons in Times Beach.

I have attached some information regarding Senator Eagleton's efforts to change the Superfund Act to further aid businesses with problems such as you outlined in your letter.

I would be glad to meet with you or any of the business persons in Times Beach to further discuss this problem.

Sincerely,

Mark Van Matre

Mark Van Matre, Staff Asst.
SENATOR THOMAS F. EAGLETON
Old Post Office
815 Olive, Room 224
St. Louis, Missouri 63101

MVM/agv

Attachment

cc: Marianne Hrin
Gary Waltman
Charles Stone
Melba Perschbacher
Gerald J. Hatcher
Gary Overton
Doris Walters
Paul Benson
Wayde S. Dake
James F. Curtis

Senator Tom Eagleton

FRANKLY SPEAKING

WASHINGTON, D.C.

FOR RELEASE
JUNE 20-24, 1983

SOME VICTORIES IN THE FIGHT AGAINST DIOXIN

There has been a lot of news coverage recently about the chemical by-product dioxin; so much so that dioxin is almost becoming a household word. This is a tragic situation, but I am doing all in my power to see that this chemical does not become a Missouri way of life, and that all victims are completely helped.

The spraying of contaminated waste oil that happened statewide in the early 1970's has disrupted the lives of many Missourians. Unfortunately, dioxin is quickly becoming a national nightmare, and it's time that strong measures are taken to ensure that as few people are affected as possible.

In an attempt to improve the situation at one site, the federal governmen under the Environmental Protection Agency's Superfund Act has recently agreed to buy out Times Beach and relocate all the residents of that area. This action is of crucial importance; for this is the first step in helping those who otherwise would lose nearly everything.

As an improvement to this act, I have introduced a bill with Senator Danforth that would further aid victims or businesses that might be forced to close under a health advisory. The Superfund Act currently provides only for the payment of temporary relocation housing for those individuals removed from a hazardous waste area. Our bill would also assume the payment of principal and interest on the business debt of those businesses caught in the evacuation area.

Another matter of crucial importance is the investigation of how, and in what amounts, dioxin affects the human body. In a recent action, the Senate Appropriations Committee earmarked $6 million for investigation of the health consequences of exposure to dioxin and similar chemicals, and for soil research.

In addition, the Committee also adopted language I proposed which will make available the funds needed for medical testing and monitoring of workers who were exposed to dioxin at three St. Louis truck freight yards.

Finally, the town of Sturgeon, Missouri is another site which needs further investigation. The EPA initially did a cleanup there in 1979 after a dioxin spill. Research done immediately after the cleanup showed that no distinguishable amounts of dioxin remained. However, concern has resurfaced that dioxin may still be present in the environment. I have urged EPA to return to Sturgeon immediately to renew the investigation.

Dioxin pollution is a crisis as severe as any that could come from a natural disaster. I am going to see that it is treated as such. All Missourians, whether in Times Beach or at the St. Louis truck yards, can be assured that their health will remain of utmost concern to me.

Our state must remain a safe and beautiful place to live.

STATEMENT BY SENATOR THOMAS F. EAGLETON BEFORE THE ENVIRONMENTAL POLLUTION SUBCOMMITTEE

WEDNESDAY, JUNE 8, 1983

HELP DIOXIN VICTIMS

Mr. Chairman, I am pleased to provide the Subcommittee on Environmental Pollution with my thoughts on S. 1412, a bill to authorize community relocation and business protection in case of toxic substances contamination.

By the most unfortunate set of circumstances, Senator Danforth and I have become somewhat expert on the intricacies of the Superfund Act (Comprehensive Environmental Response Compensation and Liability Act).

It has been a painstaking and frustrating process to watch the Superfund Act be put to its first practical test in meeting a large toxic substances emergency.

As the Subcommittee knows, Missouri has a dioxin crisis on its hands. Currently, there are 31 confirmed sites. An additional 102 sites have been under investigation. Luckily, 75 have been cleared, but new sites are called in for investigation on a weekly basis.

All of the poisoned earth is a result of a hauler of salvage oil, Russell Bliss, who sprayed waste oil to keep down the dust. It is a common practice in many rural areas. The waste oil was spiked with a sludge that Mr. Bliss had been paid a fee to remove from the Northeastern Pharmaceutical and Chemical Company. The spraying occured on city streets, (Times Beach); in trailer parks (Quail Run Park); in residents' back yards (Minker-Stout homes); on stable runs (Shenandoah Stables); in commercial businesses (Baxter-Nurseries); and sometimes truck freight yards (Hall Street Truck Sites). The result is that many of our Missouri residents are innocent victims of toxic dioxin spraying.

These Missouri residents are facing health tests and concerns, some face relocation from their homes, some are having to permanently relocate their families to new homes, and one whole town is being abandoned altogether. All of this is through no fault of the residents.

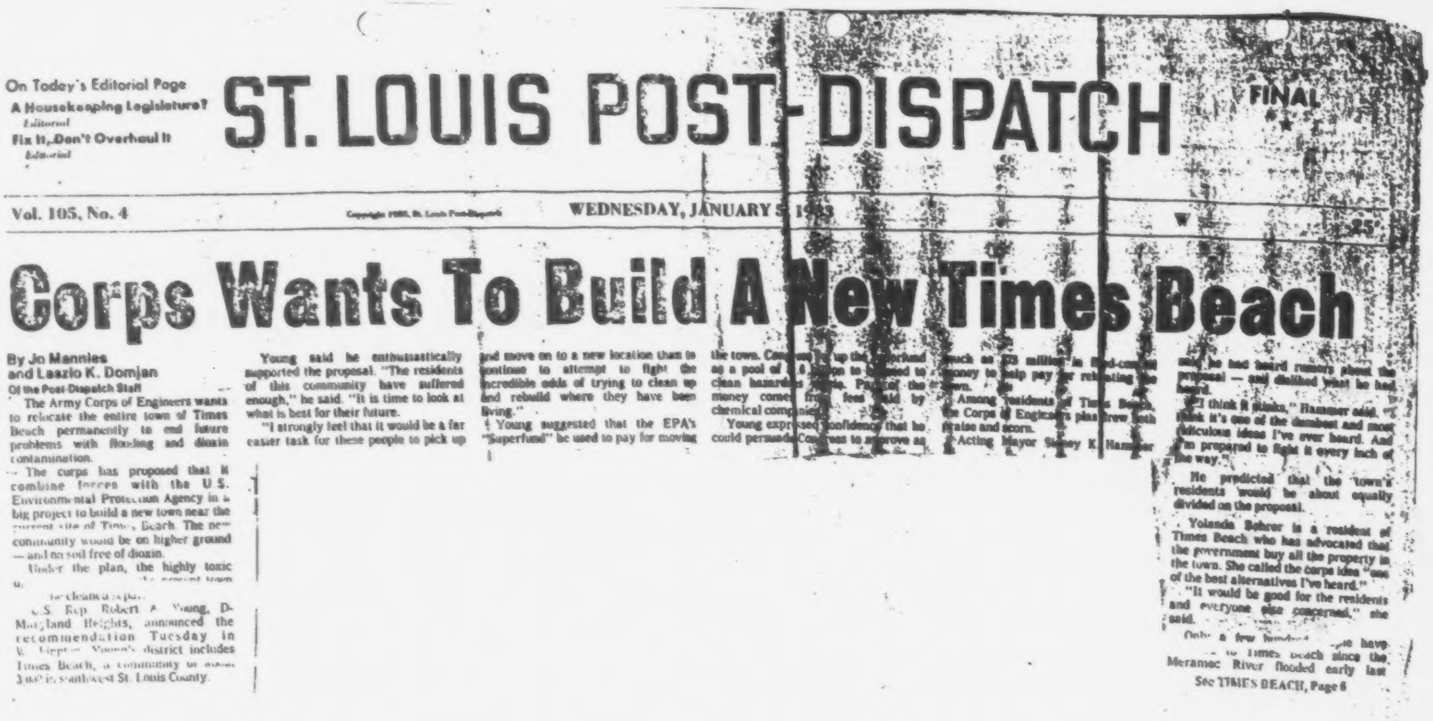
The bill that Senator Danforth and I have sponsored is an effort to assist victims of toxic waste sites, who are advised to relocate because of the imminent danger. The Superfund Act currently provides for the payment of temporary relocation housing for individuals removed from a hazardous waste area. It does nothing to further aid the victims or businesses, which may be forced to close under the health advisory. This bill addresses this gap in the Superfund Act.

For instance, in Times Beach, Missouri, 30 businesses were forced to close their doors when the news of the dioxin hit. Several months went by before the Environmental Protection Agency reached its decision to buy out the town. In those interim months, businesses were almost forced into bankruptcy because they were unable to pay the bare minimum expenses needed to remain solvent. In some cases, individuals not only were told to move from their homes, but the businesses they worked for were also closed, leaving them without means to support their families. Victims of natural disasters caught in this emergency condition are provided for under the Disaster Relief Act. This bill provides the same kind of assistance for victims of man-made disasters.

Secondly, the bill will provide payment of principal and interest on business debt for those businesses that are caught in the evacuation area. It seems only fair that we try and assist these people in this interim period, so that they have a chance to rebuild their businesses and lives at the end of the disaster.

I am pleased that the Committee has agreed to hear the testimony on this bill today. It shows the commitment that the Senate Environment and Public Works Committee has shown in helping to assure that the Superfund Act is administered in the manner Congress intended when it passed the bill in 1980.

Last week, New Jersey found dioxin sites in Newark. Next week it could be another state. I feel that this bill will help ease the pain of many of our Americans who are instantly caught in the nightmarish experience of our hazardous waste legacy.



On Today's Editorial Page
A Housekeeping Legislature?
Editorial
Fix It, Don't Overhaul It
Editorial

ST. LOUIS POST-DISPATCH

FINAL

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Corps Wants To Build A New Times Beach

By Jo Mannies
and Laszlo K. Domjan
Of the Post-Dispatch Staff

The Army Corps of Engineers wants to relocate the entire town of Times Beach permanently to end future problems with flooding and dioxin contamination.

The corps has proposed that it combine forces with the U.S. Environmental Protection Agency in a big project to build a new town near the current site of Times Beach. The new community would be on higher ground — and on soil free of dioxin.

Under the plan, the highly toxic [illegible] the present town [illegible] be cleaned up.

U.S. Rep. Robert A. Young, D-Maryland Heights, announced the recommendation Tuesday in [illegible]. Young's district includes Times Beach, a community of about [illegible] in southwest St. Louis County.

Young said he enthusiastically supported the proposal. "The residents of this community have suffered enough," he said. "It is time to look at what is best for their future.

"I strongly feel that it would be a far easier task for these people to pick up and move on to a new location than to continue to attempt to fight the incredible odds of trying to clean up and rebuild where they have been living."

Young suggested that the EPA's "Superfund" be used to pay for moving the town. Congress [illegible] up the Superfund as a pool of $1.6 billion to be used to clean hazardous waste. Part of the money comes from fees paid by chemical companies.

Young expressed confidence that he could persuade Congress to approve as much as $[illegible] million in [illegible] money to help pay for relocating the town.

Among residents of Times Beach, the Corps of Engineers plan drew both praise and scorn.

Acting Mayor Sidney K. Hammer said he had heard rumors about the proposal — and disliked what he had heard.

"I think it stinks," Hammer said. "I think it's one of the dumbest and most ridiculous ideas I've ever heard. And I'm prepared to fight it every inch of the way."

He predicted that the town's residents would be about equally divided on the proposal.

Yolanda Bohrer is a resident of Times Beach who has advocated that the government buy all the property in the town. She called the corps idea "one of the best alternatives I've heard."

"It would be good for the residents and everyone else concerned," she said.

Only a few hundred [illegible] have [illegible] to Times Beach since the Meramec River flooded early last

See TIMES BEACH, Page 6

FROM PAGE ONE

[illegible], covering all but one building.

And before the flood, Times Beach had problems with dioxin contamination. The EPA took soil samples from Times Beach just days before the flooding and found dioxin levels as much as 100 times above the recommended "safe" level for human exposure.

Environmental engineers for the EPA were scheduled to start taking soil samples today to determine whether the flooding had affected dioxin levels in the soil.

Young contended that the relocation proposal would be endorsed later this month in a report by the "hazard-mitigation team" set up by the Federal Emergency Management Administration.

That panel is made up of representatives of 10 federal agencies in addition to FEMA and the Corps of Engineers. Its task is to examine the problems caused by the flood and recommend ways to prevent them from recurring.

But Young's prediction was disputed by Patrick J. Breheny, FEMA's regional director and head of the team. Breheny said in an interview after Young's announcement that the relocation proposal would not even be included in the team's report.

"It's fine if the corps and the EPA want to work out something together, but it's not going into the report," Breheny said. "That's because it's not strictly dealing with flood-hazard mitigation."

Rick Alstadt of the Corps of Engineers called his agency's suggestion "a very preliminary discussion on whether or not this can happen." He said the proposal had been made to get the reaction of other federal agencies.

"By combining our authorities, the EPA and the Corps of Engineers could solve the problems of flood control along the Meramec and remove the dioxin," [illegible]

Alstadt is with the Missouri River division of the corps and is the agency's representative on the hazard-mitigation team.

William Hedeman, head of the EPA's Superfund program, said that the proposal to move the n had taken him "somewhat by surprise."

Hedeman said the idea was premature be se the EPA was "just beginning to determ if a contamination problem exists or if it is simp flood-debris problem." If the problems are only du to the flood, only FEMA, not EPA, would be concer ed, he said.

Congressional approval would be needed be re the corps recommendation could be carried out, h id.

FEMA's acting deputy director, Lee M. T mas, also noted that the hazard-mitigation tea was interested only in flooding problems, not dio . But Thomas added that recommendations for rel ating Times Beach residents had een included . the team's reports. Moving a small communit or a neighborhood in a flood plain can be t only permanant way to avoid flooding problems, he d.

Money for relocation might be availab rom federal agencies, including FEMA, Thomas sa That agency recently helped to pay for the relocat of a flood-prone neighborhood in Mobile, Ala., he s

But Times Beach probably would be unab get help with relocation from FEMA becau the community had rejected a federal flo lain ordinance limiting construction in such eas, Thomas said.

A spokesman for Young said the new Time ach would still be "close to the river" that residen love so much."

The $75 million in flood-control money Yo wills seek would include $20 million already aut zed. The money was allocated in 1981 in law deauthorizing the Meramec Dam proposal. ou g's legislative aide, Jim Webb, said Young was c nfident that he could persuade Congress to incre e that authorization by as much as $55 million.

Times Beach is believed to have been contaminated by dioxin in the early 1970s whe waste oil laced with dioxin was sprayed on the town's unpaved streets to control dust. The streets h ve been paved since then.

Dioxin is generated as an unwant the manufacture of certain products, cleansers, pesticides and wood preser highly toxic even in minute concentra hazard posed by the extremely low leve is unknown.

Dioxin can cause a severe skin di chloracne. Tests on animals show that cancer and birth defects and diseases ystem, liver, kidneys and bladder humans are suspected but not proven.

Mr. **Levitas**. Thank you, Mrs. Leistner, for that incredible statement. I think I have been reading and hearing as much about this problem as anybody else in Congress. I am sure there are others who have done as much. But I have never had it driven home to me as poignantly as listening to your presentation, which you described as the unexaggerated truth. I don't know how it could be exaggerated, quite frankly.

Mrs. **Leistner**. Mr. Chairman, I have only touched the tip of the iceberg.

Mr. **Levitas**. That is what I am afraid of. But I do want to thank you for that incredible statement. Your entire statement and attachments will be, without objection, made part of the record.

Before I proceed with a few questions that I have, and perhaps some other members of the committee, I note the presence of our colleague from Missouri, who has been very involved in expressing concern in this area. In fact, he and I had a conversation about this as recently as yesterday. Congressman Richard Gephardt.

I would like to invite Congressman Gephardt to, if he so desires, make a statement. If he also desires, he may join us on the panel here following that statement.

Mrs. **Leistner**. One more thing, Mr. Chairman. I would like at this time to leave with the committee a report prepared by the city of Times Beach that was presented to the President. It talks of dead birds and animals in the city. Also the interim report of the Missouri Dioxin Task Force, which was sent to me last week by the Governor's office.

Mr. **Levitas**. Are those documents already attached to your report?

Mrs. **Leistner**. No, sir, they are not.

Mr. **Levitas**. The subcommittee will, of course, be happy to receive those documents as part of your presentation, and they will obviously then be made part of the record for our use and examination.

Mrs. **Leistner**. Thank you.

[The information received follows:]

938-5332

CITY OF TIMES BEACH
235 Grove Road
TIMES BEACH, MISSOURI 63025

January 31, 1983

As the governing body of the City of Times Beach, St. Louis County, State of Missouri, we have prepared the following report. In the report we feel you will find what we deem as the most urgent needs of our people.

Our people are the blameless victims of a disaster that is too big for them to handle alone.

Our people have feelings of alienation, helplessness, and confusion which has created a great deal of stress among them.

We feel as good citizens, they deserve respect and prompt help from their government.

We feel, as you read the following report, you will find an earnest plea for help. We have joined together to put forth a united voice to inform our U. S. Senators, State Senators, and State Representatives of the unique and dangerous peril we are now facing.

Our people need and want an immedate buy-out of their property and, if possible, relocation of the City itself so they may be able to stay close by their friends, neighbors, and loved ones.

Our plea is one we hope will be heard and acted upon immediately for the lives of our people depend upon your answer.

May God be with you and lend you guidance in the days to come. Please give us your commitments and your stand on this matter in writing.

Respectfully Submitted

Board of Alderman
City of Times Beach

cc: Gov. Bond, Lt. Gov. K. Rothman, Att. Gen. John Ashcroft, EPA, CDC, FEMA, DNR, Pres. Reagan

TABLE I

A brief survey of clinical entities and symptom complexes seen be the Staff of Logan College of Chiropractic at the Times Beach Health Center.

Sinusitis, cephalgia, hypertension
Vesicular erruption on chin, Cephalyia
Sinusitis, fatigue, pharyngitis
Non-specific upper respiratory infection, sinusitis, pneumonitis
Cervical arthralgia, hyperextension
Sinusitis, Upper respiratory infection, pharyngitis
Cervical arthralgia
Lumbalgia
Myofascitis right and left shoulder, cervical artharalgia
Duodinal ulcer, hiatal hernia
Severe acne-two years duration, 20 yrs. resident, sinustis, upper respiratory infection, intestinal flu
Bronchitis with pneumonitis, probable hypothyroid
Tonsilitis, lethargy
Generalized muscle pain
Cephalygia, flu, nausea, vomiting, general body aches, laryngitis
Tetanus reaction, axillary carbuncle, vague neurologic symptoms (parasthesia - hands and feet)
Anorexia, upper respiratory infection, lethargy
Pharyngitis, upper respiratory infection, diarrhea, postural hypotension
Coccal arthralgia
Contact dermatitis (carpet)
Thoracic outlet syndrome
Myocitis, cervical and dorsal arthralgia, upper respiratory infection
Inflammation of auditory canal
Upper respiratory infection, pneumonitis, sinusitis
Pharyngitis, cephalgia, tonsilitis
Lumbalgia
Lumbalgia
Anorexia, cervical and thoracic arthralgia, probable generalized contact dermatitis, diarrhea, enlarged thyroid, migrane, numbness arms and hands with edema, insomnia
Hypertension, shoulder arthralgia, pneumonitis
Pneumonitis
Lethargy, chest pain
Upper respiratory infection, cephalgia, nausea
Diabetes, insomnia
Cephalgia, lumbalgia
Pain auricular cyst, pustular irruption over buttox and back of legs
Lumbalgia
Strep throat
Cephalgia, blurred vision, conjunctivitis
Apparent contact dermatitis, previous history of dermitis 8-9 yrs.
Pharyngitis, pneumonitis
Cephalgia, thoracic myofascitis
Cephalgia
Arthralgic left foot
Lumbalgia, cervical arthralgia
Pneumonitis
Lumbar myofascitis
Cephalgia, cervical arthralgia

Table I (con't).

Thoraco-lumbar arthralgia
Lumbalgia
Lumbalgia
Upper respiratory infection
Migrane
Laryngitis
Upper respiratory infection
Upper respiratory infection
Cephalagia, cervico-thoracic arthralgia
Cephalgia
Cephalgia
Lethargy, Cephalgia
Strep Throat
Cervical arthralgia
Severe non-defined neurologic disorder
Shock
Diabetes, varacosities
Cat Bite
Upper respiratory infection, cephalgia, rhinitis
Cervical arthralgia
Cephalgia, lethargy
Cephalgia, sprained right ankle
Cervical arthralgia
Cephalgia, Lethargy
Histoplasmosis, laryngitis
Otitis media
Otitis media, canker sores
Lumbalgia
Hyperactivity, dermitosis, asthma, failure to thrive, arthralgia
Open lesion right leg (11 month duration), two miscarriages
Flu symptoms
Cephalgia, cervical arthralgia
Cephalgia, lethargy
Siunitis, Upper respiratory infection
Cephalgia, arthralgia right knee, postural hypotension
External ear infection
Hypertension, dermatitis, basal cell carcinoma 2 years, cephalgia
Strep throat
Cephalgia, cervical arthralgia
Hepatomegalia, cephalgia
Thoracic arthralgia
Cervical and lumbar arthralgia
Cephalgia
General muscle pain
Ptosis left eye lid
Thoraco-lumbar arthralgia
Gastritis, cephalgia
Allergic dermatitis, rhinitis
Follicular tonsilitis
Pharyngitis, tonsilitis
Cervical arthralgia
Cephalgia
Vertigo
Lethargic, vague aches and pains

Table I (con't)

Hypertension, cerebral anorysm, epilepsy
Severe generalized acne-3 yrs. duration -resident 5 yrs, vertigo, cephalgia
vague neurologic symptoms
Hyperactive
Upper respiratory infection, pneumonitis
Hypertension, cephalgia, lumbalgia
Cephalgia
Hypertension, hemoptosis-6 months, Upper respiratory infection
Conjunctivitis, flu symptoms, cephalgia
Rhinitis
Lethargy
Digestive disorders since 1973 - oil dumped into his garden area
Cephalgia
Arthralgia right hip
Spastic diarrhea
Ear infection

- COMPILED BY BOARD OF ALDERMAN CITY OF TIMES BEACH

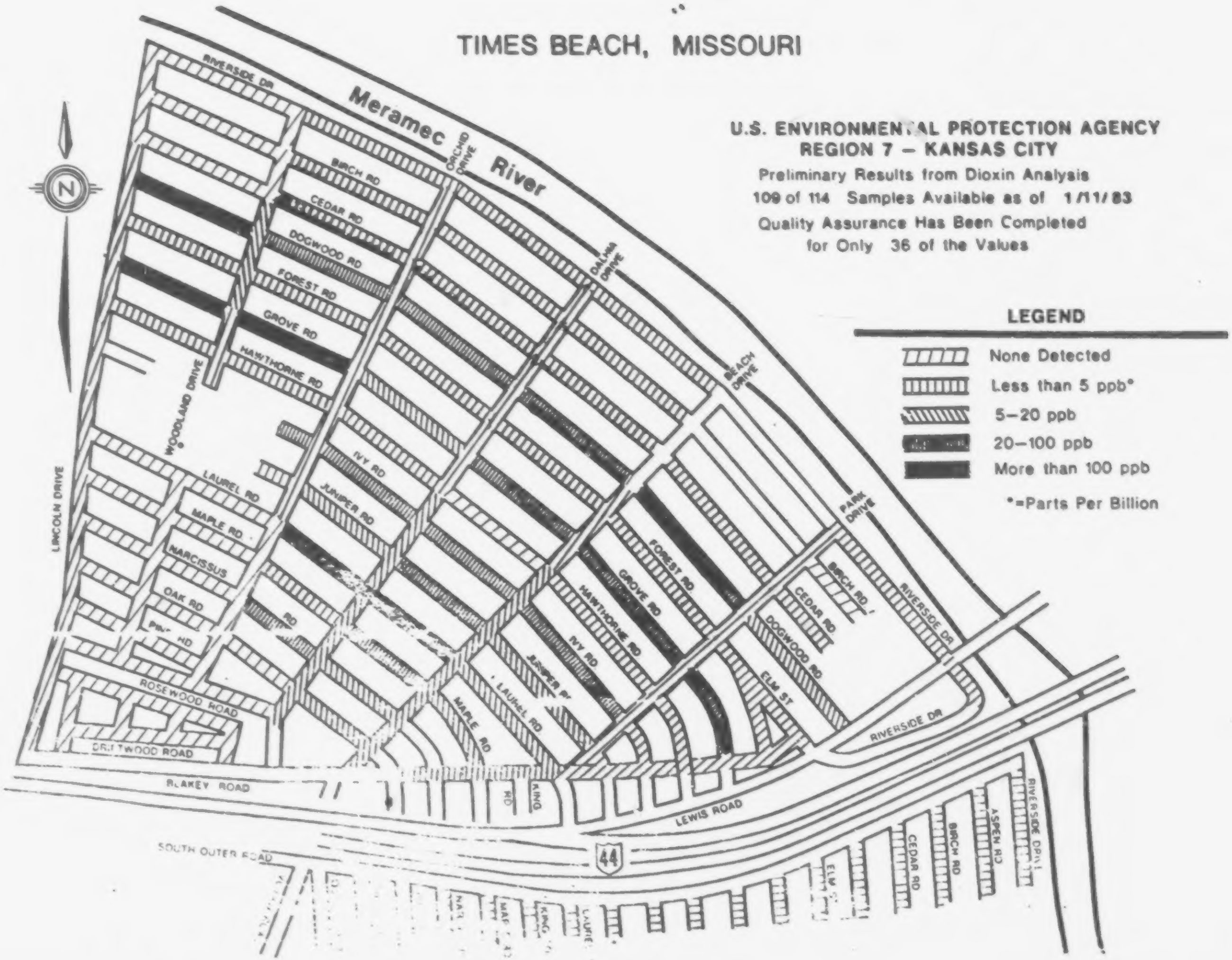
TIMES BEACH, MISSOURI
U.S. ENVIRONMENTAL PROTECTION AGENCY
REGION 7 – KANSAS CITY
Preliminary Results from Dioxin Analysis
109 of 114 Samples Available as of 1/11/83
Quality Assurance Has Been Completed
for Only 36 of the Values
LEGEND
None Detected
Less than 5 ppb*
5–20 ppb
20–100 ppb
More than 100 ppb
*=Parts Per Billion
N
Meramec River
RIVERSIDE DR
BIRCH RD
CEDAR RD
DOGWOOD RD
FOREST RD
GROVE RD
HAWTHORNE RD
ORCHID DRIVE
DALHIA DRIVE
BEACH DRIVE
PARK DRIVE
WOODLAND DRIVE
LINCOLN DRIVE
IVY RD
JUNIPER RD
LAUREL RD
MAPLE RD
NARCISSUS
OAK RD
ROSEWOOD ROAD
DRIFTWOOD ROAD
BLAKEY ROAD
SOUTH OUTER ROAD
KING RD
ELM ST
LEWIS ROAD
44
ASPEN RD

TABLE 2

ANALYTICAL RESULTS*
TIMES BEACH VOLATILES ANALYSIS

Parameter	Approximate Concentration (ppb in soil)
acetone	8[illegible],000
methyl ethyl ketone	4[illegible],000
methyl isobutyl ketone	36,000
mesityl oxide	9,000
toluene	120,000
3-hexene,2,5-diol	14,000
ethylbenzene	170,000
cumene	24,000
m-xylene	510,000
o,p-xylene	130,000

10 [illegible]
Chemicals [illegible] [illegible]
of [illegible]ate [illegible] property of
& parks. Times Beach [illegible]
[illegible] PCB's & [illegible]

---

* Sample #10B

BUSINESSES OF TIMES BEACH

BUSINESS NAME	TYPE OF	PROPERTY VALUE	INVENTORY	GROSS YRS [illegible]
Gary Waltman T. Beach, Mo.	Liquor Store Owner			$ 300,000
Charles Stone Easy Living 1327 Dahlia T. Beach, Mo. Tel. # 938-5065	Mobile Home Park & Laundramat	$3,500,000 park $ 200,000 laund.		$ 360,000
Bill Perschbacker 1327 Dahlia T. Beach, MO. Tel. # 809-775-1271	Mobile Home Sales Lot	$ 175,000 7-11 Store $ 325,000 Mobile Home Sales Lot		$ 18,000 7- 11 Store $ 15,000 Mobile Sales Lot
Jerry Hatcher P. O. Box 600 1305 Blakey T. Beach, Mo. Tel. # 938-5770 532-4122	Construction Equip.	$ 500,000	$ 300,000	$1,250,000 to $2,500,000
Sid Hammer 220 Birch T. Beach, Mo. Tel. # 938-5064	Plumbing Repair	$ 70,000		$ 65,000 to 70,000
James Curtis 510 Hawthorne T. Beach, Mo. Tel. # 938-4952	Excav. & Const.	$ 250,000 $ 200,000 Church		$ 8,000 $ 22,000 Rental $ 20,000
Ralph [illegible] T. Beach, Mo	CB Shop	$ 130,000		$ 60,000

BUSINESS NAME	TITLE OF BUSINESS	PROPERTY VALUE	INVENTORY	GROSS YRS. SALES
Ron Smith Frontier Lounge T. Beach, Mo.	Lounge Operator			$ 300,000
Bill Buchanan Frontier Lounge 701 N. Outer Rd. T. Beach, Mo. Tel. # 938-6627	Lounge Owner	$ 150,000		$ 7,800 Rental
Steve Hrin Southtown Auto T. Beach, Mo. Tel. # 938-5400 742-3416	Auto Body Shop	$ 170,000	$ 45,000	$220,000
Paul Benson Forest Rd. T. Beach, Mo. Tel. # 257-4329 257-6111	Auto Body Shop	$ 260,000	$ 75,000	
Ken Hutcheson Times Beach Bait & Tackle Eureka Heating & Cooling Forest Rd. Times Beach, Mo.	Bait Shop Heating Co. Out of business	$ 150,000	$ 18,000	
Joe Petro S. Outer Rd. T. Beach, Mo.	Commercial Building Owner	$ 175,000		
[illegible] Waltman S. Outer Rd. T. Beach, Mo.	Pallet Company			Partner of J. D. Lindly
Sal[illegible] [illegible]mer [illegible] Lounge [illegible] Mo.	Western Lounge	Information not Available		

BUSINESS NAME	TYPE OF BUSINESS	PROPERTY VALUE	INVENTORY	GROSS YRS. SALES
J. D. Lindly T. Beach. Mo.	Logger	$ 65,000	$ [illegible],000	$ 500,000
Ken Gr[illegible] Riverside Driv T. Beach, Mo. Tel. # 938-5503	Logger	Inf. not Available		
Don Johnson T. Beach, Mo. Tel. #	Logger	$ 165,000		$ 225,000
Grace Newson T. Beach, Mo. Tel. # 587-2500	Beauty Operator	Inf. not Available		
Southland Corp. Seven-11 Store T. Beach, Mo. Tel. # 427-5711	7-11 Store			
Gateway Products T. Beach, Mo. Tel. #		$ 75,000	$ 15,000	$ 850,000
Rowell Const. T. Beach, Mo. Tel. #	Const. Contractor	0		$ 200,000

BUSINESS NAME	TYPE OF BUSINESS	PROP	INVENTORY	GROSS YRS. SALES
J. S. [illegible]ers KOA Campground 1380 S Outer Rd T. Beach, Mo Tel. # [illegible]-544[illegible]	Campground	$ 800,000	Inc.	$ 230,0[illegible]
Laine Jumper 410 Dahlia T. Beach, Mo. Tel. 938-6077	Building Contractor	$ 189,300	51,000	$ [illegible]30,000
Vance Truitt 1402 Blakey Rd. T. Beach, Mo. Tel. # 938-5850	Oil Company	$ 325,000	Inc.	$2,500,000
Wade Dake Forest Dr. T. Beach, Mo. Tel. # 587-7242	Auto Repair	$ 150,000	$ 73,000	$ 70,000
Gary Overton 1000 Blakey T. Beach, Mo. Tel. # 938-5407	General Contractor	$ 200,000	$ 60,000	$ 75,000
Walter Adams 1004 Blakey T. Beach, Mo. Tel. #938-6373	Excavator	$ 150,000	Inc.	$ 35,000
Lloyd Wieda 223 Grove T. Beach, Mo. Tel. # 938-4916	Florist	$ 90,000	Inc.	$ 6,000

Times Beach Chronology: 2 Months Of News About A Troubled City

By Daniel O'Neill
Journal Staff Writer

It has been nearly two months since news first circulated that Times Beach was one of many sites in Missouri being investigated by the Environmental Protection Agency (EPA) for possible dioxin contamination.

In that time, the city has absorbed a cruel combination of natural and man-made disasters that threaten its very existence and have made it the source of local and national news almost daily.

The news has been sometimes confusing, sometimes repetitive and often conflicting. The following is a chonological look at the recent events that have affected Times Beach as they were reported in the local media.

WEDNESDAY, NOV. 10

The *Journal* reports that a list compiled by the EPA reveals Times Beach is among sites to be investigated for possible dioxin contamination. The list, which was not intended to be publicized, includes Times Beach among areas sprayed by an Ellisville waste oil dealer, Russell Bliss, who is known to have purchased waste-oil laced with dioxin.

When contacted by a Times Beach official, Bliss assures the city that no dioxin was in the oil he used to spray Times Beach streets.

WEDNESDAY NOV. 17

The *Journal* reports that an EPA spokesman has notified Times Beach officials that the town is high on the agency's priority list to be investigated for possible dioxin-contamination.

In making the announcement of the notification, Times Beach Mayor Joe Capstick says, "They don't know exactly who has it, but they have to assume that it's possible we have it."

WEDNESDAY, NOV. 24

Local daily newspapers report that federal health and environmental officials considered steps to seal off dioxin-contaminated areas in the state as early as last summer. Plans included evacuation of residents in areas of Jefferson County.

Dioxin was generated from a pharmaceutical plant in Verona, Mo. as an unwanted byproduct in the making of other chemical products such as disinfectants.

Dr. Renate Kimbrough, a spokesman for the U.S. Centers for Disease Control (CDC) in Atlanta, advises that concentrations of dioxin above 1 part per billion parts of soil are, in her opinion, cause for immediate action.

TUESDAY, NOV. 30

The EPA begins testing streets in Times Beach. It is also reported that more than 100,000 gallons of waste oil that may have been contaminated with dioxin were sprayed on most of the streets in Times Beach by Russell Bliss in 1972.

Some Beach residents remember the spraying and recall such things as streets turning purple, a funny smell like paint thinner and birds leaving the area.

WEDNESDAY, DEC. 1

Residents of Times Beach plan a fund-raising drive to hire Envirodyne Engineers Inc., a private firm, to test for dioxin.

Times Beach resident Laine Jumper said "Residents want to know right now whether the dioxin is here. We appreciate what the EPA is doing but they're not fast enough."

EPA officials say that if dioxin is found in the first tests, more tests will have to be taken.

Jeff County Journal 1/19/83

THURSDAY, DEC. 2

An expert on dioxin says residents of contaminated areas need not worry about health effects unless they develop chloracne, a disfiguring skin ailment. Robert A. Neal, president of the Chemical Industry Institute of Toxicology - an industry financed institute that studies toxicological concerns related to the manufacture, use and disposal of chemicals - tells Beach residents that there is "no evidence that dioxin caused long-term health problems."

"If there is no chloracne," Neal said, "one can be reasonably assured that serious illness from exposure is not resulting."

The same day, Russell Bliss says that half the dioxin-contaminated oil he possessed was sprayed on his farm in Rosa[illegible] Mo. and at his three horse arenas in eastern Missouri. Bliss says the remainder of the oil was burned as heating oil. "I hope my [illegible] rots in hell if I'm lying," Bliss adds. "It looked like good oil to me."

Health officials describe the form of dioxin produced by the company in Verona - Northeastern Pharmaceutical Co. - as the most poisonous of the 75 varieties of dioxin. Officials call it 150,0[illegible] times more toxic than cyanide.

SATURDAY, DEC. 4

Week-long rains continue and officials begin warning of [illegible] high flood waters. Civil [illegible] news advises residents [illegible] Beach to gather their [illegible] and evacuate the town [illegible] flood waters hit. Most [illegible] don't believe the Meramec will reach the predicted [illegible] and few pay any attention to [illegible] warnings.

SUNDAY, DEC. 5

The Meramec River overflows its banks and rages through the area, completely inundating Times Beach. Waters rise up to four feet in one hour and reach record levels. Most people are caught by surprise in Times Beach and virtually every home is damaged or destroyed

WEDNESDAY, DEC. 15

Preliminary tests done by Envirodyne Inc. show that a section of Times Beach City Park contains at least 10 different toxic-waste chemicals. The test was done after Envirodyne was hired by a citizens' group to test other soil samples in the city. Representatives of the company decided to test the park soil after smelling a curious odor in the area.

FRIDAY, DEC. 17

EPA officials tell residents not to jump to conclusions about how much of the town is contaminated with dioxin. However, Art Spratlin, deputy director of air and hazardous material programs at the EPA regional office in Kansas City, expresses fears that the EPA and the city may face an unprecedented cleanup. Spratlin says the "potential is mind-boggling."

Test results by Envirodyne show levels of dioxin up to 16 parts per billion in Times Beach, exceeding the danger level of one part per billion.

TUESDAY, DEC. 21

The EPA announces that three toxic waste sites in Missouri are on its priority list of sites nationwide. The areas are the Shenandoah Stables at Moscow Mills, the Syntex facility in Verona and the Stout and Minker residential areas near Meramec Heights in Jefferson County.

SUNDAY, DEC. 26

The EPA announces that it plans to begin tests in Times Beach to determine how the recent flooding affected contamination in areas of the community. The agency also recommends that residents in Times Beach, driven from their homes by the flood, stay away and that those who have returned to their homes, leave. The recommendation comes after results of preliminary tests by the agency reveal levels of contamination in excess of 100 parts per billion.

Meanwhile, Mayor Joseph Capstick resigns and Alderman Sidney Hammer is named acting mayor.

TUESDAY, DEC. 28

Former mayor Capstick says he believes the EPA and health officials are overreacting to the problem. Many residents voice similar sentiments as they return to their damaged homes. Some flood victims in the area begin receiving disaster relief checks from the government.

Acting mayor Hammer accuses federal authorities of acting too slowly on the dioxin question. Hammer says, "The EPA must think we're a bunch of jerks and country bumpkins." A spokesman for the EPA indicates his office is working as quickly as possible and promises to "pull out all the stops" to solve the problem.

WEDNESDAY, DEC. 29

Nearly 1,000 people turn out at Eureka Junior High School for a hearing held to discuss health problems. They voice anger and criticism at the way state and federal authorities have handled the situation.

State Rep. Bob Feigenbaum tells the crowd he will introduce a bill that will create a state "Superfund" to help clean up the waste.

The Federal Emergency Management Agency (FEMA) announces it will bring a number of mobile homes to the area for those driven from their homes.

THURSDAY, DEC. 30

Despite the possibility of dioxin contamination, Pat Breheny, regional director of FEMA, says officials have decided to haul flood debris from Times Beach because leaving it strewn about the city poses an immediate hazard.

President Reagan orders federal agencies - EPA and FEMA - to give him a report on the Times Beach situation.

FRIDAY, DEC. 31

Results of additional soil tests, taken before the flooding, show more contamination. The EPA releases results indicating dioxin levels in several streets and they announce plans to do more testing. Officials also plan to examine residents for correlations between health problems and dioxin contamination.

Meanwhile, plans to remove flood debris are halted until new tests show whether the debris is contaminated.

A St. Louis paper reports that the county police refuse to patrol Times Beach because of the dioxin. City officials had asked the county for police assistance after having to release four city policemen because there was no insurance to cover them.

SUNDAY, JAN. 2

A spokesman for the White House says the president has been briefed on Times Beach and is "continuing to look at the situation."

The state decides to send mental health workers to the city to help counsel residents and help them cope with the strain of the situaton.

WEDNESDAY, JAN. 5

The U.S. Army Corps of Engineers proposes to move the entire town of Times Beach and build a new town on higher ground. U.S. Rep. Robert Young, whose district includes Times Beach, supports the proposal enthusiastically.

Hammer says, "I think it stinks. It's one of the dumbest ideas and most ridiculous ideas I've ever heard. I'm prepared to fight it every inch of the way."

State natural resources officials suggest that the flood debris be moved to a sanitary landfill and local authorities meet to decide where to take the waste.

THURSDAY, JAN. 6

Hammer announces that he could support a proposal by the Corps and Young to build a new town. "Ninety-nine percent of the people would support it if they give us something comparable to what we had," says Hammer. The acting mayor also indicates that the removal of flood debris will begin the next day. The trash is to be hauled to Bob's Home Service, a toxic-waste landfill in Warren County.

Meanwhile, tests show that the highest concentration of dioxin is in a residential area just west and north of the center of town. Test samples that had shown levels above 100 parts per billion were taken from the area on the northeast corner of the city park.

U.S. Sens. Thomas Eagleton and John Danforth call for federal hearings on the dioxin problem and say the "EPA is still dragging its feet in getting the testing teams assembled and on the job."

The previous night, nearly 500 residents met at Eureka Junior High School and vented their anger at delays in the cleanup and the uncertainty surrounding the dioxin. At the meeting Hammer displayed a large map from the EPA depicting the wide extent of the dioxin contamination. The map, derived from tests taken before the flood, shows that most of the city's road beds are dioxin-contaminated with levels ranging from below five parts per billion to more than 100 parts.

Residents of Wright City, where Bob's Home Service is located, express uneasiness about accepting debris from Times Beach at the dump site.

FRIDAY, JAN. 7

President Ronald Reagan orders the formation of a special task force to coordinate flood and dioxin cleanup in Times Beach. Missouri Gov. Christopher "Kit" Bond sent a letter to Reagan stating "I know of no other community that has experienced this magnitude of simultaneous trauma."

The EPA raises questions about toxic levels in flood debris, forcing the state to delay plans to move the debris to a landfill.

Meanwhile, officials in Warren County threaten to go to court to block dumping of the waste in Wright City.

SUNDAY, JAN. 9

Members of Reagan's task force arrive in Times Beach, along with Bond, spurring the start of the long-awaited debris clean up.

Officials order that the debris be loaded into trucks and transported to the hazardous waste landfill in Wright City. Hammer says, "Finally, something is starting to happen" while cautioning that citizens will "take matters into their own hands if the cleanup doesn't continue."

Hammer also makes a plea for the president to come to Times Beach to view the situation first-hand, adding: "If we can't get help from this country, we'll go to another nation for help."

Officials of Warren County obtain a court order from Circuit Court Judge Edward B. Hodge of Warrenton blocking the transfer of the debris to Wright City.

MONDAY, JAN. 10

Tractor-trailer trucks are loaded with flood debris and await the go-ahead to begin transporting the possibly dioxin-contaminated cargo out of Times Beach. The transfer has been temporarily stopped by Judge Hodge's restraining order. State officials say they will try to dissolve the order.

TUESDAY, JAN. 11

Warren County officials are told to post $75,000 bond by 9 p.m. if they wants to maintain the temporary court order blocking the transfer of debris. The restraining order will expire if the bond is not posted. A dozen trucks, piled with debris, wait in Times Beach.

Residents of Times Beach collect more than 500 signatures on a petition asking the president to affect a federal buy out of their flood-ravaged, dioxin-contaminated town.

WEDNESDAY, JAN. 12

The restraining order blocking the transfer of flood debris from Times Beach to Bob's Home Service in Wright City is lifted after Warren County residents fail to post the necessary bond to maintain the court order. Trucks loaded with trash begin rolling out of Times Beach by [illegible]. Tests results show no harmful levels of dioxin in the flood debris.

THURSDAY, JAN. 13

More than 100 people sign a letter asking foreign countries for help in the crisis. Times Beach alderman Harold Goodman says: "If it will help, the people will sign anything. We're that desperate. We'll declare ourselves a foreign country." Letter organizers say that copies of the letter will be sent to the governments of Sweden, Great Britain, Japan, Australia, Belgium, Canada, Norway, France and the Netherlands.

SATURDAY, JAN. 15

Residents of Times Beach and Warren and Jefferson counties join forces to protest and draw attention to the dioxin problem with a demonstration on I-44. Organizers expect protestors to number more than 1,000.

"REPORT ON THE CITY OF TIMES BEACH"

1. WELFARE OF THE PEOPLE:

a. Stress has caused several of our citizens to have nervous breakdowns and heartattacks. There are cases of child abuse, wife abuse, our people are always on edge; in short, our people are leading a non-existing life-style.

b. Symptoms of being exposed to Dioxin (list enclosed).

c. Our children are being harassed, we have numerous cases where our children come home from school and go directly to bed and stay there all day and all night. The laughter is gone from our children; they have no permanent home to go to, they worry about having to move away from friends and school. There is a growing concern in our children about how the Dioxin has affected them--will they be living ten, twenty, or more years from now.

d. Our people are still living in motels, camps, with relatives, and we have some living five or more in one-bedroom houses.

e. A few of our people are still living in Times Beach because they have no place to go; with the fear of what the Dioxin will do to them, they don't allow their children to go outside because of this fear.

f. The Missouri Housing Development Commission and the Federal Emergency Management Agency's proposed housing for our people is disastrous. In order to apply for this housing our people cannot make over $23,000.00 a year or their percentage rate rate will be 12%; on a $42,000.00 home that would mean payments of $400.00 to $500.00 or more, with a large down payment. Also, the lots being offered are outside St. Louis County and out of our school district. There is no way our Senior Citizens can afford even the lowest percentage rate of 4% much less, being eligible for a loan.

g. Any human being needs stability in their lives; our people do not have any--we are in a temporary state of existance with no future to look forward to.

h. Our people are faced with delinquent mortgages and loans they cannot pay. Harrassment from these loan companies and banks because they are unable to make payment. Our property values are -0- because of the Dioxin.

i. The insurance coverage on the people's houses is being cut-off. The people took the advice of CDC and left their homes--now their insurance on these homes is being cancelled.

j. Our businesses lay in ruins; because of the Dioxin, we have over thirty businesses in Times Beach being forced to close their doors.

k. In regards to the Superfund moneys: Our people are being made to feel in order to use the Superfund money to help us, it would be charity and would be a waste on the part of the EPA to use the Fund in this way. As United States citizens and tax-payers we feel that we have the right to expect all the help available from any agency in our Government.

"The American Dream" has been destroyed; our people are beyond the breaking point; we need immediate answers and committments now--we will not last much longer--our plea is for our people and our children. Please bring back the smiles and the laughter of our children; now there are only tears of sorrow. Our children's lives and happiness are important to us.

2. THE CITY OF TIMES BEACH (GOVERNMENT):

   a. Financially our City is broke we cannot meet our debts, fix our road, water systems, or repair our city buildings.

   b. There is no financial aid for our city because of the Dioxin and being in a Flood Plain.

   c. Lay-offs of our City personell has begun; as well as our Police officers, because funds are not available to pay them.

   d. Our City will not be able to hold on much longer, and we need answers and committments now.

3. IN THE YEARS TO COME (FUTURE PROBLEMS):

   a. Will our people because of the Dioxin be able to get any type of Insurance Coverage?

   b. Will our people have the Medical Assistance they need in the future?

CITY OF TIMES BEACH
235 Grove Road
TIMES BEACH, MISSOURI 63025

[illegible] January 31, 1983

The following accounts can be verified by neighbors, relatives, and medical records:

In one block of Grove Street live only two families. This particular block was found to have well in excess of 100 ppb's of dioxin.

In the first family these problems are noted: The husband had complicated illnesses which included 70% loss of hearing, lapse of memory, blood disease, and severe hardening of the arteries which brought on a fatal stroke. He died 2½ years ago. It is also noted that he had large clusters of blackheads around his eyes, ears, and mouth that when agitated became extremely sore and bloodshot. The wife presently suffers from a severe skin disorder in the palms of her hands which leaves them very dry, cracked, and often bleeding.

Of the second family: The wife suffers from a similar skin condition. She also has 80% loss of hearing and a lapse of memory. She has kidney problems, and an uncharacteristic feebleness that she has required hospitalization at least six times in the past two years. The husband has also been hospitalized several times in the past two years and presently suffers from leg problems and a stomach disorder.

It is also known of several deaths of small animals in a one block area. These include at least nine dogs which died in very similar ways. These dogs, normal and healthy, grew to be several weeks old: all were active and ahd good appetites. Suddenly, each in isolated cases lost all appetites, would not move unless helped, would bleed at the rectum, and finally died. These pups; four out of five of one litter, four dogs of a neighbor, and one dog of another neighbor were born at different times yet showed common problems.

In the same area, many dead birds were found. Many of the squirells were found dead, and those as well as some which were seen about ready to

> die, would have no hair on their tail or body.
>
> One man who lives on another street found to contain over 100 ppb dioxin, has also had major medical problems requiring hopitalization and surgery. He also reported finding a great many birds in his yard dead or dying. For a while he would gather them up and burn them with his trash. One day he called the Humane Society about the problem and they requested he catch some of the live birds and they would take them for testing. He caught three and called the Society back, but they never came for the birds; so they died and he disposed of them as well.
>
> Finally, the account of a young family living on another "hot" street and often walked to church; which is on another "hot street". The husband, 29, has been told by his doctor that his kidneys and liver are in the condition of what he would expect to find in a much older man who had smoked and drank for a long time. Having been quite active in a church which teaches against both, he has neither smoked or drank. The wife, 25, has in the past two weeks undergone a radical mastectomy which doctors said is extremely rare for a young lady of her age and coming from a family with no history of breast cancer. The daughter, 4, has a bladder condition as well as a skin problem.

As stated at the outset, all of this can be verified by neighbors, relatives, and medical repor .

ILL MO bobcat P.O. BOX 600 • EUREKA, MISSOURI 63025 • (314) 938-5770

December 28, 1982

TO: Officials of the United States Government

From: Gerald J. (Jerry) Hatcher

Horace Greeley said, "The way to do things is to begin". I am beginning!!

I am a United States citizen, Missourian, Businessman of Times Beach for 12 years and a voting taxpayer. I have located a business in Times Beach by choice, and have lived outside Times Beach by choice. I have had no vote in what the town wanted to do about business license fees, road repairs, utility taxes or flood insurance. I have complied with the wishes of the town, because of their vote. I feel that I have been delt with fairly by the town authorities.

Now that dioxin has been discovered, I find the government says not to help anyone who didn't vote for our programs! Kick them, keep them down and let our United States citizen, Missourian, Businessman and voting taxpayer save himself. Don't help, unless he wants to relocate. He didn't like our programs, so let him sink. I will not sink!! But, I will remember!

If I lived on the coast of the U.S. of A., and had to face all of the disasters they face there, would I be asked to relocate or be denied a loan? There seems to still be people there! I do not feel that the government is looking out for the good of its citizens when they will not give a loan to a citizen to rebuild their homes or businesses, but will give to foreign governments that repay poorly or never! THAT money comes from the United States citizen, Missourian, Businessman and voting taxpayer.

Whether the government realizes it or not, the people in Times Beach lived here for reasons not much different than people living in New York, Miami or St. Louis. This is where they want to live or do business! What we need is not how to tear down the lives of the people in Times Beach, but how to build them up. LOANING not giving is a self help program.

A large automobile manufacturer was loaned USA dollars that they paid back. That company is a group of people! I would not be too surprised if the people at that company voted against some government programs that they didn't like, but they got a loan!

Times Beach is a town that needs USA dollars. Why don't they get a loan? No loans for people voting against a government program! They are a small group of USA people. Is that why, small? I have read where Times Beach is a mistake. Times Beach is not a mistake! It is a group of people that will survive! They will vote, work, pay taxes and continue to know what caring is all about! I am not sure about the U. S. Government and the elected officials or some of the other citizens that live in their ivory towers!

Gerald J Hatcher

Gerald J. Hatcher
U.S.A. Citizen
Missourian
Businessman
Voting Taxpayer

South Town Auto Body West, Inc.
905 BLAKEY
TIMES BEACH, MO. 63025
938-5400

Jan. 18, 1983

Dear Mr. President:

Please give this letter your immediate attention! We are in a crisis situation and cannot wait any longer for a solution.

I am writing this letter to charge the Environmental Protection Agency as being directly responsible for destroying business in Times Beach, Mo.

On December 5, the horrible chain of events began when our building was filled with 8 feet 4 inches of flood water. We stood on the highway (I-44), taking pictures, with tears streaming down our faces - 23 years in business, a lifetime of hard work, destroyed by the flood!

On December 7, when the flood waters had receded, my husband and our two sons went to see the damage that had been wrought by the flood. The sight that met their eyes was far worse than their greatest expectations. Our beautiful office and reception area was totally destroyed. The suspended ceiling was ripped down, cabinets and their contents were torn off the walls, the paneling was bulged out from the weight of the saturated insulation, all the equipment (tire changers, wheel balancer, paint mixing equipment, grinder, buffers, adding machines) were covered with mud. About four inches of mud and slime covered the floors, the 8 ft. cyclone fence and gate which enclosed the entire parking area was forced to the ground by the force of the water. The possibility of restoring our business to a operable state seemed like too monumental a task to even attempt. And yet, what was the alternative? We had too many years of hard work and money invested in this business. We still owe $75,000.00 on the loan for the building and $6,000.00 on the frame rack. We've put over $40,000.00 of our life's savings into this business. My husband and our two sons derive their income from this business. We have house payments and utility bills to pay every month.

So, with the help of numerous friends and members of the family, the monumental task began. They worked 10 hours a day, seven days a week for three weeks - cleaning out the mud, hosing off the equipment and drying it off, getting as much of the equipment as possible back in operation. Finally, on December 27th we hung out our sign to announce, "YES - WE ARE OPEN!" Our spirits were high and we felt that we had come through this terrible disaster in pretty good fashion. We called in jobs that had been scheduled for the week the flood hit. Our customers told us that they would wait until we re-opened, rather than take their cars somewhere else to be repaired. People were coming in for estimates, ordering tires, and things were looking pretty optimistic! And we did it all ourselves! During the flood disaster, much assistance was given to the residents of Times Beach from federal agencies, state agencies, the Red Cross, the Selvation ARmy and numerous charitable and community organization. Nothing was offered to the businesses that were flood victims! Nothing but Small Business Loans at 8% interest. We figured we didn't need any more loans, we already owed enough money. Business has been totally ignored during this whole crisis!

On December 30, the final blow came! The city put up road blocks at all entrances to the city, at the suggestion of the E.P.A. The E.P.A. had ordered all residents to leave Times Beach and ordered the general public to stay out of Times Beach because of possible toxic contamination. The next few days were like a nightmare! Every local newspaper had front page pictures of the ominous signs, showing a skull and crossbones and large block letters stating: "DANGER - DO NOT ENTER - POSSIBLE TOXIC CONTAMINATION". Every television station headlined their newsreports with the signs and the story. As the days went by, the news of dioxin contamination in Times Beach was getting national coverage.

THE DAY THAT THE SIGNS WENT UP, OUR BUSINESS CAME TO AN ABRUPT HALT!!!

Insurance agents, who regularly recommend us to their customers, told us that they had sent at least ten (10) people to us for estimates! But they came back with estimates from other shops, expressing a fear that they might jeopardize their health by coming into Times Beach! And really, who could blame them?

But just how dangerous is the dioxin contamination? The tainted oil was sprayed on the roads in 1971, and, as far as I know, their is no documented evidence that anyone here has suffered from exposure to this supposed contamination. In fact, no evidence has been given that anyone, anywhere, has suffered from exposure to dioxin contamination. EPA has no answers for us! Anytime they are present at a meeting or interviewed by the media, all they have said is, "We really don't know what the effects of exposure to dioxin have on human life." So, why the terrorist tactics? Why the isolation of an entire city? Why the signs with skull & crossbones? Why the men dressed in white coveralls and respirators? Believe me, the psychological damage done to the people of Times Beach by the EPA is far worse than the exposure to dioxin has been. The psychologic damage is evident on the faces and in the voices of everyone here. We have no direction. We have no defense against this disaster. We are confused. We are prone to burst into tears, for no specific reason. The frustration is unbearable.

But the damage is done and irreversible. The general public has been terrorized and Times Beach has been stigmatized. No matter what the EPA test results show, our business in Times Beach is finished.

The EPA is directly responsible for the loss of our business and must be held responsible for restitution for that loss ! BUY OUT is the only answer and we can wait no longer. If no money is forthcoming by FEBRUARY 1st, we will be forced to close our doors and file for bankruptcy. Five more people will join the ranks of the unemployed. Time is of the essence! We must have ACTION NOW!

Please respond to this letter at once.

Sincerely,

Marianne Hrin, Secretary-Treasurer

P.S. I am enclosing a letter, giving a short history of our business and pictures of the flood and the EPA damage.

ILL MO bobcat P.O. BOX 600 • EUREKA, MISSOURI 63025 • (314) 938-5770

For 11 years, I have owned an equipment dealership in the City of Times Beach. I would like to help you understand the problems incurred by someone involved with a City that has been flooded and contaminated by dioxin. The City of Times Beach voted against Federal Flood Insurance and must pay the consequences of the decision. I did not have a voice in the voting, because I chose to live elsewhere. I lost economically, the same as they. I am not concerned, nor do I ask for consideration from anyone, as a result of the flood.

I need help with the dioxin contamination problem. This is a man-made problem that I cannot control! I have been forced out of my building and off land where I normally do business. Because of the C.D.C. statement on December 24, 1982, no one will come into my place of business. S.B.A. informs me that they will loan money, but the land in Times Beach must be sold. If I can't do business there, how can anyone else? I have to pay rent for our alternate location, the loan payment on the original building and a new loan means more payments, combined with the extra expense of a forced move! The residents of Times Beach have been helped to relocate. The Government will give business establishments no such help! However, I could lose it all because I don't have a viable business, due to the dioxin statement by C.D.C.

I was refused a hand shake by a customer, because he felt I'd contaminate him! This may not seem to cause an economic impact, but if one thinks about not shaking the hand, he may not want to buy the equipment! Another customer wanted to make sure I had no dirt on a machine or truck when it was delivered!

I certainly do not know the answers, but know we must have one!

Sincerely,

ILL-MO BOBCAT, INC.

Gerald J. Hatcher
President

GJH/jd

ATTENTION HOME OWNERS OF TIMES BEACH:

If you the people and Homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so, please sign this petition, Thank You.

Mr. & Mrs. Richard [illegible]

Mr. & Mrs. Robert Loving

Robert Loving

J. Downing

Larry W. Taylor

Mr & Mrs Mark R Coffield 323 Dogwood

Doris Rodgers 308 Narcissus

[illegible] Rodgers " 539 Dogwood

Tom Manion ~~[illegible]~~ Birch 233 Riverside

Charles E Miles 319 Cedar Rd

[illegible] A Seitz 325 DOGWOOD RD

Dawn M. Seitz 325 DOGWOOD RD.

Lawrence C [illegible]

Douglas S King 601 Hawthorn 63025

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.
(PRE-FLOOD VALUE)

NAME	ADDRESS	PHONE NUMBER
Gary D Waltman	539 Dogwood	938 6105
Eula Waltman	539 Dogwood	938 61[illegible]
Eldon Faber	403 Forst	None
Freda Faber	403 Forst	None
J D Lindley	521 Cedar	58[illegible]-3228
[illegible] WALTMAN	540 Dogwood	587-2647
Ronald Waltman	[illegible]land	587-2[illegible]
[illegible] Lindley	521 Cedar	587-3228
Betty J Lindley	526 Dogwood	938-587[illegible]
Milton D. Lindley	526 Dogwood	938-5876
DeWayne Lindley	520 Dogwood	None
Charles Wells & Vivian	408 Grove	25[illegible]
Arthur & Mary L Richard	611 Riverside Dr	Times Beach
Donald & Susan Johnson	501 Birch	9385[illegible]

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

(PRE FLOOD)

NAME	ADDRESS	PHONE NUMBER
[illegible]	[illegible]	938-6033
Buddy Frey	500 MAPLE	225-4767
C. F. Stone	1309 Dahlia	938 5065
B.V. O. Poertner	4[illegible] NARCISSUS	257-6692
[illegible] Hendrickson	412 Maple Rd	938 6709
Evelyn Zufall	320 Hawthorne	938-5487
Lowell Zufall	"	"
Carolyn O'Leary	319 Hawthorne	587-7980 938 5270
1 R.A. Villing	325 Grove	587-7932
Patsy Crane	304 Grove	587-2610
Rose Elliott	321 Hawthorn	587-7000
2 Shirley Falls	501 Blakey	587-2286
3 Fay Burton	309 Hawthorn	587-2357 938-6923
1 Steve Berner Debbie Berner	200 Juniper	587-7356

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.
(PRE-FLOOD)

NAME	ADDRESS	PHONE NUMBER
James F. Curtis (Rev.)	510 Hawthorn	938 4952
Helen Curtis	501 Dogwood	
L. M. Curtis	524 Grove	
KEN GRUBBS	509 RIVERSIDE	938-5503
HERB BRADSHAW	507 RIVERSIDE	938-4993
[illegible]	337 Riverside	938-5670
Seymour Fourks	321 Riverside	938-6082
Victor Williams	504 [illegible] Eureka	
Mel Mooreland	436 Birch	587-3887
NAME	ADDRESS	
Nathan LaMar	1415 Blakely Rd	587-7395
DOYLE SHAW	423 CEDAR	587-2054
Bernard Essen	220 Forest	938-6694

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
Harlan W Bucker	617 Riverside Dr Times Beach	587-2393
Bobbie Bucker	617 Riverside Dr Times Beach	587-2393
Debbie Mason	510 Dahlia Times Beach	
Michael Mason	510 Dahlia Times Beach	
Nancy Miller	310 Grove Times Beach	587-79[illegible]
Elizabeth Cantrell	537 Lawn Times Beach	
Lucille Lampen	305 Riverside Times Beach	
Jim Piercy	207 Dogwood Times Beach	
Jean Piercy	207 Dogwood Times Beach	
Gary Overton	111 Maple Times Beach	
Catherine J. Overton	111 Maple Times Beach	
Robert [illegible]	68 woodland Times Beach	
Lloyd F. [illegible]	223 Grove Road T.B.	
Dorothy C. Wells	316 Forest Times Beach	

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and home owners of Times Beabh, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.
([illegible])

Sherry Mims 510 Narcissus Times Beach
E. Victor Williams 504 Narcissus Times Beach
[illegible] 327 [illegible] Times Beach
Dwight H Creech 89 W Pine TIMES BEACH
Dave Buchanan 525 BIRCH Times Beach
Charlie Buchanan 416 FOREST Times BEACH
Bill Buchanan 418 FOREST Times BeaCh
Bill Buchanan 215 DOGWOOD Times Beach
Ronald L Meyer 219 RIVERSIDE TIMES BEACH
Peggy L Meyer 236 Cedar TIMES BEACH

20. Kathy Jumper 410 Dahlia Times Beach
214 Cedar
512 Ivy

21. LaVerne Baker 629 Riverside Rd. Times Beach

22. Dave Buchanan 525 BIRCH RD Times Beach

23. Pearl James [illegible] [illegible] Times Beach

24 Mr & Mrs Dan Johnson 329 Riverside Dr Times Beach.

25 Charles & Maxine McDaniel 239 Cedar Times Beach

Albert J. Cantrell 537 Laurel Times Beach

Donna D Wood 4[illegible] OAK - TIMES

Dennis Kaden 119 W Pine
Lynn Hill 516 MAPLE
Christina Kaden 119 W Pine
James Jiny 207 Dogwood
Leroy & Florence Goettle 405 Park
Lloyd & Violet Lake
Emmett Womack, Betty
Mr. & Mrs. Alvin Mullen 333 Riverside
Mr. & Mrs Willard Kalgren 615 Riverside
Mr & Mrs Leo Fritz 610 Grove
425 Birch
Linda Windle 616 Cedar
Dorothy Duffer 339 Dogwood
Ralph Penn 601 Grove
Robert Stuart 413 Cedar
Mr. & Mrs. Ralph Jackson 603 Forest
F.C. Frank 136 Birch Rd.
Vernon Shipley 609 Cedar
Paul Aulds 347 Narcissus
Eula Waltman 539 Dogwood
Roy Hamil 324 Birch
Walter Lawer 1415 - Bleakly road
Joe LINDLEY 521 - CEDER
Roger Phillips 217 Birch
John Sowards 215, 212, 210 Birch
Dave Burton 525 BIRCH
Mr & Mrs Darrel Martin 52 Smokeland
Ray Wehner 15 Aspen Dr. " 14
Mr " & 521 Dogwood Time Beach
James Tozlner 311 Forest
Mary Miller 551 Maple

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You. (Pre Flood Value)

NAME	ADDRESS	PHONE NUMBER
[illegible] E Maxwell	212, Blumke Rd	933-5557
[illegible] Maxwell	521 Dogwood	257-6693
Wayne Wilson	538 Maple St	587-2611
[illegible]	[illegible] Dogwood	
[illegible] Wittman	[illegible]	
[illegible]	213 Dogwood	
[illegible]	213 Dogwood	
[illegible]	[illegible]	
[illegible]	202 [illegible]	
[illegible] Naughton	421 Oak	87-2785
[illegible] Naughton	421 Oak	" "
[illegible]	201 [illegible]	
[illegible]	[illegible]	935-[illegible]
[illegible]	[illegible]	" "

ATTENTION HOME OWNERS OF TIMES BEACH:

If you the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so, please sign this petition, Thank You.

Errol Cates 405 Grove Rd. ~~[illegible]~~ Larry Baum
Rudolph Cox 519 Oak Rd.
Virginia Cox 519 Oak Rd.
Henry Hinton 224 Juniper
Kate Hinton 224 Juniper.
Elvi Jordan 424 Beach Dr.
Mr. and Mrs. Charles Martin
Virginia Morris
Margaret Eberhardt
Pat Crawford 510 Ivy Rd
Linda Crawford 510 Ivy Rd
[illegible] Chronister 470 Maple
[illegible] Chronister 420 Maple
[illegible] 401 Hawthorne
[illegible] 401 Hawthorne
[illegible] Baum 321 Ivy
Lois Baum 330 Ivy
Alice Short 403 Hawthorn
Mike Short 403 Hawthorn
[illegible] Pifer 310 Grove
[illegible] Fisher 620 Cedar
[illegible] Murphy 319 GROVE
[illegible] Stone 1327 Dublin
Frank Hanner 437 Riverside
Madeline Hanner 437 Riverside Dr

[illegible]ION HOME OWNERS OF TIMES BEACH:

If you the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so, please sign this petition. Thank You.

1) William E Funck Jr 520 Birch Times Bch
2 Kenneth L. Kropper 533 Birch Times Beach
3 Walter Adams 207 Laurel 928-6373
4 Lois Jumper 410 DAHLIA [illegible] 310 SUNSHINE [illegible]
5 Joan Caito 326 Birch Rd
6. Tom Phillips 241 BEACH RD TIMES BEACH
7. Stacy Berner 200 Juniper Times Beach
8 Lee & [illegible] Coffman Times Beach
9. Carolyn O'Leary 319 Hawthorn
10 Mary Smith 405 Laurel
11.) Kevin Young 605 Juniper
12 Les Berner 314 Hathorne Time Beach 30 yrs
13) Wallace & Pat Maxwell - 112 Rosewood Rd.
14) Rose [illegible] 528 Oak Rd.
15) Joe Ann Portwood 101 Driftwood Rd. Times Beach Mo.
16) Chester Jeremane Pollard 217 Hawthorne
17) Roma Dipaoli 1409 Oakley St.
18) Milton & Betty Lindley 526 Dogwood [illegible]

ATTENTION: HOME OWNERS OF TIMES BEACH, MISSOURI,

If you the people and home owners of Times Beach had an opportunity to sell your house now at market value, WOULD YOU? If so please sign this petition. Thank you.

1. Kelvin J. Harvey 329 Birch Times Beach, Mo. 63025
2. Otto Jumper 316 Cedar
3. Robert E Harvey #2 Elm Time Beach, Mo 938-5[illegible]
4. Harold L Laycock [redacted] 204 Woodland
5. Bette J. Laycock 204 Woodland 391-6993
6. Christopher Adams 1006 Beach 391-6993
7. Walter Adams 1004 Beach 587-3618 938-6373
8. [illegible] Pavlish 307 Birch 391-0401
9. Juanita Barnes 1201 Dahlia 938-5536
10. [illegible] Richard 611 Riverside Dr 587 [illegible]
11. [illegible] Biermann 711 [illegible] 938 [illegible]
12. Lewis Biermann [illegible]
13. [illegible] Stewart [illegible] Times Beach [illegible]
14. [illegible] Stewart [illegible] Times Beach [illegible]
15. James & Betty DeClue 527 Oak Rd. times beach, Mo [illegible]
16. [illegible] Atchison [illegible] Bradley [illegible] Beach
17. Joyce Hall 530 Primrose Times Beach, Mo 587-3[illegible]
18. Elmer Hall 1407 Bradley Times Beach 587 [illegible]
19. David H Hall 535 Forest Times Beach, Mo 587-706[illegible]
20. James & Virginia Davis 32[illegible] Dogwood Times Beach [illegible]

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
Lary W. Ewiott	21 Driftwood	587-2501 or [illegible]
[illegible]	24 [illegible]eland	569-634[illegible]
Jack Johnson	201 Ivy	966-3349 587-3452
[illegible] JOHNSON	803 BLAKEY	567-6263 938-4149
Len Norman	210 Beach —	—
DeWayne A. Linilly	520 Dogwood	
[illegible] Linilly	520 Dogwood	9385876
Kenneth C. Paul	615 Park Dr	587-7169
Danny Biermann	215 Hawthorne	587-3224
[illegible] Biermann	215 Hawthorne	587-3224
David E. Freeman	612 Grove	837-5983
Fred G. Terreau	[illegible] RD	587-2638 343-8533
Emil E. [illegible]eimer	505 Forest	938-6163
[illegible]	232 Forest	938-6613

ATTENTION HOMEOWNERS OF TIMES BEACH:

If you the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition, Thank You.

1 Ray Wehner 2. John Lynn
15 Aspen Dr
Times Beach MO. 63025

3. John Sowards 938-4980 215 Birch, 212 Birch & 210 Birch Times Beach 630..
4 Geo. Sowards 632 Birch Times Beach, mo 63025 587-7432

5 6 Mr & Mrs. Ardie Jolliff - 409 Forest, 421 Forest, 421 GROVE
Times Beach Mo. 63025 - (314) 938-420
(Single family dwellings)

7 NORBERT F. SCISM 411 MAPLE TIMES BEACH 938-6622
8. Faye Buxton 309 Hawthorn, Times Beach
9 Don Buxton " " "
10 Ernest Jones 537 Birch 938-6948
11. Patsey Jones 537 Birch 938-6958
12 Charlie 13 Julie Buchanan 416 FOREST 938-5530
Boyd Beckmeyer 123 IVY 938-6474
1 LaDonna Beckmeyer 123 IVY 938-6474

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
Carol S Worthy	614 Birch Dr. Times Beach	257-2148
John Worthy	614 Birch Dr. Times Beach	257-2148
Gary Gray	531 LAUReL	343-6693
Beverly Biermann	215 Hawthorne	825-6439
Danny Biermann	215 Hawthorne	825-6439
Carol L Utz	401 Hawthorne	394-9162
Danny Utz	401 Hawthorne	394-9162
Russell Utz	401 Hawthorne	394-9162
Alan Manetzke	317 PARK	587-2874
Celine Oswald	501 Beach	938-5093
Larry D. Curtis	510 Hawthorn	938-4952
Harold Goodman	56 WOODLAND	257-38

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
William Henry Reid	406 Dogwood	
[illegible] Reid	406 Dogwood T.B.	
[illegible] Brown	518 Narcissus T.B.	

NAME	ADDRESS	
[illegible] Bennett	407 Riverside Times Beach	
Fred G Ferreau	804 BLAKEY RD	
[illegible] Summerholder	200 Forest Times Beach	
[illegible] Summerholder	202 Forest Times Beach	

NAME	ADDRESS	PHONE NUMBER
William E Turner Jr	520 Birch	938-4214
[illegible] Kloepper	532 Birch	938-553
[illegible] J. Turner	520 Birch	938-4314
[illegible] Kloepper	532 Birch	938-558
[illegible]	407 Riverside Dr.	

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
Sylvia Winebaugh	109 Forest Hill Times Beach	699-177
Shelia Hamil	224 FOREST TIMES BEACH	938617
Connie La Crone	211 Dogwood Times Beach	257-375
Duke La Crone	211 Dogwood Times Beach	257-3
Lewis L Biermann	710 Park Times Beach	225-4
Linda D Biermann	710 Park Times Beach	22547

ATTENTION HOME OWNERS OF TIMES BEACH:

If you the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition, Thank You.

1. [illegible] 1127 BLAKEY 587-7436
2. Linda Stewart 1127 Blakey 587-7436
3. Mary Smith 405 Laurel 938-6272
4. Lem Smith 405 LAUREL 938 6272
5. James P. Lindquist 609 Birch - 938-6917

Audrey Carr 522 Cedar Rd 587-3282
Lloyd Crane 318 GROVE 587-3094
Henry Horton 224 Juniper 5877256
Kath Horton 224 Juniper 5877256
Harold D Johnson 517 Riverside 467-6624
Norman L Richard 611 Riverside Dr. 587-3077
Howard Baird 519 Riverside 938-6306
Dorothy Meyer 236 Cedar 587-2049
Bobby L. Meyer 236 Cedar 587-2049
Linda Armamon 4 H Elm 587-2564
Grace & Frank Everhart 420 Ivy 938-4415
Emma J. Kunce 520 Birch 938-7314
Linda Klopper 533 Birch 938-5583
Clifton Neeley 328 Birch — 587-2735

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? If so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
[illegible]	[illegible] Grove	
[illegible] Doorack	328 Grove	587-[illegible]
Ione Lotter	225 Grove	587-7438
Mrs Edward Means	[illegible] Narcissus	
Robert Lyle	316 Narcissus	
Thelma Lyle	" "	
Ester White	[illegible]3 Dahlia	[illegible]87-237[illegible]
Betty [illegible]	5[illegible]26 Oak	
1 [illegible] Kerns	53[illegible] Oak	
Russ Lichler	514 Oak	
Mary Lichler	" "	
2 Roy Mason	553 Laurel	587-35[illegible]
3. Betty Mason	553 Laurel	[illegible]-3590
1. Ray [illegible]	[illegible] Chieftain	

ATTENTION HOME OWNERS OF TIMES BEACH:

If you, the people and homeowners of Times Beach, Missouri had an opportunity to sell your house now, at market value, WOULD YOU? so please sign this petition. Thank You.

NAME	ADDRESS	PHONE NUMBER
Charlie & Julie Buchanan	416 FOREST	938-5530
Billy – Marjorie Buchanan	418 FOREST	938-6627
Ernest & Virgie Hance	147 Cedar	587-2075
Linda Keith Young	605 Juniper	938-6219-532-15
ROBERT LOWERY	406 LAUREL	938-6709
Steve Berner	200 Juniper	5877356
Bud ELLIOTT	321 HAWTHORNE	9384477
Leo Berner	314 Hawthorne times Beach	5873360 Boyer
Tom Donna Hance	323 CEDAR	587-3608
Mr & Mrs Leo Coffman	238 Forest Times Beach	93868[illegible]0
Marilyn & Bill Lawter	532 Maple	938 5035

INTERIM REPORT OF THE MISSOURI DIOXIN TASK FORCE

submitted to
Governor Christopher S. Bond

JUNE 1, 1983

June 1, 1983

Honorable Christopher S. Bond
Governor
State of Missouri
State Capitol
Jefferson City, MO 65101

Dear Governor Bond:

I hereby convey to you the interim report of the Dioxin Task Force as requested in your executive order 83-5.

Since its inception in February of this year, the task force has studied the volume of information on dioxin presented in papers and speeches by experts in the field. The recommendations in this report reflect that study.

The task force appreciates comments on this report as well as further guidance on the final report or any other matter you wish the task force to address.

Respectfully submitted,

James A Finch

James A. Finch, Jr.
Chairman
Governor's Dioxin Task Force

JAF:brd

PREFACE

Explanation of Terms

This report deals with one extremely toxic member of a class of compounds commonly called "dioxins". Within the 75 possible chlorinated dioxins, there are 22 isomers of tetrachlorodibenzo-p-dioxin (TCDD). In Missouri, the contamination is almost exclusively with one highly toxic member of this family, the member "2,3,7,8-TCDD". For simplicity and unless otherwise stated, this report uses the terms "2,3,7,8-TCDD", "TCDD", and "dioxin" interchangeably, all referring to one member of a family of compounds.

EXECUTIVE ORDER
83-5

WHEREAS, extensive laboratory testing has confirmed the presence of dioxin at several locations in Missouri; and,

WHEREAS, dioxin is a potentially deadly chemical which threatens the health and safety of thousands of Missourians,

NOW, THEREFORE, I, CHRISTOPHER S. BOND, GOVERNOR OF THE STATE OF MISSOURI, by virtue of the authority vested in me by the Constitution and laws of Missouri, hereby establish the Governor's Task Force on Dioxin (hereinafter the "Task Force") which shall be composed of such residents of Missouri as from time to time may be appointed by the Governor. The Task Force shall include representatives of Missouri higher education institutions and Missouri companies which have expertise regarding hazardous wastes, as well as concerned citizens. The Chairman of the Task Force shall be designated by the Governor.

The purposes of the Task Force shall be as follows:

1. To examine and evaluate all known methods, processes and technologies for the destruction and containment of dioxin, particularly with respect to dioxin as it exists as a contaminant in soil and stream sediment at various locations in Missouri;

2. To examine and evaluate the public health dangers associated with dioxin, particularly with respect to dioxin as it exists as a contaminant in soil and stream sediment at various locations in Missouri;

3. To prepare and present, not later than June 1, 1983, an interim report which, inter alia, provides specific recommendations for solving those public health and environmental problems occurring in Times Beach, Missouri which are the result of dioxin contamination exacerbated by widespread flooding; and

4. To prepare and present, as soon as practicable but not later than October 1, 1983, a final report which includes the evaluations set forth in paragraphs 1 and 2 above, and which recommends a practical and effective plan of action for implementing comprehensive and permanent solutions to the public health and environmental problems caused by dioxin contamination in Missouri.

The Director of the Department of Natural Resources and the Director of the Division of Health shall make available such staff and support resources as are needed by the Task Force in the course of its deliberations. All meetings of the Task Force shall be open to the public, and a record shall be kept of all Task Force proceedings.

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Members of the Task Force shall receive no compensation for their services, but shall be reimbursed from funds provided through the Department of Natural Resources for their reasonable and necessary expenses. The Task Force may contract for the services of such consultants and experts as it deems necessary and shall pay for such services from funds provided through the Department of Natural Resources or the Division of Health.

The Task Force shall meet at the call of the Chairman, or at the call of the Governor or his designate, and shall dissolve upon the issuance of its final report, unless extended by subsequent Executive Order.

IN TESTIMONY WHEREOF, I have hereunto set my hand and caused to be affixed the Great Seal of the State of Missouri, this 14th day of February, 1983.

Christopher S. Bond
GOVERNOR

ATTEST:

James C. Kirkpatrick
SECRETARY OF STATE

GOVERNOR'S DIOXIN TASK FORCE

The Honorable James A. Finch, Jr.
Judge
Chairman, Governor's Dioxin Task Force

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Jeff Staake, Dioxin Activities Coordinator

Governor's Remarks
Dioxin Task Force Meeting
February 23, 1983

Judge Finch, Members of the Task Force, Ladies and Gentlemen:

Let me extend first of all my warm welcome and my sincerest thanks to all of you who have agreed to serve on the task force. We've brought this distinguished group together to assist us in solving what has become to be Missouri's most serious challenge we face in the environment today. That is, of course, ridding our environment of dioxin, a deadly chemical which threatens the health and has disrupted the lives of many Missourians. I am most grateful that you've accepted this challenge to serve on the task force. It is a very timely meeting that we have today and there is much work to be done. As Fred indicated, as you know, yesterday the EPA and the federal task force on Times Beach proposed a buy-out for the residents of the Times Beach area. We are hoping also to have a similar conclusion to the Minker residence site, which will enable us to get to work immediately on the most pressing and most immediate problem, that is moving people from the sites from direct contact with dioxin or the likelihood of it. It will enable you to concentrate on the scientific responses which we must make on a longer term basis to the problems of dioxin. During the past few months, obviously, we have been bombarded with a great deal of information on this deadly chemical and what it means to the residents of many Missouri communities. I've had the opportunity over the last several months to visit personally with the people whose lives have been disrupted, whose families have been subjected to a great deal of concern over the presence of dioxin and to their fears as to what it might cause them in the future in the way of health and other problems.

In southwest Missouri where much of the dioxin was produced citizens are fearful there, however, on a continuing basis. Their streams and waterways may have been contaminated threatening wildlife, fish, as well as the health of area residents. In this area of the state where tourism is so very important we have to take strong action to ensure that clean waterways will continue to provide us with abundant fish, wildlife, and water supply. The people of southwest Missouri know that we are doing everything possible to protect the natural surroundings while we look for a way to remove permanently the threat of dioxin from their environment.

Or eastern side of the state, as you well know, families have been told to leave their homes to protect themselves from the health hazards of dioxin contamination in their yards and the streets of their communities. Gardens where once vegetables grew and children played are now empty because the fear of dioxin contamination has made citizens fear for their health and the health of their children in particular.

I believe the time has come for us now to provide some straightforward answers and a plan of action to combat the presence of dioxin in a way that will lower public anxiety and lead to some long-range permanent solutions. We were all very much relieved, as I indicated earlier, to hear that the residents of Times Beach will be offered a buy-out from the federal government. Yesterday I met with the leaders of the Missouri General Assembly to ask them for their approval and I've also asked the Missouri Legislature to provide our share of the cleanup and relocation expenses. The action at Times Beach and what we hope will occur at the Minker residence site should help relieve the human suffering that these families have endured. Still, other families at other sites, discovered and yet to be discovered, await the answers that I hope this blue ribbon task force will find. That is a way to remove permanently the dangerous levels of dioxin from the soil in Missouri and to control dioxin where it is present in levels above that which is safe.

Let me provide you with a brief recount of the events which led up to our current situation. Let this be helpful for background for you and give you context within which to place your considerations. Fred Lafser and his staff will be providing you with more details on the work of the Department of Natural Resources. Dr. Hotchkiss will be giving you briefings on the health aspects and the determinations by the federal government, in particularly the Centers for Disease Control. Also, you will hear from Mr. Rice with the comments on the role of the Environmental Protection Agency, what they have done and what they seek to do in the future.

You might recall that last year, last December actually, after months of wrestling with this problem and sometimes making successful advances with the federal government and sometimes not, I went to Washington and met with top EPA officials personally and that meeting gave us the information that we needed to protect the health and safety of people in the Jefferson County area who had been exposed to dioxin contamination. We finally learned that the EPA's tests showed there that the levels of dioxin at the so-called Minker residence site were in sufficient quantities that the health experts believed that they were significant risks to the people living there. On December 8, I ordered that the state offer temporary relocation assistance to the families at the Minker site and the Division of Health was ordered to begin medical evaluations of the individuals who may have been exposed to the dioxin. I also reiterated my requests at that time that the EPA Superfund be used to find a permanent solution to the contamination of these areas and we requested that the EPA accelerate its testing process on samples from the sites identified potentially as dioxin sites throughout the state of Missouri.

After that as you well know the heavy rains of December added another and even more volatile dimension to the dioxin problem. Times Beach, the site of EPA testing for dioxin on road beds, had been virtually destroyed by severe flooding, 100-year floods which afflicted that area. As the flood waters were receding on December 23, just about Christmas Eve, the U.S. Department of Health and Human Services Center for Disease Control in Atlanta advised us that the pre-flood test samples of the Times Beach areas showed levels of dioxin that could be dangerous. The CDC recommended because of the uncertainties left behind in the mud and debris from the flood that non-emergency cleanup activities in Times Beach cease, that residents who had temporarily relocated stay out, and that residents who had already begun to move back in, leave. That day at my request the EPA agreed to send in new testing teams to conduct post flood dioxin tests. The federal Disasters System Center in Fenton, Missouri agreed to reopen on December 27 and stay open as long as necessary to help the Times Beach residents relocate. I then directed our Department of Public Safety, Division of Health to go back to Times Beach and assist with law enforcement and public health activities.

Since that time state officials have been working as they could with a variety of federal and local officials who assist with relocation plans, test sampling, and cleanup activities. As we developed a solution to the flood debris problem in Times Beach, however, as we continued to urge the EPA to speed up its testing, it became clear that fragmented federal responsibility was becoming a substantial impediment to both emergency responses and long-term solutions. In some instances the recommendations and ideas of one particular federal agency did not square with the advice which was being given by other federal agencies. Therefore, I wrote and telephoned the White House to ask the President to immediately establish a federal task force to bring together all appropriate agencies and that a single federal agency and official be designated to take the lead responsibility in Times Beach. The President that same day appointed a federal coordinating group, task force, and named Lee Thomas of the Federal Emergency Management Agency as Chairman. The next day Mr. Thomas and I visited Times Beach and had discussions there and in St. Louis. We continued to work with federal officials to expedite their handling of the problems in all areas of Missouri. It appears that we are beginning to see some significant progress in handling the human problems associated with dioxin. Yesterday as we said the federal officials announced they would buy out the dioxin contaminated property in Times Beach, giving residents there a chance to move out permanently and to begin to rebuild their lives elsewhere.

Meanwhile the residents of the Minker site with whom I visited on Monday are still waiting for an answer to their problems. I strongly urged, and repeat that urging, that federal officials also buy out the homes at the site. We've been promised an answer three weeks from the time of that request which is about two weeks from today.

Unfortunately, as we all know, the Missouri dioxin problems do not stop at Times Beach or the Minker site or even the eastern Missouri sites. Because hazardous waste generators and haulers were unregulated for so many years in Missouri and elsewhere the problem of dioxin and other hazardous chemicals is likely to appear and re-appear throughout Missouri as it will throughout the nation for years to come. Extensive sampling and testing by the U.S. Environmental Protection Agency has confirmed the presence of dioxin at 22 sites in Missouri. I expect that more will be found as the testing continues on the some 80 to 90 sites identified as potential dioxin sites throughout the state. We really are in a situation where we have no precedent for solving this problem. No where else has there been found so much large volume of contaminated soil scattered at various locations under different conditions. That is why the state of Missouri needs your help to solve this complex problem. I ask you today to study very carefully all angles of the problem, keeping in mind the human suffering and the human health hazards that occur, as well as the dangers to the environment, and recommend to me solutions that are practical and technologically sound. To this end I request that you examine and evaluate all known methods, processes and technologies for the destruction and containment of dioxin as it exists as a contaminant in soil and stream sediments at various locations in Missouri. I also ask that you investigate the costs of each of these methods and the length of time required to take remedial action. To the extent possible, I would suggest that you coordinate your study with the ongoing investigations and studies of the U.S. Environmental Protection Agency. Yesterday, Mr. Hedeman in announcing the Superfund activities said that the EPA will be working with the state of Missouri to develop means of controlling, destroying, and containing dioxin. You may want to visit some of the sites where cleanups are underway or the sites where innovative solutions are being tested. Fred Lafser has already pointed out that we have already discovered through the efforts of Syntex some means of destroying dioxin where found in highly concentrated forms and we have some expertise which has already been developed in this area. I am confident that the problem can be solved but it will only be solved by cooperative effort by state, local and federal agencies. At several of the larger sites, obviously, if extensive cleanup is required to protect the health of citizens, we are going to need federal Superfund assistance once again. We also are going to need the assistance of the best knowledge, the best ability, in the private sector. The number of nationally known companies with a reputation for helping solve dioxin problems have written to state government and suggested ideas for dealing with the dioxin problem. I am sure that they would be willing to discuss these ideas with you. We hope that from these sources and other sources which will be available to you, we will be able to come up with some answers. Again, Mr. Lafser and his staff will be able to assist you in your task.

The heart, of course, of the dioxin problem is the human health problem. There has been a great deal of confusion and anxiety regarding the public health dangers associated with dioxin, particularly as it exists as a contaminant in soil and stream sediments in locations throughout Missouri. I'd ask that you examine the data, the analyses provided by the Federal Centers for Disease Control and the Missouri Division of Health. Based on this examination I'd like you to assess for us the public health dangers of dioxin contamination and recommend what other services the state should be providing. Bob Hotchkiss of the Division of Health will be presenting information on these issues this morning and he and his staff will be able to assist you in that part of the assignment.

You should also be aware that I have recommended to the Missouri General Assembly legislation to help combat the dioxin problem. I think some of these actions represent the basic commitments that our state must make. I proposed in January an immediate $1 million in emergency appropriation for immediate relocation of families and cleanup activities. As I mentioned earlier, I have just yesterday urged the General Assembly to appropriate on an emergency basis another $3.5 million for the state match for the buy-out of the Times Beach property and relocation of citizens from that area.

Second, I have proposed that we immediately add five new environmental health officers to the Division of Health on an emergency basis and add two more environmental health officers next year.

Third, we proposed the hiring of a hazardous waste criminal investigator and three environmental landfill specialists to the staff of the Department of Natural Resources so that we may follow up and investigate suspicious circumstances and proceed with prosecution of anyone violating our hazardous waste laws.

Fourth, I've proposed an expansion of the Missouri Superfund, increasing fees on hazardous waste generators, transporters, and waste site operators, to provide us with the mechanisms and the resources to deal with these problems.

As you see the fifth proposal I made to the legislature was the Governor's Task Force of scientific advisors and concerned citizens, the long-term solution, and that is you, ladies and gentlemen. When I said we were going to get a blue ribbon task force, I was generally aware that we had some very unusual and special talents in this state and your presence here today, your willingness to serve and your dedication to these efforts convinces me that we do have the resources in our state that we need to solve the problem.

I'm happy to report that the bills that have been recommended are progressing in the General Assembly and with passage of the legislation we should be able to provide some immediate relief and establish the groundwork for better management of hazardous waste in the future. In the meantime, I turn to you for your help in identifying the permanent comprehensive solutions to the dioxin problem in Missouri. Make no mistake about it, this is a serious and difficult challenge. The nation is watching to see how Missouri will solve the dioxin problem we have here. We must show that we are up to the challenge and prepared to emerge as a national leader in responding to hazardous waste problems. Missouri is not the only state that has hazardous waste problems. They have a particular problem because of the way in which the dioxin was spread across our state and it is a particularly difficult challenge, but the expertise, the knowledge and the information we gain here not only can help the citizens of our state, but I believe can help provide some ideas and leadership for other states in the nation as they too must face their hazardous waste problems.

In the end I trust that we will not only solve the problem, but will restore pride that we have in our state, a pride that has been sagging in some areas in recent months as we have seen on television and heard on the radio and read in the newspapers as the nation has focused on our dioxin problems. When we have found those solutions, Missourians will thank you for the important role that you have played. Today once again, I extend my sincere thanks to you for your agreement to accept this responsibility. I've asked for your time, your dedication, your patience, and your expertise. That combination I am convinced will enable us, in cooperation with the other agencies and the other entities involved, to come up with the solutions that we must have.

x

EXECUTIVE SUMMARY

TASK FORCE RECOMMENDATIONS

This interim report is not limited to Times Beach, but has been expanded to include the Minker/Stout site because this later site is considered to be unstable since movement of contaminated soil is occurring. Following is a summary of the actions that are needed to deal with the problem at these sites. The final report will deal with the problem as it exists statewide.

MINKER/STOUT

- the buyout should be expedited.
- access to the site should be restricted.
- initial stabilization measures should be implemented.
- surface and groundwater sampling should be conducted.
- planning for final remedial action should continue with all deliberate speed.
- more complete health studies should be considered.

TIMES BEACH

- the buyout should be expedited.
- access to the site should be restricted.
- additional sampling of the surface and groundwater should be conducted.
- other than limiting access, no initial remedial actions are recommended unless contaminant migration is found.
- additional soil sampling should be done to determine extent and depth of the concentration.
- more complete health studies should be considered.

AVAILABLE TECHNOLOGY

- test burn soils with varying concentrations of TCDD to demonstrate destruction by incineration and to determine if the ash is non-hazardous.
- continue study and research on solvent extraction and fixation.
- locate site for a storage treatment facility.
- landfilling is to be used only as a last resort.

FURTHER RESEARCH

- the human health effects of dioxin.
- the health of residents of contaminated sites.
- the bioavailability of dioxin in soil.
- stability and transport of dioxin in the environment.

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INTRODUCTION

TASK FORCE ACTIVITY

For the past four months, the Dioxin Task Force, appointed by Governor Bond, has conducted an intensive study of the dioxin situation in Missouri. The Task Force has heard testimony and reviewed studies made by experts from throughout the country regarding the environmental and health effects of dioxin. The Task Force also has conducted a study of available technology for disposal of dioxin and has made an assessment of those technologies based on applicability to the specified sites being considered in this report.

As requested by Governor Bond, the recommendations in the interim report deal specifically with the Times Beach site. In addition, the Task Force also related all available information to the Minker/Stout site, another area of high priority. While many recommendations made will be applicable to other sites in Missouri, detailed discussion of those sites will not be made until the final report due in October.

HISTORICAL SUMMARY

In 1969, the North Eastern Pharmaceutical and Chemical Company (NEPACCO) began producing 2,4,5-trichlorophenol (2,4,5-TCP) as an intermediate for hexachlorophene in a Verona, Missouri manufacturing facility leased from the Hoffman-Taff Chemical Company and later Syntex Agribusiness Inc. In the manufacturing process, the product stream was distilled and filtered to remove impurities. Dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD), which was formed in trace quantities during the manufacturing process for the TCP, was removed from the product during filtration and distillation. The filtration and distillation residues thus became contaminated with dioxin. Unfortunately, it was not known at the time the still bottoms contained TCDD.

Missouri's current dioxin dilemma can be traced to May 1971. At this time the problem came to the attention of state and federal officials when a child was hospitalized after playing in a horse arena where numerous horses and other animals had become ill and died. Between 1971 and 1974 officials of the Missouri Divison of Health (DOH) and the federal Centers for Disease Control (CDC) conducted an investigation to determine the cause of these problems. In the investigation, it was discovered that some additional people were ill and that several stables had been sprayed in 1971, as a dust control measure, by Russell Bliss, a waste oil hauler from St. Louis. It was not until July of 1974 that it was determined that a toxic form of dioxin (TCDD) was the causative agent in the oily wastes. Until that time, there was neither reason to suspect that TCDD was involved, nor reason to link these problems with NEPACCO's manufacturing processes in Verona, Missouri a few years earlier.

Meanwhile, the contaminated soil at the horse arenas had been removed by the owners of the arenas and disposed of in various locations. Some of the contaminated soil from the Bubbling Springs arena in Fenton was used as fill material at the Minker and Stout residences in Jefferson County.

On August 2, 1974, the CDC informed the Division of Health that dioxin had been found in the samples collected at the Shenandoah Arena (31.8 to 33 ppm). The CDC then accepted the invitation of Dr. Denny Donnell, state epidemiologist, to participate in a renewed investigation. Dr. Patrick Phillips, of the Division of Health, and Drs. Coleman Carter and Matthew Zack (CDC) began an intensive investigation of the problem.

On August 11, 1974, Dr. Phillips met with Judy Piatt, owner of the Shenandoah Arena, to explain the toxic effects of TCDD and to refer the exposed persons to family physicians for evaluations. On August 13, Phillips learned that the Bubbling Springs arena also had experienced similar horse illnesses. On August 14, 1974, Dr. Phillips interviewed persons connected with Bubbling Springs and obtained information about animal and human illnesses; he also obtained information about sites where the excavated arena soil had been taken (Minker and Stout residences). Phillips interviewed Vern Stout and Valerie Minker on August 14 concerning soil excavated from the Bubbling Springs Ranch arena and learned that the soil was deposited at the Minker residence in March 1973. He informed both persons of probable dioxin contamination and obtained samples of soil for analysis. Test results later showed the soil from the Minker residence to contain 0.74 ppm dioxin and soil from the Stout residence to contain 0.44 ppm dioxin.

The dioxin investigation continued in August and September of 1974 and the toxic wastes were traced to their origin at the Syntex plant in Verona. It was discovered that NEPACCO had bought equipment and leased plant space from Hoffman-Taff (since acquired by Syntex Agribusiness, Inc.) and had used Bliss Waste Oil for disposal of some of its wastes. It also was learned that a tank containing approximately 4,300 gallons of distillation residue wastes from NEPACCO's operation still existed on Syntex plant property at Verona. Tests later showed that TCDD was highly concentrated in the tank's contents at approximately 350 ppm.

For the next few years this tank containing high concentrations of TCDD became the chief concern and focal point of activity on the Missouri dioxin issue. On October 2, 1974, Dr. Clark W. Heath, Jr., director of the Cancer and Birth Defects Division, Bureau of Epidemiology, CDC, wrote a letter to Dr. Denny Donnell, Jr. at the Missouri Division of Health, emphasizing the definite hazard of the wastes at the tank in Verona: "...it is important for the public safety that this material be promptly and safely destroyed." This letter apparently served to focus the attention of state officials on the potential hazard of the wastes in the tank at Verona. Health recommended incineration of wastes from the tank and deep burial of the tank itself.

The final CDC report on the investigation (March 1975) pointed out that the expected half-life* of dioxin was one year and recommended: a) that inaccessible contaminated soil remain undisturbed; b) that soil in residential fills be removed to a landfill; c) that the contents of the storage tank (at Verona) be properly incinerated; d) that the tank be marked and deep buried; and e) that symptomatic persons be followed up. This report was reviewed by the DOH and transmitted to the Missouri Department of Natural Resources (DNR). Because of earlier CDC advice, Missouri officials focused on items c) and d). All symptomatic persons were known to be under medical care.

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*For a discussion of the term "half-life," see Chapter 7.

The recommendation to remove contaminated soil from residential locations was discussed by DNR and DOH. In light of the knowledge of the expected half-life of dioxin in soil in 1975, levels approaching minimum detectable limits at that time, and the apparent stability of the sites, it appears that a decision was reached not to remove the soil but to resample in an effort to confirm the anticipated degradation rate of dioxin. Dr. Phillips visited the residential sites in September 1976; he confirmed that the soil was not eroding and that vegetation had been established, and he took additional samples which were sent to the CDC lab. (Results were obtained in a phone conversation between Dr. Phillips and Dr. John Liddle, head of the CDC Lab, on November 22, 1982.)

From October 1974 to August 1977, there were many frustrating attempts to find a method for incinerating the wastes from the tank in Verona. In August 1977, Syntex officials reported on a new photolysis process which could detoxify dioxin. Essentially, this process involved breaking down compounds with ultraviolet light. Continued investigation showed this to be a preferable method of destruction, and in September 1979, a final decision was made to use the photolysis method. By August 1980, virtually all the 4,300 gallons of waste had been processed. Nearly 13 pounds of dioxin had been destroyed, reducing the original 343 parts per million dioxin content by more than 99 percent. Syntex had become the first company to successfully and safely degrade large amounts of dioxin by photochemical means.

While the Verona tank problem was being solved,the EPA received an anonymous call alleging that other sites in the southwestern part of the state had received NEPACCO wastes. Follow-up investigation led to the discovery of the Denney Farm site; Baldwin Park in Aurora; the wastewater treatment school in Neosho; the Crider, Erwin, Ray, and Rusha farm sites near Verona, and the potential dioxin contamination of the Spring River.

Beginning in October 1979, attention was focused on the Denney Farm cleanup. The actual cleanup, conducted by Syntex with EPA approval, began in September 1980 and was completed in the summer of 1981.

In January 1981, the wastewater treatment school at Neosho was sampled; certain areas were secured and partially cleaned up by EPA.

In November 1981, Ron Crunkilton of the Missouri Department of Conservation and Dan Harris of EPA collected fish from the Spring River to be tested for dioxin. Preliminary test results received in February 1982 indicated the presence of dioxin in the fish tissue. The Missouri Department of Natural Resources, Division of Health, Department of Conservation, and the EPA conducted public meetings in March 1982 to announce the fish sampling results and issued an advisory to limit consumption of fish from the Spring River.

Also, in early 1982, EPA officials announced their intention to resample the horse arenas and the associated sites where excavated soil had been placed. This sampling took place in May and June 1982; the results were obtained in August and made public on August 18. Subsequent events leading to intensive sampling of numerous sites in eastern Missouri are well known. The recent buyout of the residents of Times Beach has made national headlines out of Missouri's dioxin problem.

Overall, the 11-year-long investigation of dioxin had several phases:

May 1971 - August 1974

Phase I: First appearance of animal and human health problems at horse arenas and investigation determining that dioxin in wastes from the plant at Verona had been the cause.

October 1974 - October 1979

Phase II: Attention focused on the highly contaminated wastes in a tank at the Verona plant and the search for methods to destroy the dioxin in the tank.

October 1979 - April 1982

Phase III: Discovery and investigation of numerous sites in southwest Missouri including Denney Farm site and Spring River.

May 1982 - Present

Phase IV: Resampling of horse arenas and associated sites and investigation of new leads in eastern Missouri. Continued sampling of the Spring River and examination of other potential dioxin sites in southwest Missouri.

In Phase I, the investigation was primarily conducted by the Missouri Division of Health and the Centers for Disease Control.

As a result of state reorganization, the Missouri Department of Natural Resources was established on July 1, 1974. DNR established a hazardous waste project in January 1975 and initiated a major survey of hazardous waste generators in the state in order to determine the type and quantity of waste being generated.

DNR, DOH, and EPA cooperated during the Phase II efforts to find a method of destroying the wastes in the tank at Verona. From January 1975 through June 1977, DNR's primary emphasis, however, was on research and legislative activities leading to the passage of the Missouri Hazardous Waste Management Law in 1977 and later amendments in the Special Session of 1980. From July 1977 to the present, DNR's primary activity in hazardous waste has focused on the development and implementation of regulations to prevent mismanagement of currently generated hazardous wastes. However, DNR also has been increasing its assistance to EPA in the investigation and clean-up of abandoned and uncontrolled hazardous wastes sites, particularly since the 1980 amendments to the state law where authority for clean-up and some funding were provided. In fact, a 1980 report to the Governor prepared to support the legislation indicated there were approximately 100 such sites in Missouri.

In Phase III, the EPA was clearly in the lead in the investigation of newly discovered sites in southwest Missouri, and EPA continues as the lead agency in Phase IV with assistance from state agencies and other federal agencies. The passage of the federal "Superfund" Law in December 1980 greatly augmented the EPA's ability to investigate and cleanup abandoned and uncontrolled hazardous waste sites.

Some controvery has resulted from the March 31, 1975 CDC memo which, among other things, recommended excavation of fills at Minker and Stout sites. Although the records are not definitive and most of the personnel involved are no longer with state government, it is inferable from the facts at hand that state officials chose to concentrate their rather limited staff resources on the Verona tank wastes for several reasons:

1. The very high levels of dioxin in the tank at Verona.
2. The assumption that the TCDD in the tank would never degrade.
3. The advice from CDC and other sources that TCDD in the soil at other sites would degrade relatively quickly with probable half-life between 0.24 and 1.0 years.
4. The lack of a disposal or detoxification method for large volumes of soil.
5. The lack of clearly defined clean-up authority.
6. Lack of clean-up funds and staff for investigation and follow-up.
7. An assumption that the contaminated soil of Minker/Stout was stabilized and would not erode onto neighboring properties. Thus, as far as state and federal officials knew, all relevant parties had been notified when the Minker and Stout families were notified in 1974.
8. Less precise methods of measuring TCDD than are now available.
9. At Verona, a private party (Syntex) existed who was willing to provide money and expertise.

The dioxin issue in Missouri has been clouded with a lot of unknowns and controversies. Consider that the work extended for five years over how to detoxify the 4,300 gallons of concentrated material in the Verona tank that NEPACCO left behind. What to do with the dioxin-laced soils still is a question today and illustrates the frustrations and limited options available.

Overall, history shows the clear need for the statutes which were passed in the late 1970s and early 1980s. Legislators acted first to prevent new problems from the mismanagement of hazardous wastes. Then statutes were enacted to authorize clean up of the old abandoned and uncontrolled waste sites. Passage of the federal "Superfund" law has boosted efforts to clean up these dumps. Through adequate enforcement of the waste management laws now on the books, Missouri can prevent problems like the dioxin dilemma from ever happening again.

DIOXIN INVESTIGATION: CURRENT STATUS

The Missouri dioxin investigation began in the spring of 1971 when various parties began a search for the cause of animal deaths at a horse arena. After the problem was traced to dioxin in the summer of 1974, the investigation was focused on locating the source of the contaminant. The dioxin was quickly traced to the former North Eastern Pharmaceutical and Chemical Co. (NEPACCO) plant at Verona. It further was determined that the Bliss Waste Oil Co. had been subcontracted to dispose of wastes from the site, but instead mixed the wastes with used oil and sprayed several locations for customers as a dust control measure.

During the next several years, the investigation continued in the southwest Missouri area. Government officials concentrated on finding the means to destroy a large tank of highly contaminated material that was left by NEPACCO at its former plant site, a facility since purchased by Syntex Agribusiness Inc. Investigators worked to locate other areas nearby where dioxin-contaminated wastes were reported to be buried.

The pace of the investigation picked up dramatically in the summer of 1982 when results of an Environmental Protection Agency (EPA) testing program indicated that the dioxin molecule is far more persistent in the environment than originally thought. From late summer until mid-January 1983, the Environmental Protection Agency committed substantial resources to locate and document potential dioxin sites in the eastern Missouri area, while still maintaining efforts as necessary in southwest Missouri. Sampling was arranged where indicated. Some assistance was provided by specially assigned personnel from the Department of Natural Resources, who worked under the EPA's direction.

In mid-January, as a result of the increasing number of sites where follow up was required, the Department of Natural Resources' Division of Environmental Quality did assign personnel to coordinate a state investigation and complement the federal effort. The state and federal investigation coordinators agreed that the EPA would continue to handle primarily sites in southwest Missouri while state personnel would assist with the rapidly expanding caseload in eastern Missouri. During the major portion of the recent state and federal investigation, a combined force of about five persons has been following up on leads, interviewing, taking statements, seeking access permission, sketching and photographing sites, and outlining suspect areas in preparation for sampling. As new leads are acquired, the coordinators decide who will assume responsibility for followup.

An original list of 40 suspect sites has grown quickly, with changes occurring so fast that it is difficult to provide an accurate report on the status of the investigation. On a daily basis, sites are confirmed, new leads are acquired, old leads are found groundless, and sometimes one site becomes two.

Investigators are looking for information about sites that may have been sprayed by the Bliss Oil Co. with waste oil or other material at some point after May 1971. Sites sprayed close to this time are of greatest concern. Leads have been provided by arena owners, former Bliss employees, concerned citizens, and the state's "dioxin hotline". Investigators even pickup leads in the course of casual conversations. All leads are taken seriously and scheduled for followup according to their apparent value and risk posed to human health and the environment.

As of May 26, there were 31 sites in the state where dioxin contamination has been confirmed. An additional 102 sites were under investigation. There were 76 sites that have been investigated and determined by EPA to require no further action at this time.

CHAPTER 1

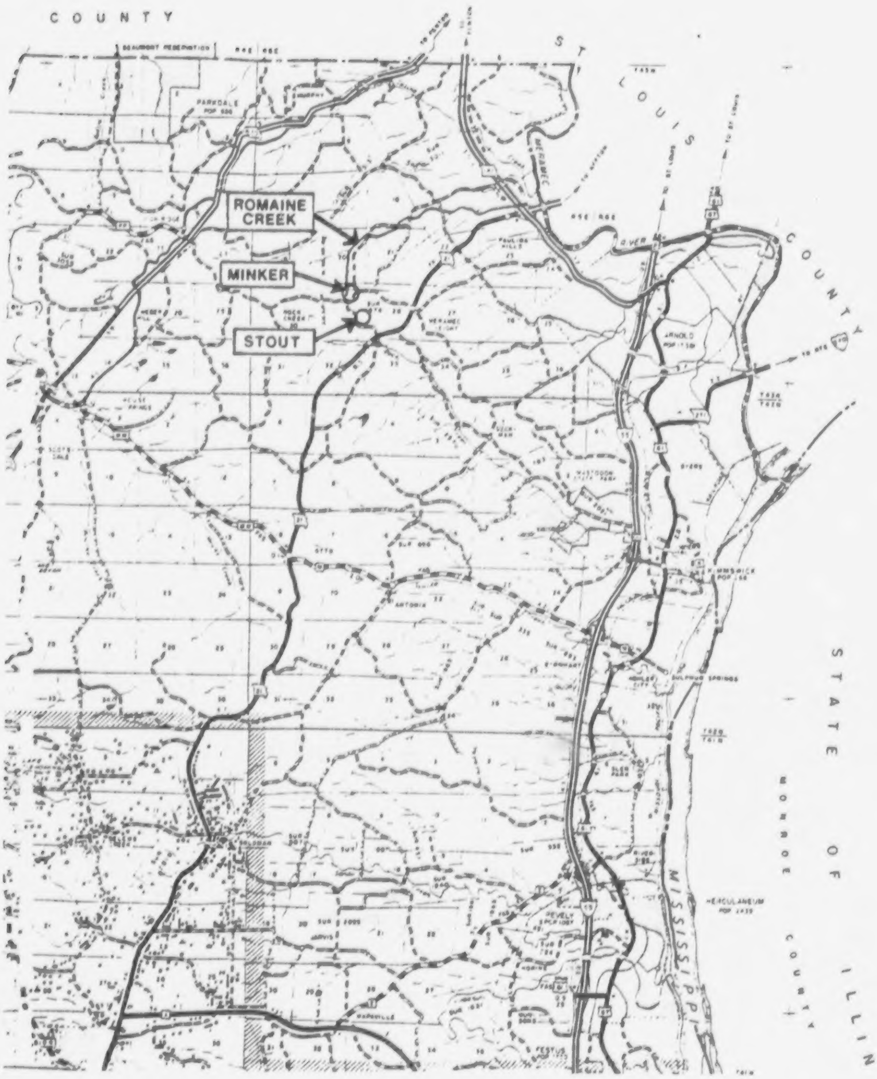
SITE SUMMARIES

This report contains specific information on the Minker/Stout/Romaine Creek and Times Beach sites. Therefore, this chapter presents information for these sites:

Location
Accessibility
Historical Summary
Site and Contaminated Area Description
Geologic and Soils Description
Unique Site Characteristics

Site maps for the Minker, Stout, and Romaine Creek sites were taken from information presented to the Task Force by CH_2M Hill, which is contained in Appendix VII. CH_2M Hill is EPA's feasibility contractor for the Minker/Stout sites.

Location maps and brief summaries of the present 31 confirmed dioxin sites are contained in Appendix VI. As the above information is compiled for all of the confirmed dioxin sites, it will be included in the final report.


COUNTY
ST LOUIS COUNTY
ROMAINE CREEK
MINKER
STOUT
PARKDALE
MERAMEC
RIVER
ARNOLD
KIMMSWICK
ANTONIA
SULPHUR SPRINGS
HERCULANEUM
PEVELY
FESTUS
MISSISSIPPI
STATE OF ILLIN
MONROE COUNTY

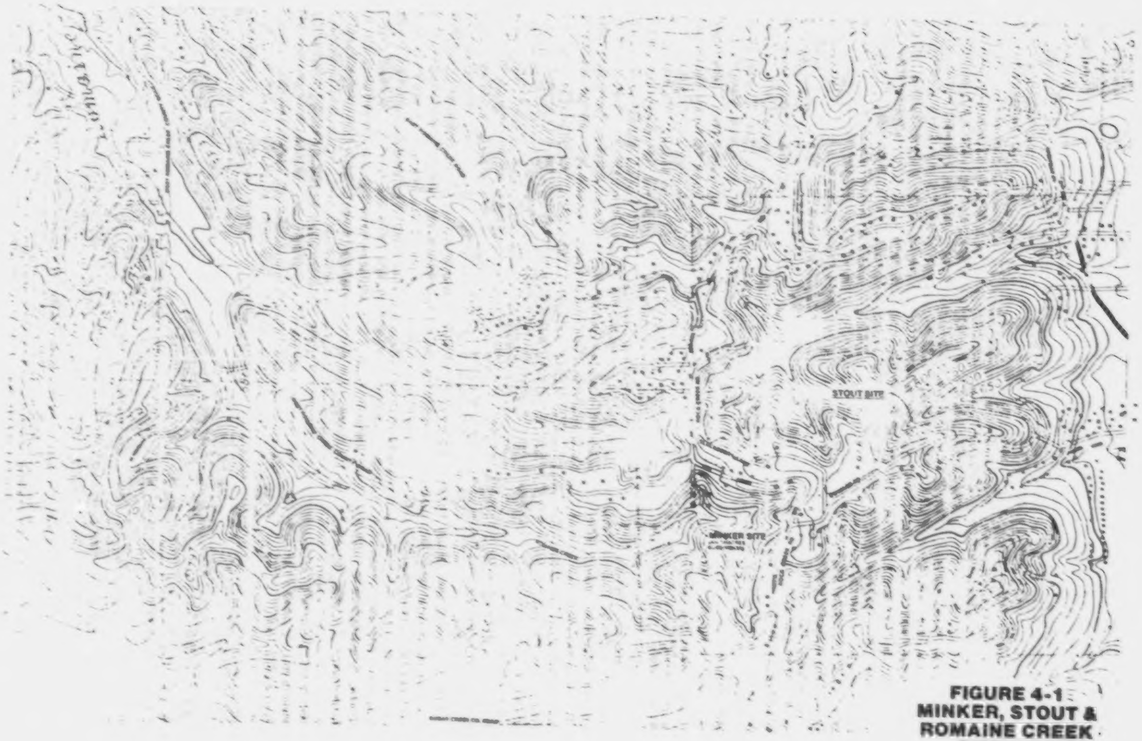

STOUT SITE
MINKER SITE

FIGURE 4-1
MINKER, STOUT &
ROMAINE CREEK
SITE MAP

MINKER RESIDENCE

Location

Legal Description: Anita Acres, a resubdivision of the north half of Lot 35 of Sunset Acres Subdivision in U.S. Survey No. 1974

By extrapolation of section lines: SE 1/4, NE 1/4, NE 1/4, Section 29, T. 43 N., R. 5 E., 5th P.M.
Maxville Quadrangle
Jefferson County
Latitude: 38° 26' 25"
Longitude: 90° 28' 10"

Address: 4037 West Rock Creek Road
Imperial, Missouri 63052

Accessibility

Access is west approximately 1.3 miles from Missouri Highway 21 on West Rock Creek Road, or northwest approximately 1.3 miles from Missouri Highway 21 on Swaller Road and West Rock Creek Road. West Rock Creek and Swaller Roads are low type bituminous.

History Summary

In June 1971, the Bubbling Springs Ranch horse arena was sprayed with contaminated waste oil. After several incidents of horse illnesses, the arena owner hired a private contractor to remove the soil which was suspected to be causing the problems. In March 1973 about 850 cubic yards was excavated, of which an estimated 260 cubic yards was used for fill at the Minker residence. The Center for Disease Control sampled the site in 1974 and found levels of 2,3,7,8-TCDD ranging from 85 ppb to 740 ppb. This site along with Romaine Creek and the Stout Site is included on the National Priorities List. A State Superfund contract has been signed with the State for these three sites. The contract covers relocation and a feasibility study for the sites.

Site Description (see maps)

The Minker Site lies at the head of Romaine Creek. The Minker house sits near the top of a ridge and the fill area is immediately to the south of the house. The toe of the fill abuts the neighboring yard. The fill was placed in a gully which carries storm drainage to Romaine Creek. Apparently, the fill began to erode almost immediately and most of the fill has now eroded downslope. Hence, the contaminated soil has been widely spread around other residences' yards downslope and into Romaine Creek. EPA sampling during 1982 and 1983 found 2,3,7,8-TCDD levels up to over 300 ppb. Volume of material has been estimated at 5000 to 8000 cubic yards for the Minker and Stout Sites combined.

Several homes are located across West Rock Creek Road from the Minker site. Testing has revealed no TCDD contamination of these properties.

Geologic and Soils Description

The Minker Site is in an upland setting underlain primarily by residual soil derived from weathering of a cherty limestone. A surface veneer of loess is present in limited areas and is usually not a factor in construction projects in the area.

The thickness of the residual soil can range from a few feet upwards in excess of 80 feet. For an average thickness, an assumption of 20 feet is valid for residual soil on the uplands and hillslopes. The clay portion in this soil could be as high as 40% or 50% with most of the remainder of the fine textured portion being made up of silt. The coarser fraction consists of angular to subangular chert fragments and these may range from 10% to as much as 40% of the soil deposit.

The Romaine Creek valley contains material eroded from the residual soil on the hillslopes. Thus, most of the alluvial material is a chert gravel material, with the surface soil being silt rich. The Bubbling Springs horse arena is located on the floodplain of the Romaine Creek.

In conclusion, the contaminated material used as fill at the Minker Site was similar to the residual soil, but having a higher silt content. Due to being spread out by erosion and mixed with residual material, the contaminated soil is primarily a stoney clay.

Unique Site Characteristics

The Minker Site is located in a dramatic topographical area which will complicate any construction or remedial activity. The contaminated material was located in a residential area, in yards and readily accessible to humans. The material is spreading through a large area (Romaine Creek), greatly enlarging the affected area.

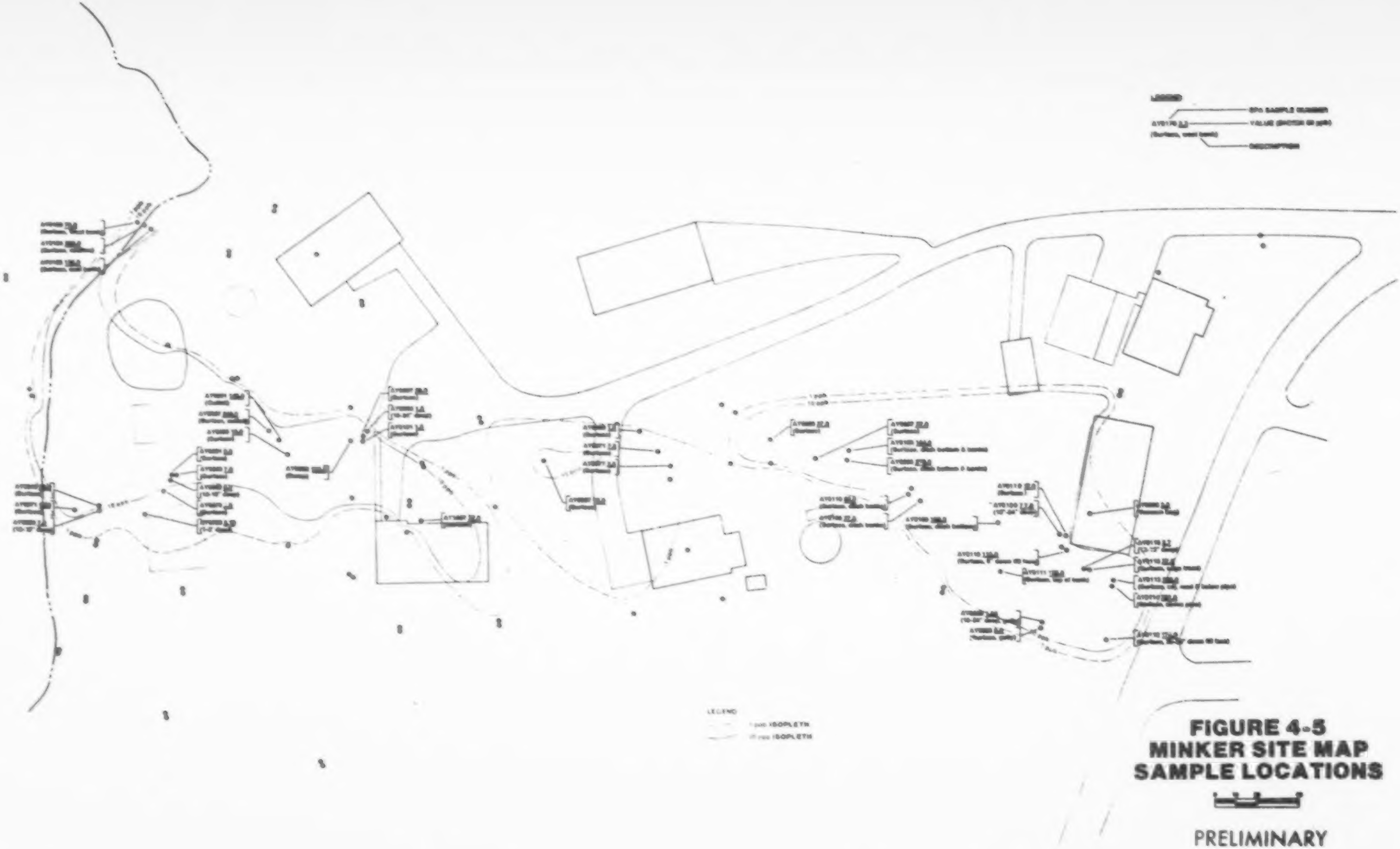

LEGEND
1 ppb ISOPLETH
10 ppb ISOPLETH

FIGURE 4-5
MINKER SITE MAP
SAMPLE LOCATIONS

PRELIMINARY

STOUT RESIDENCE

Location

Legal Description: U.S. Survey No. 1974

By extrapolation of section lines: SW 1/4, NE 1/4, SE 1/4
Section 29, T. 43 N., R. 5 E., 5th P.M.
Maxville Quadrangle
Jefferson County
Latitude: 38° 26" 0"
Longitude: 90° 28' 05"

Address: West Swaller Road
Imperial, Missouri 63052

Accessibility

Access to the site is northwest approximately 0.7 mile from Missouri Highway 21 on Swaller Road. Swaller Road is low type bituminous.

History Summary

In June 1971, the Bubbling Springs Ranch horse arena was sprayed with contaminated waste oil. After several incidents of horse illnesses, the arena owner hired a private contractor to remove the soil which was suspected to be causing the problems. In March 1973 about 850 cubic yards was excavated, of which the majority was used for fill at the Stout Site. The Center for Disease Control sampled the site in 1974 and found levels of 2,3,7,8-TCDD in the range of 170 ppb to 440 ppb. This site along with Romaine Creek and the Minker Residence Site is included on the National Priorities List. A State Superfund contract has been signed with the State for these three sites. The contract covers relocation and a feasibility study for the sites.

Site Description (see maps)

The Stout Site sits slightly to the east side of a north-south ridge. The fill material was used to make a level lot for two trailer pads. Reportedly, the fill operation was already in progress when the contaminated soil was brought to the site. Contamination was found throughout the fill by EPA sampling at levels from 1.0 ppb to 300 ppb, down to a depth of 20 feet. It is believed that 20 feet is the maximum depth of fill at any point. Volume of material has been estimated at 5,000 to 8,000 cubic yards for the Minker and Stout Sites combined.

Geologic and Soils Description

The Stout Site is in an upland setting underlain primarily by residual soil derived from weathering of a cherty limestone. A surface veneer of loess is present in limited areas and is usually not a factor in construction projects in the area.

The thickness of the residual soil can range from a few feet upwards in excess of 80 feet. For an average thickness, an assumption of 20 feet is valid for residual soil on the uplands and hillslopes. The clay portion in this soil could be as high as 40% or 50% with most of the remainder of the fine textured portion being made up of silt. The coarser fraction consists of angular to subangular chert fragments and these may range from 10% to as much as 40% of the soil deposit.

Surface runoff at the Stout Site would travel in a south-southeasterly direction to enter the main stem of Rock Creek east of Country Club Manor.

The contaminated fill originated from the floodplain of the Romaine Creek Valley, which consists of eroded material from the hillslopes, a clay and chert gravel material with a silt rich surface soil. Thus, the fill would partly consist of a stoney, silty clay material.

Unique Site Characteristics

The Stout Site is located on an extremely steep slope, which will complicate any remedial activity.

PRELIMINARY

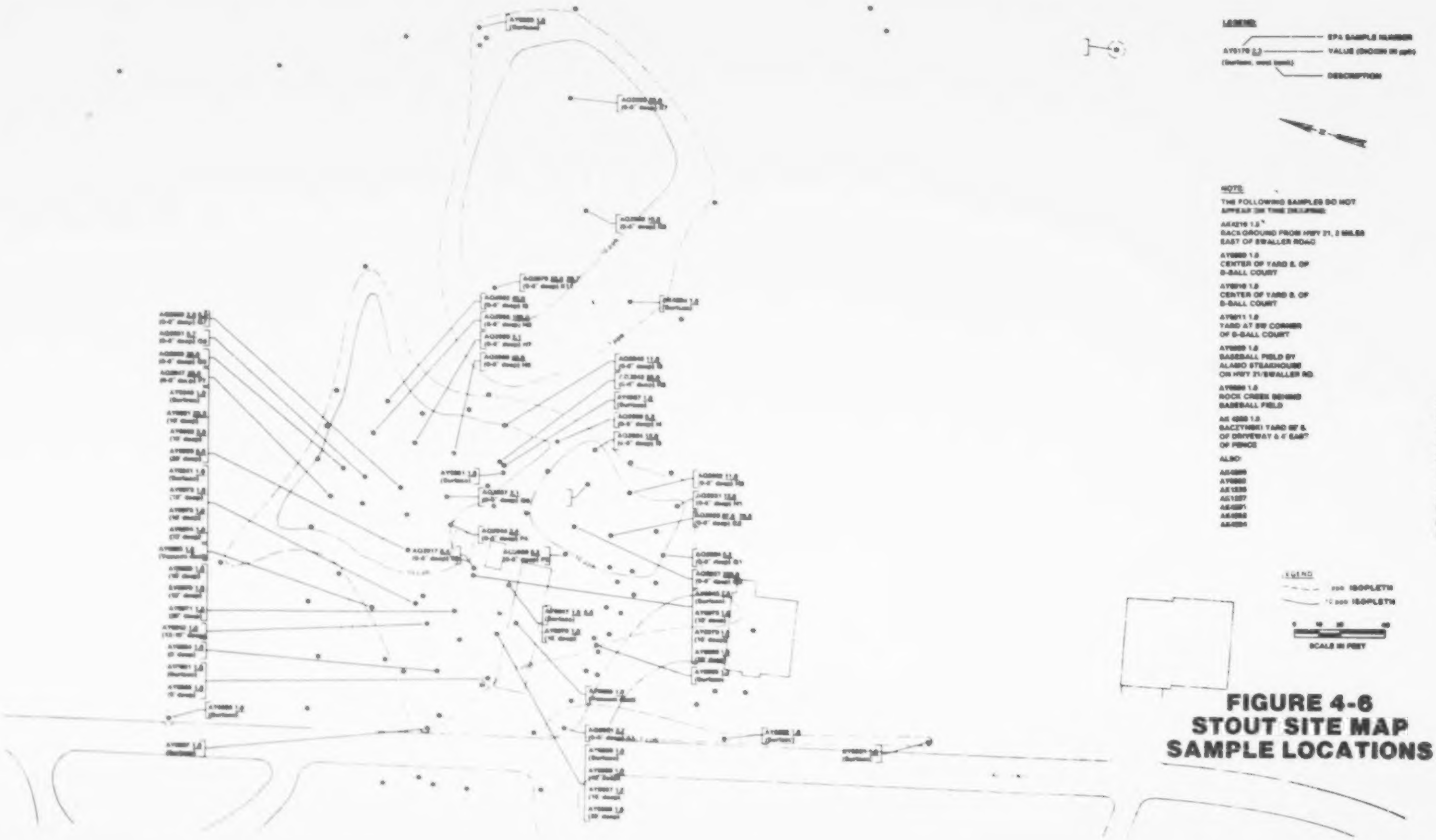

LEGEND:
EPA SAMPLE NUMBER
VALUE (DIOXIN IN ppb)
DESCRIPTION
NOTE:
THE FOLLOWING SAMPLES DO NOT APPEAR ON THIS DRAWING:
BACKGROUND FROM HWY 21, 2 MILES EAST OF SWALLER ROAD
CENTER OF YARD S. OF B-BALL COURT
CENTER OF YARD S. OF B-BALL COURT
YARD AT SW CORNER OF B-BALL COURT
BASEBALL FIELD BY ALAMO STEAKHOUSE ON HWY 21/SWALLER RD
ROCK CREEK BEHIND BASEBALL FIELD
OF DRIVEWAY & 4' EAST OF FENCE
ALSO:
LEGEND
ISOPLETH
ISOPLETH
SCALE IN FEET

FIGURE 4-6
STOUT SITE MAP
SAMPLE LOCATIONS

ROMAINE CREEK

Location

Begins: NE 1/4, Sec. 29, T. 43 N., R. 5 E., 5th P.M.
Maxville Quadrangle
Jefferson County

The creek flows approximately 5 miles northeasterly to enter the Meramec River at the Jefferson County - St. Louis County boundary.

Accessibility

Romaine Creek Road and West Romaine Creek Road generally parallel the creek, with several crossings. The head of Romaine Creek can be entered from the Minker site. Romaine Creek Road is low type bituminous.

History Summary

The Bubbling Horse Ranch horse arena, sprayed with contaminated waste oil in 1971, was the origin of the contaminated soil in Romaine Creek. When the arena was excavated and the material moved to the Minker Site, the soil began to erode and move into Romaine Creek. Contamination of Romaine Creek has come primarily from the Minker Site, and to a lesser extent, possibly from the Ruth Sullins property and the Bubbling Springs Ranch horse arena. The Romaine Creek site along with the Minker and Stout sites is included on the National Priorities List. A State Superfund contract has been signed with the State for these three sites. The contract covers relocation and a feasibility study for the sites.

Site Description (see map)

The majority of contaminated stream sediment has been found within approximately one mile downstream of the Minker site, in the intermittent portion of the creek. 2,3,7,8-TCDD levels range up to 272 ppb, and generally decrease as the distance from the Minker site increases. Since the contaminated soil was transported into the creek by erosion, it generally does not extend outside of the creek banks. Contamination has been found down to two feet below the surface. Beyond one mile downstream of the Minker site, the 2,3,7,8-TCDD levels are near or less than 1 ppb. Contamination levels of 0.3 ppb and 0.79 ppb found downstream of the Bubbling Springs Ranch could have originated from either the horse arena or the Minker site. Contaminated soil is continuing to be moved during periods of rain. Due to the geology of the area, there is a potential for movement of soil particles in Romaine Creek into the subsurface and groundwater.

Geologic and Soils Description

A highly permeable stoney clay residual soil exists in the upper Romaine Creek watershed, allowing movement of surface water into the subsurface probably including sediment transport 50 to 100 feet or more. The upper part of Romaine Creek, approximately one mile, is considered a losing stream. From the point of water flow, near the northwest corner of Sec. 21, T. 43 N., R. 5 E., a continuous and probably perennial flow would be expected all the way to the Meramec River.

The direction of flow of subsurface water lost into the upper watershed of Romaine Creek including the area of the Minker Residence is anticipated to be northeast. This water and possibly sediment would be expected to emerge as stream flow in the main stem of Romaine Creek or one of its tributaries. It is thought that Bubbling Springs would not receive this localized flow due to bedrock structure and lithologies. It is believed that groundwater recharge to Bubbling Springs is regional. A study currently being conducted by the U.S.G.S. and Missouri Division of Geology and Land Survey will further define the complex hyrology of the Romaine Creek Basin.

It appears that contaminants eroded from the horse arena at Bubbling Springs would not recharge groundwater, but would move into Romaine Creek and stay in the stream channel to its conjunction with the Meramec River.

Unique Site Characteristics

The contaminants in Romaine Creek are currently very mobile. They also pose a threat to water and aquatic life.

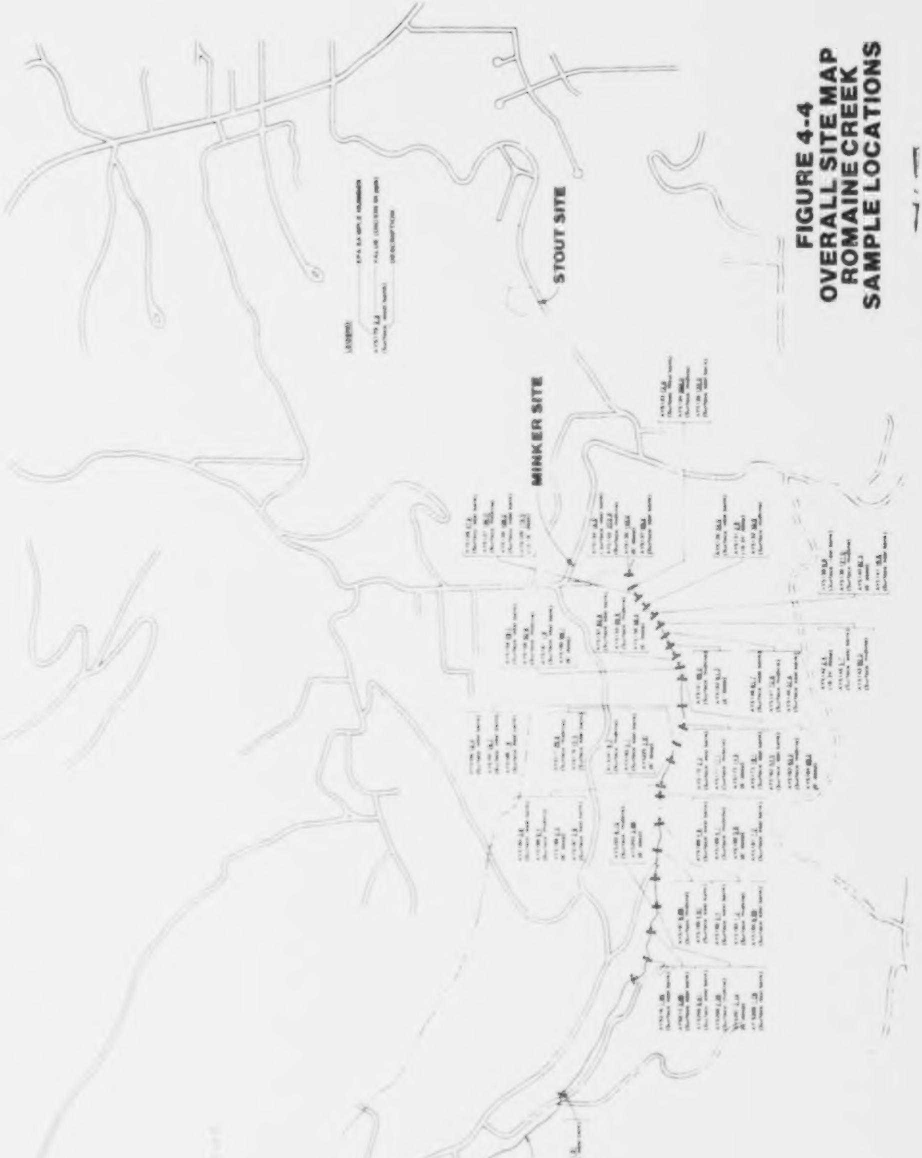
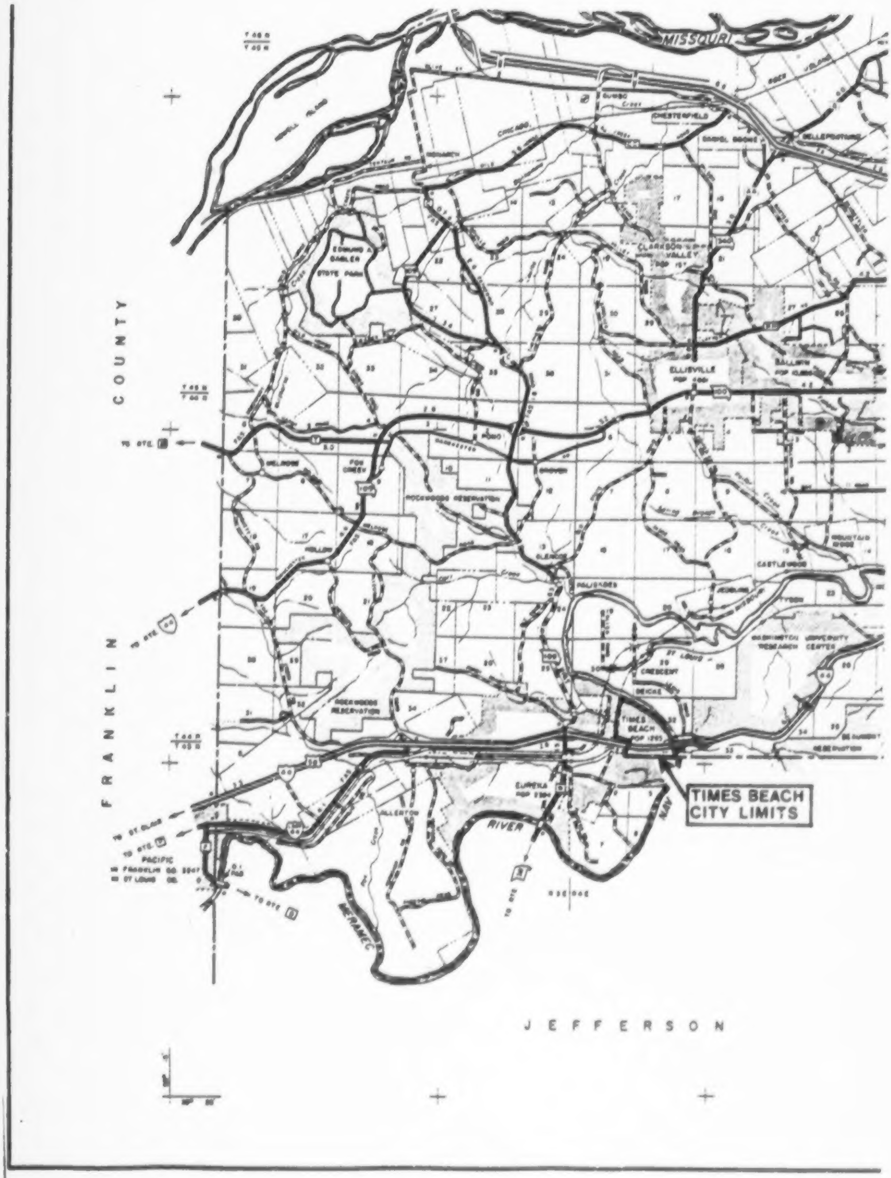

STOUT SITE
MINKER SITE
ROMAINE CREEK

FIGURE 4-4
OVERALL SITE MAP
ROMAINE CREEK
SAMPLE LOCATIONS


MISSOURI
CHESTERFIELD
CLARKSON VALLEY
ELLISVILLE
EDWARD A. BABLER STATE PARK
ROCKWOODS RESERVATION
GLENCOE
CASTLEWOOD
CRESCENT
TIMES BEACH
EUREKA
ALLENTON
PACIFIC
RIVER
MERAMEC
FRANKLIN COUNTY
JEFFERSON
TIMES BEACH CITY LIMITS

TIMES BEACH

Location

Legal Description:
Floodplain of the Meramec
River, principally W 1/2, Sec. 32,
and E 1/2, E 1/2, Sec. 31, T.44 N.,
N., R. 4 E., 5th P.M.
Manchester Quadrangle
St. Louis County
Latitude: 38° 30' 33"
Longitude: 90° 36' 08"
Population 2,061

Accessibility

The City of Times Beach can be entered by any of three routes. Access is gained by exiting Interstate 44 onto the northern outer road from either east or west of the city. The third access point is from the south from Route W onto local roads.

History Summary

In June 1972, a city ordinance was passed to contract with a waste oil hauler to spray the roads for dust control. Apparently all of the gravel streets were oiled that summer twice and a third time where needed, as recalled by residents. In 1973, the roads were again sprayed by contract. The agreement was to have approximately ten miles of road oiled. Five additional streets had been paved so less oiling was done that year. EPA sampled the roads and right-of-ways in November and December 1982, and found 2,3,7,8-TCDD levels up to 127 ppb. In December 1982, the Meramec River flooded the town. EPA sampling in January 1983 following the flood showed that the contaminated soil remained quite immobile throughout the flooding. On February 22, 1983, the EPA Administrator announced a $33 million pledge from superfund to purchase the Times Beach property under a relocation plan to be developed and implemented by the Federal Emergency Management Agency (FEMA). EPA is planning to have a feasibility study conducted to determine the scope and costs of cleanup alternatives. The city is on the National Priorities List.

Site Description (see maps)

Times Beach is principally bounded by the Meramec River, Interstate 44, and the Burlington Northern Railroad tracks. Being in the 100-year floodplain, the area is relatively flat. The majority of the city's 28 miles of paved and gravel road, shoulders, and ditches are contaminated. Maximum levels of 2,3,7,8-TCDD are shown on the city map. Contamination has been found down to at least two feet below the surface. The City of Eureka, population 3,862 lies immediately to the south and west of Times Beach. None of Eureka's streets were oiled and no contamination has been found within the city. Results of all groundwater sampling in the area have been negative.

Geologic and Soils Description

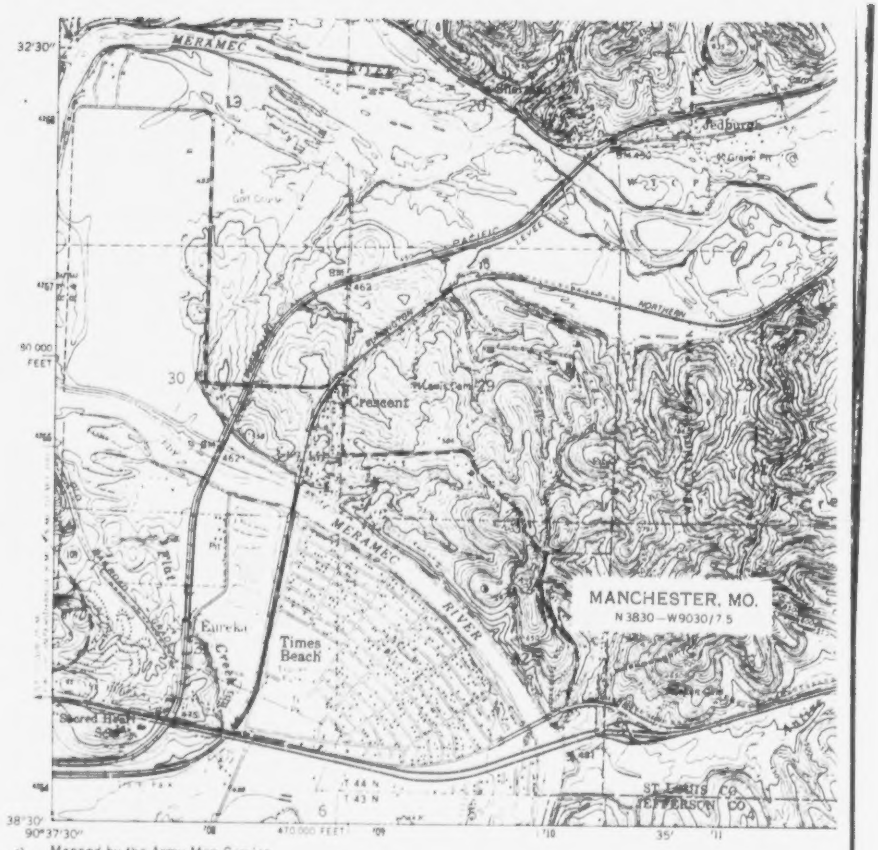
Times Beach is on an alluvial setting, underlain by alluvial silt to a depth of more than 5 feet. Below the alluvial silts; sand, gravel, and a mixture of silt, sand, and clay would be expected to a depth of from 40 to 50 feet where bedrock is encountered. The water table would be expected to be about at the Meramec River level, between 10 and 20 feet from the surface in most of the area.

The alluvial silt has a relatively low permeability and would be expected to be wet natured in that it does not readily or rapidly drain water. Due to this and the screening effect of the silt, it is not likely that soil particles contaminated with dioxin would move down into the water table.

It can be assumed that the contaminated material consists of road bed material plus native soil where the contamination has eroded into the ditches.

Unique Site Characteristics

The Times Beach site comprises the largest volume of contaminated material of all of the sites. Its location in the floodplain will require extra precautions for any remedial activity.


MERAMEC
Jedburg
Gravel Pit
PACIFIC
LEVEE
NORTHERN
BURLINGTON
Crescent
Times Beach
Eureka
Flat
Creek
MERAMEC
RIVER
Sacred Heart
MANCHESTER, MO.
N3830—W9030/7.5
ST LOUIS CO
JEFFERSON CO
T 44 N
T 43 N
32'30"
90 000 FEET
38°30'
90°37'30"
470 000 FEET
35'
(PACIFIC)
7960 I NW

Mapped by the Army Map Service
Published for civil use by the Geological Survey

Control by USGS, USC&GS, and USCE

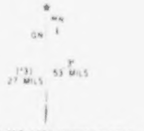
Topography from aerial photographs by photogrammetric methods
Aerial photographs taken 1952 Field check 1954

Polyconic projection. 1927 North American datum
10,000-foot grid based on Missouri coordinate system, east zone
1000-meter Universal Transverse Mercator grid ticks, zone 15,
shown in blue

Dashed land lines indicate approximate locations

Unchecked elevations are shown in brown

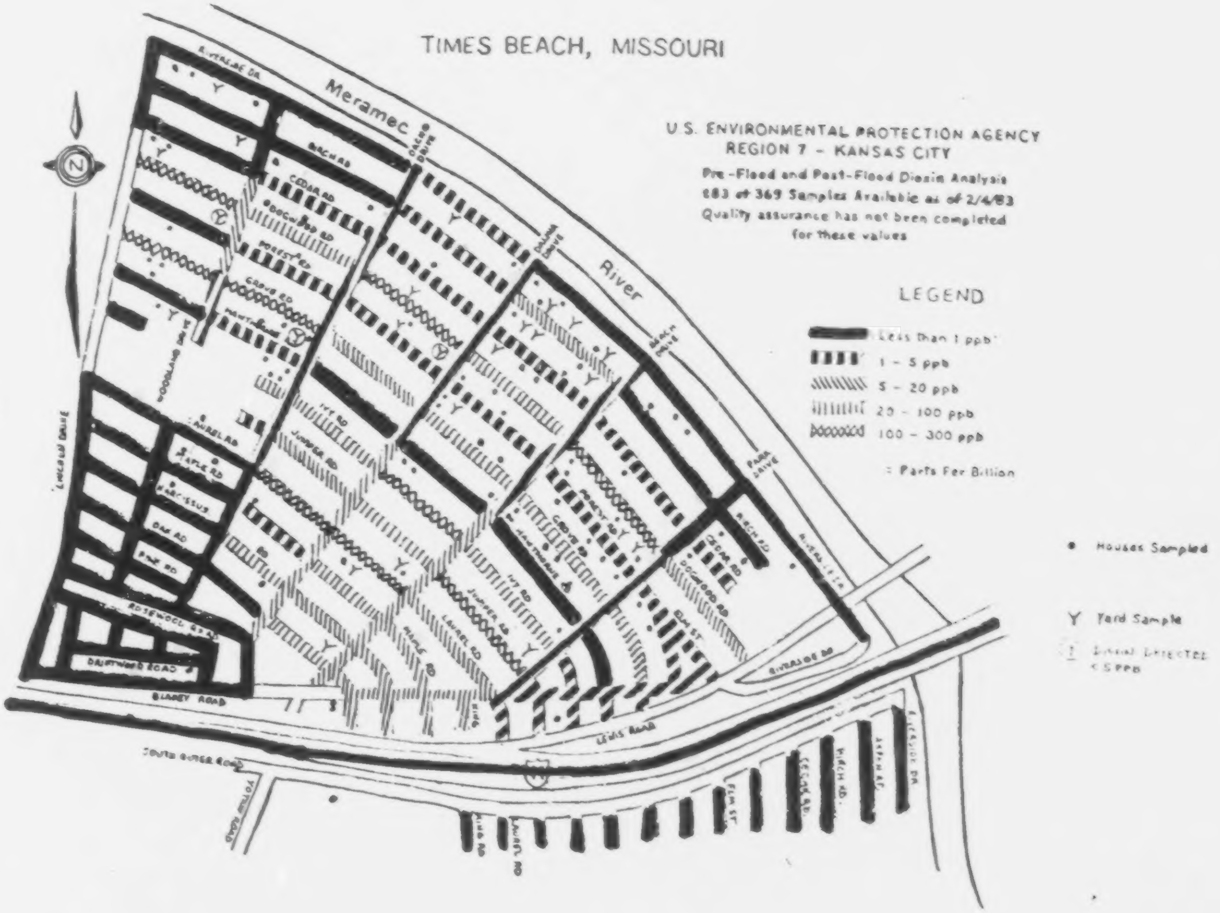
To place on the predicted North American Datum 1983
move the projection lines 2 meters south and
10 meters east as shown by dashed corner ticks


MN
GN
1°31'
27 MILS
3°
53 MILS

UTM GRID AND 1982 MAGNETIC NORTH
DECLINATION AT CENTER OF SHEET

There may be private inholdings within
the boundaries of the National or
State reservations shown on this map

THIS M
FOR SALE BY U. S. GEOLOC
AND
MISSOURI DEPA
A FOLDER DESCRIBI



TIMES BEACH, MISSOURI

U.S. ENVIRONMENTAL PROTECTION AGENCY
REGION 7 - KANSAS CITY
Pre-Flood and Post-Flood Dioxin Analysis
183 of 369 Samples Available as of 2/4/83
Quality assurance has not been completed
for these values

LEGEND
Less than 1 ppb
1 - 5 ppb
5 - 20 ppb
20 - 100 ppb
100 - 300 ppb
: Parts Per Billion
Houses Sampled
Y Yard Sample
Dioxin Detected < 5 ppb
Meramec River
Riverside Dr
Birch Rd
Cedar Rd
Dogwood Rd
Forest Rd
Grove Rd
Hawthorne
Woodland Drive
Lincoln Drive
Laurel Rd
Maple Rd
Narcissus
Oak Rd
Pine Rd
Rosewood Road
Driftwood Road
Blarney Road
Ivy Rd
Juniper Rd
Elm St
King
Oasis Drive
Dallas Drive
Beach Drive
Park Drive
Riverside Dr
Lewis Road
South Outer Road
Yotaw Road
Aspen St
Birch Rd.
Cedar Rd.
Elm St
Laurel Rd
King Rd

CHAPTER 2

SUMMARIES OF EXPERTS' PRESENTATIONS

In an effort to become better informed about the current medical and environmental issues surrounding dioxin, the Task Force has heard from a number of experts in the field of dioxin research. Following are summaries of these experts' speeches at the Task Force meetings.

William G. Dunagin, M.D.
Division of Dermatology M173
University of Missouri
Health Science Center

DIOXIN EFFECTS ON HEALTH SUMMARY

Many misleading statements have been made in the press about the health effects of dioxin. Consequently, the fear that many people in the general public have may be out of proportion to the real hazard. For example, the statement that dioxin is 180,000 times as toxic as cyanide has been repeated numerous times in the media. This is certainly not true for any species of mammal. The most sensitive mammal is the guinea pig where the single dose LD_{50} for dioxin is 250 times pure cyanide or 1000 times cyanide salts. These comparisons also assume that one is dealing with pure dioxin. Actually the dioxin in Missouri is in trace quantities expressed as parts per billion.

The single dose LD_{50} for monkeys is 50 micrograms per kilogram. Humans are probably in the same range (Dr. Neal has estimated the level as more than 100. Analogy with Yusho disease indicated the same range). Using this figure of 50 micrograms per kilo for humans, we can see that dioxin is about 10 times as toxic as cyanide.

Dioxin	50 ug/kg
HCN	500 ug/kg
Cyanide salts	2000 ug/kg
Cantharidin	300 ug/kg
Nicotine	600 ug/kg
Colchicine	300 ug/kg
Digitoxin	35 ug/kg

The last four chemicals are on the shelves of every pharmacy in Missouri. The toxicity of dioxin is not totally out of the range of many substances we are used to dealing with. Dermatologist commonly use a preparation that contains seven parts per thousand of cantharidin in order to kill warts.

Another point that is poorly understood by the general public is that dioxin must be absorbed into the body before it can produce any harmful effect. It does not release radation or vapors. Theoretically dioxin can be absorbed through the skin, intestinal tract, or lungs. Dermal absorption may have been important in 1971 when the waste oil containing dioxin was freshly sprayed on horse arenas. The situation today is different. The dioxin is attached to soil particles and is in much lower concentrations. The work of Poiger and Schlatter at the Swiss Federal Institute of Toxicology using soil from Seveso, Italy, suggests that only one percent or less of an applied dose of dioxin in a soil-water mixture is likely to be absorbed through the skin at concentrations below one part per million. This finding is not at all unusual. The percutaneous absorption of most aromatic hydrocarbons varies greatly depending on the vehicle. The amount absorbed topically is insignificant, and others have shown that inhalation of dust containing one part per billion (the level reported by EPA via newspaper accounts as the highest levels in dust from homes in Times Beach is insignificant). So for practical purposes, ingestion of dioxin is the only way that people in Missouri could get toxic effects, today.

The FDA has set levels of 50 parts per trillion of dioxin for ingesting fish on a one time basis or 25 if fish are eaten on several occasions. A 3 1/2 ounce serving of fish would weigh 100 grams. The equivalent amount of soil containing 1 part per billion of dioxin would be 5 grams. It is unlikely that a competent person would ingest more than this without being aware.

The main objective effects of dioxin are chloracne, porphyria cutanea tarda, peripheral nerve damage, and nerve conduction abnormalities (no objective evidence of organic brain damage). Other effects which have occured rarely in humans, probably only at high doses, and are known mainly from animal experiments, are hepatic necrosis, T cell depression and bleeding tendency. Other conditions such as increased triglycerides, have been reported but it is unclear if they are signigicantly increased compared to a suitable control population.

The importance of chloracne is that it seems to be the most sensitive indicator of dioxin exposure. In some examples of low level exposure to dioxin such as Seveso, Italy, and Bolsover, England, it was the only objective finding. In examples of industrial accidents where there was high level exposure, 80-90% of workers who had some of the more serious effects of dioxin also had chloracne. Chloracne was present in 90% of patients with Yusho, a disease caused by chemicals (dibenzofurans) which are very closely related to dioxin.

So for any one individual, chloracne does not have to be present. However, for a large group of people, it is very unlikely that every person would be an exception to the rule. The preliminary reports of the CDC examination of 186 residents of Times Beach were that no cases of chloracne were found. No one from any other site in Missouri today has chloracne to my knowledge. This is very good news, and indicates that the other direct toxicities of dioxin are unlikely.

Another misconception that many people have is that dioxin may cause serious toxicity a long time in the future. Actually, the principle of a long latency period applies only to cancer. Someone whose liver is in good condition today, and who receives no further dioxin, is not going to suddenly come down with massive liver necrosis or chloracne several years from now.

The question of cancer is very complicated. Basically, the doses of dioxin which have caused cancer in laboratory animals are higher than the doses that produce toxicity. This is not the usual concept that the public has of a silent killer. Dioxin is not a carcinogen in the strict sense of the term. It does not alter the structure of DNA. However, it may be a promoter or cocarcinogen. The concept that any amount at all of carcinogen is bad because lower and lower doses may still cause cancer in a lower and lower percentage of people probably does not apply in the same sense for a promoter.

Amoung humans who have had significant exposure to dioxin after industrial accidents (Nitro, West Virginia, Ludwigshafen, Germany, and Czeckoslovakia) there does not appear to be an increased incidence of cancer, overall. Some people have pointed to the date from Nitro as showing increased lymphoma-leukemia. This is not statistically significant. The same data show that cancer of the stomach and colon were less than expected, in fact the statistics would be better for saying that dioxin prevented cancer of the stomach and colon. Actually neither statement is true, but it illustrates how some people can take bits and pieces of data out of context from epidemiologic studies.

The only types of cancer which may be increased above normal levels are a diverse group of rare tumors called soft tissue sarcomas. Even this is not certain, and more work needs to be done.

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DIOXINS AND DIBENZOFURANS IN THE AQUATIC ENVIRONMENT: RESIDUES IN FISH AND ENVIRONMENTAL FATE

Polychlorinated - dibenzo-p-dioxins (PCDDs) and -dibenzofurans (PCDFs) have been measured in fish and other aquatic samples at concentrations from 1 pg/g to in excess of 1 ug/g. In order to measure these compounds at levels of 1 pg/g by GC/MS techniques, they must be separated from interfering substances (PCBs, polychlorinated-hydroxybiphenyls, -phenoxyphenols, and -diphenyl ethers) and concentrated. We employ column extraction and two sequentially linked chromatographic adsorbents to enrich PCDFs and PCDDs from other co-extractives. Fish from a variety of lakes and rivers were analyzed for PCDDs and PCDFs. PCDF residues were more complex thatn PCDD residues, and were dominated by isomers having 2,3,7,8 - chlorine substitution.

PCDF residues were present in samples from all of the Great Lakes and exceeded 100 pg/g in fish from Lakes Ontario, Huron, Michigan, and Saginaw Bay. We measured the ratio of PCB to PCDF concentrations in fish from the Great Lakes to be approximately 100,000 to 1. A composite sample of fish from the Tittabawassee River (Michigan) contained 290 pg/g of PCDFs and 223 pg/g of PCDDs. The PCDF concentrations in fish from Lake Siskiwit (on Isle Royale, Lake Superior) were determined to be 15 pg/g Fish from Lakes Huron and

Ontario, the Tittabawasse River and Saginaw Bay contained the highest concentrations of 2,3,7,8-TCDD. In the Housatonic and Hudson, the composition of PCDD and PCDF residues in sediments differed radically from residues in fish. The residue profiles observed were similar in that the higher congener groups (hepta and octachloro) predominated in contrast to residue profiles in fish where tetra- and pentachloro congeners were dominant.

Contamination from PCDFs is related closely to PCBs. PCBs contain part-per-million concetrations of PCDFs and PCBs can be converted to PCDFs by heating PCBs to temperatures of 300-500 °C in the presence of air or by low temperature burning of materials containing PCBs. TCDD is most frequently associated with chlorophenols and can be produced in high yeilds when 2,4,5-trichlorophenol is heated or burned. Distillation residues or manufacturing wastes from the production of materials made from 2,4,5-trichlorophenol have been involved with most of the TCDD related pollution problems.

Assessment of the impacts and fate of these chemicals in aquatic ecosystems is difficult because only a limited amount of chronic toxcity data is available for aquatic organisms. Exposure of fish fry to low part-per-trillion concentrations has been reported to cause decreased growth and survival. Delayed mortality, 10-15 days after exposure, has been reported.

Data reported from outdoor pond studies provide some insight into the bioavailability and distribution of TCDD in aquatic ecosystems. In one recent study investigating the fate of TCDD in small outdoor pools, a major portion of added TCDD was concentrated in aquatic plants, fish and the hydrosoil-water column interface. Bioconcentration of TCDD by fish in model ecosystem studies was reported to be approximately 6,000 times the water concentration.

No data for TCDF toxicity to aquatic organisms or PCDF bioconcentration factors from water exposure have been reported. However, based on the ratios of PCDFs to PCBs detected in fish, bioconcentration of PCDFs by fish appears to be comparable to PCBs. Fish from contaminated areas could be important as dietary sources of PCDFs and TCDD in humans. The Food and Drug Administration has recommended that fish having residues of TCDD in the range 25-50 parts per trillion not be consumed more frequently than once weekly and fish having more than 50 parts-per-trillion TCDD not be consumed.

The data obtained from analysis of fish and sediments in river systems studies thus far, suggests that TCDD and TCDF are concentrated in fish and other organisms, while the more highly chlorinated Cl_7-, andCl_8-isomers are concentrated in sediment. This altered distribution suggests that TCDD can not be considered to be unavailable to organisms when admixed with soil that is translocated into streams.

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PART I

ENVIRONMENTAL FATE OF TCDD -- CONCLUSION FROM THREE LONG-TERM FIELD STUDIES

The toxin 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is known to occur as both a contaminant of products made from trichlorophenols and as a by-product from low temperature incineration of wastes containing chlorinated precursors. The magnitude of environmental contamination by the 2,3,7,8-TCDD isomer is currently the subject of intense debate. Although a number of TCDD sources has been identified, environmental monitoring programs for TCDD have generally been unsuccessful in documenting contamination. However, with continued development of sophisticated instrumentation for detecting TCDD in picogram quantities (parts per trillion, ppt) and with renewed interest in monitoring improper disposal of hazardous wastes, additional data on the distribution of TCDD in the environment are being obtained. This paper reviews the environmental data generated from three long-term field studies conducted by the United States Air Force in five distinctly different geographic locations in the United States.

Persistence data on TCDD were obtained from field plots treated in 1972 with sub-surface applications of massive quantities of the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) in soils of Northwestern Florida, Western Kansas and Northwestern Utah. The fate of TCDD in biological systems was extensively studied in organisms that inhabited a unique 3.0 km^2 military test area in Northwestern Florida that was aerially-sprayed during the perios 1962 to 1970 with 73,000 kg 2,4,5-T containing 2.8 kg TCDD. The movement of TCDD in an aquatic exosystem has been extensively studied in an area adjacent to storage sites (Gulfport, Mississippi, and Johnston Island, Pacific Ocean) where more than 8.4 million L of surplus phenoxy herbicide had been maintained for more than six years. Thus, these studies have provided data on the persistence, movement, bioaccumulation and bioavailability of TCDD in the environment.

Soil Incorporation/Biodegradation Studies: One potential method proposed for the disposal of TCDD-contaminated phenoxy herbicides was subsurface injection or soil incorporation of the herbicide at massive concentrations. The premise for such studies was that high concentrations of the herbicides and TCDD would be degraded to innocuous products by the combined action of soil microorganisms and soil hydrolysis. In order to field test this concept, biodegradation plots were established in three climatically different areas of the United States; Northwest Florida (Elgin Air Force Base), Western Kansas (Garden City), and Northwestern Utah (Air Force Logistics Command Test Range Complex). The Utah site had alkaline soil and a mean annual rainfall of 15 cm, while the Kansas and Florida sites had acidic soils and 40 and 150 cm of annual rainfall, respectively.

Although the method of application was designed to simulate subsurface application of the herbicides and TCDD, the actual concentration of the chemicals varied significantly between locations. The Utah site received concentrations of herbicide exceeding 30,000 ppm (and 140 ppb TCDD) while the

Kansas site had soil concentrations of 2,000 ppm herbicide and 280 ppt TCDD. The TCDD was extracted from soil by using a 1:1 cold solution of hexane and acetone, followed by an acid wash. Confirmation of the compound was done by gas chromatography-mass spectrometry. The results from monitoring the fate of TCDD in these plots indicated that the TCDD rapidly "disappeared" so that less than 15 percent was recovered two years later. Because the plots showed similar patterns in TCDD disappearance, it was suggested that the analytical method was measuring only "free" TCDD and not "total" TCDD. Indeed, examination of archived soil samples using exhaustive extraction techniques revealed that more than 80 percent of the applied TCDD could be recovered. The conclusion of the study was that TCDD underwent a "binding" with soil over time and that a simple extraction technique was insufficient to recover the total TCDD. These data are illustrated in Figure 1.

Fate of TCDD in an Ecosystem: Field studies of TCDD were conducted during 1973-1979 on a unique 3.0 km^2 military test area (Test Area C-52A, Eglin Air Force Base, Florida) that was aerially-sprayed with 73,000 kg of 2,4,5-T during the period 1962-1970. Analysis of archived samples of the formulations indicated that approximately 2.8 kg of TCDD were applied as a contaminant of the herbicide. However, 2.6 kg were applied to a 37 ha test grid (Grid I) from June 1962 through July 1964. Levels of <10 to 1,500 ppt could be found in the top 15 cm of soil 14 years after the last application of herbicide on this site. Nevertheless, analysis of 61 soil samples suggested that less than one percent of the TCDD remained on the test area.

Over the years of observation, approximately 341 species of organisms have been observed and identified as associated with that test area. To date, over 300 biological samples (plant and animals) have been analyzed for TCDD. TCDD residues have been found in a wide spectrum of animals collected from the test area. Approximately one-third (21) of the different species examined for TCDD residue have been positive. In general, the levels of TCDD in the organisms appeared to be close to the mean levels of TCDD found in soils.

Field toxicological investigations were conducted during 1973-1978 on populations of the beachmouse, Peromyscus polionotus. Liver tissue from 36 individual beachmice inhabiting the test site contained 300 to 2,900 ppt TCDD. Although bioconcentration factors (mean liver concentrations divided by mean soil concentrations) ranged from 6 for females to 18 for males a bioavailability mechanism for TCDD in soil may have been operative (see figure 2). Whole body analysis for fetuses from test area females indicated apparent placental transport of TCDD. Histopathological examinations were performed on 255 adult or fetal beachmice from the test area and control area. Mircoscopic examinations were performed on 18 different tissues from each animal. The tissues were first examined on a blind study basis and then re-examined on a control versus test basis which demonstrated that the test and control mice could not be distinguished histopathologically. The mean number of fetuses per observed pregnancy was 3.1 and 3.4 for the test area and control area, respectively. A two-factor (treatment and year) disproportional analysis of covariance of organ weights revealed that liver weights for pregnant beachmice from the test area were significantly heavier (P<.01) than liver weights of pregnant females from the control area, and these differences were consistent over the 5 years of observation. These studies suggested that long-term, low-level exposure to TCDD under field conditions has had minimal effect upon the health and reproduction of the beachmice.

TCDD in Herbicide Storage Sites: During the summer of 1977 the United States Air Force disposed of 8.4 million L of TCDD-contaminated phenoxy herbicide by high temperature incineration at sea. This operation, Project

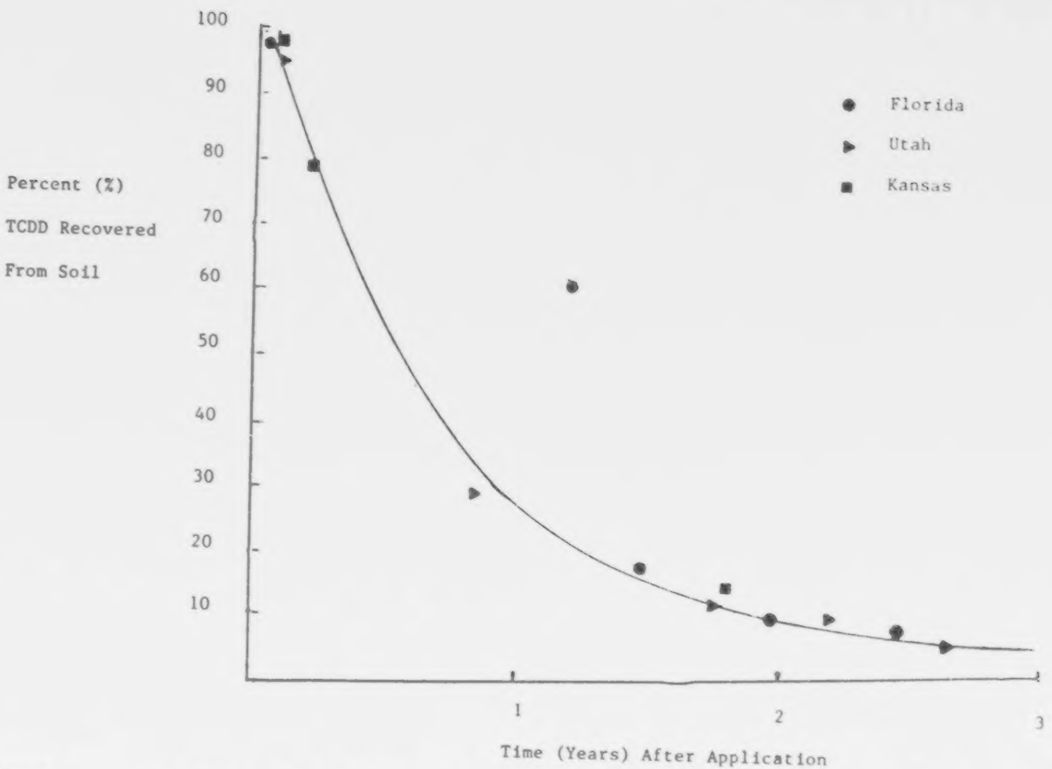

Figure 1. Recovery of 2,3,7,8-TCDD (percent) from three geographically distinct field sites at selected sampling times (years) following soil incorporation of contaminated phenoxy herbicides . [Subsequent re-analysis of soil samples following exhaustive extraction resulted in recovery of more than 80 percent of the original (incorporated) TCDD.]

BIOAVAILABILITY OF TCDD

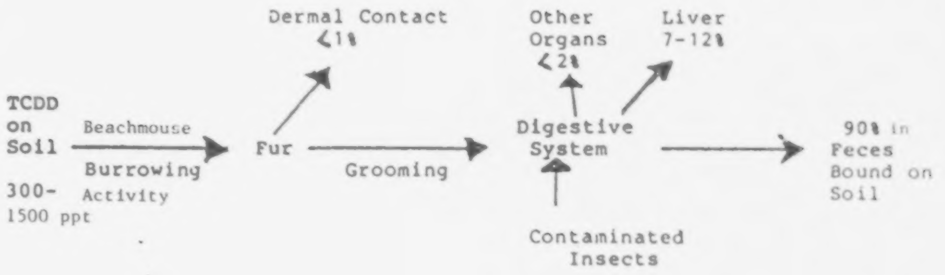
TCDD on Soil
300-1500 ppt
Beachmouse
Burrowing Activity
Fur
Dermal Contact <1%
Grooming
Digestive System
Other Organs <2%
Liver 7-12%
Contaminated Insects
90% in Feces Bound on Soil

Figure 2. Scheme showing routes of TCDD contamination of the Beachmouse, *Peromyscus polionotus*, and the impact of bioavailability of TCDD in soil on exposure. Data are from environmental fate studies of TCDD on Test Area C-52A, Eglin AFB, Florida, 1973-1979.

PACER HO, was accomplished under the very stringent criteria set forth in an United States Environmental Protection Agency (EPA) ocean dumping permit. Among the numerous conditions of this EPA-approved disposal operation was the requirement for the USAF to conduct an extensive storage site reclamation and environmental monitoring program. Details of the proposed site monitoring program were prepared and approved prior to the disposal of the herbicide. The plan recommended that soil samples from the storage areas at both the Naval Construction Battation Center (NCBC), Gulfport, Mississippi and Johnston Island, Pacific Ocean, be collected and analyzed for herbicide and TCDD after the completion of transfer operations. These analyses were to aid in the establishment of a schedule for future monitoring. The site monitoring program would be flexible to requirements generated by construction of any facility on the storage areas and would be concluded upon mutual agreement of all agencies involved. This, the objectives of a site monitoring program were:

1. To determine the magnitude of herbicide and TCDD contamination on the storage areas.

2. To determine the soil persistence of the two phenoxy herbicides (2,4-D and 2,4,5-T) contained in the formulation and the TCDD contaminant.

3. To monitor for any movement of TCDD residue from the sites into adjacent water, sediments and biological organisms.

4. To recommend techniques for managing the storage areas with the ultimate goal of returning the areas to full beneficial unrestricted use.

In July 1977, a preliminary sampling study was initiated. This consisted of assessing the heterogenity of the soils on the sites and the heterogenity of the herbicide concentrations. Twelve sites were selected for sampling at each location; six were in areas of obvious spills and six in areas that showed no spill. Not only were the spills discernible by sight but also by smell. The results from this preliminary study indicated that significant concentrations of herbicides, phenols and TCDD were detected in soils from spill sites. Variations in concentrations and in the ratio of acids to esters suggested that the spills were from different time periods. Accordingly, a more extensive protocol was proposed for future sampling.

The protocol recommended that the sites selected within the two storage areas for monitoring of residue be determined by whether a spill had occured or not occurred at that specific location. The basis for determining a spill was whether a herbicide stain was discernible (heavy, light, absent) and whether a herbicide odor was detectible (strong, mild, absent). Thus, within the storage area numerous locations were found that had a heavy stain and strong odor (presumably representing a recent spill); a light stain and milk odor (presumably representing an older spill); and no stain and no odor (presumably representing an uncontaminated area). Fourteen replication of each treatment were then randomly selected to represent the storage area (thus a total of 42 permanently marked sampling locations at both NCBC and Johnston Island). Twelve of these locations had been tentatively located and marked in July 1977 with the remaining 30 locations and marked in January 1978 with sampling being conducted on these dates, as well in November 1978. In collecting the soil samples, a 8 cm square was marked 15 cm away from the site marker pin. At each sampling time soil was taken from a different "point of the compass" with reference to the marker pin to insure a fresh and undisturbed profile. At the designated site a 8x8x8 cm cube of soil was removed with a ceramic spatula which was rinsed with acetone between uses to prevent carry-over of residue and mircoorganisms. Wherever possible, sediment samples were collected from the drainage areas in a similar manner. Biological samples were collected periodically in these drainage systems at varying distances from the storage sites. Residue studies of areas on the Naval Construction Battalion Center and Johnston Island resulted in the following conclusions:

1. Approximately 1 ha of the 5 ha areas were significantly contaminated with phenoxy herbicides and the associated dioxin (2,3,7,8-TCDD).
2. Levels of 2,4-D and 2,4,5-T herbicides in selected samples from the top 8 cm of soil profile were greater than 100,000 ppm in 1977, but rapidly decreased to one-third that level within 2 years.
3. Levels of TCDD exceeded 0.5 ppm in the soils of many spill sites and have shown trends to decrease over the 3 years of observations.
4. No accurate estimate of TCDD persistence is possible from these studies.
5. Soil penetration of the herbicides was low while soil penetration of TCDD was very low but measurable.
6. Soil sterilization did not occur as a result of herbicide or TCDD contamination, but rather poliferation of certain microflora occurred under high levels of residue.
7. Aquatic organisms found in the drainage system of the storage site at NCBC were significantly contaminated with TCDD (as high as 8 ppb TCDD). The level of TCDD in the organisms and associated sediment decreased rapidly with distance from the site so that samples collected at a distance of 2 km had low ppt levels of TCDD.
8. The principle recommendation for present management of the 5 ha areas at the Naval Construction Battalion Center and Johnston Island is that the areas be left undisturbed permitting the continuation of "natural" degradation of the herbicides and TCDD. Specific recommendations to prevent further movement of contaminated soil from the area included both limiting access to the storage areas and preventing water and wind erosion by stabilizing any potential sites for erosion.

PART II

DIOXIN IN BODY FAT AND HEALTH STATUS: A FEASIBILITY STUDY

Since 1978 there has been mounting concern over human exposure to 2,3,7,8-TCDD. Much of this concern has been expressed by veterans of the Vietnam war who believe that they were exposed to the TCDD-contaminated herbicide, 2,4,5-T, an ingredient of Agent Orange, the major defoliant used by the United States Armed Forces in Vietnam.

During a 5-year period from 1965 to 1970, the U.S. Air Force applied more than 40 million liters of Agent Orange containing more than 92 kg TCDD in South

Vietnam. Some two million American military personnel served 1-year tours during the same period. Many veterans of that era have reported medical problems that they ascribe to exposure to Agent Orange. Their complaints have ranged from tingling in the extremities to rare forms of cancer. Some veterans have fathered children with birth defects and have suggested that the TCDD is the cause. Accordingly, the Veterans Administration (VA) has initiated extensive health studies of veterans exposed by Agent Orange and its dioxin contaminant during the Vietnam Conflict.

Since TCDD is known to accumulate preferentially in the adipose tissue of certain species of laboratory animals, it was suggested, early in the history of the Agent Orange issue, that the analysis of human fat for TCDD might provide a way to determine earlier Agent Orange exposure. A relation between the presence of this substance health problems had also been suggested. Although methods for TCDD analysis have imporved in recent years, no such study had been carried out in humans with known exposure to herbicides containing this toxic contaminant. Consequently the VA embarked on a small feasibility study to test the methodology and to determine whether conclusions might be drawn regarding the significance of the results. The study was carried out in three groups of adult males as follows:

(1) Twenty Vietnam veterans all but one of whom claimed health problems related to Agent Orange exposure and who volunteered for the fat biopsy.

(2) Three U.S. Air Force officers with known heavy and relatively recent exposure in connection with herbicide disposal operations but who did not serve in Vietnam.

(3) Ten veterans with no service in Vietnam and no known exposure to herbicides who were undergoing elective abdominal surgery and who volunteered to serve as controls.

Methods: The prodedure called for the removal of 10-30 grams of subcutaneous adipose tissue from the abdominal wall. This was accomplished surgically under local anesthesia. Precautions were taken before, during and after the procedure to avoid contamination by products, e.g., hexachlorophene, that could contain TCDD. Specimens were collected in glass containers previously rinsed with acetone and dried before use. All tissues were refrigerated during shipment to the assay laboratory. Each of the volunteers had a medical history, physical examination, and routine clinical chemistry. The details of military service in Vietnam from the volunteer's report and his service record were examined to evaluate his potential exposure to military herbicides using the dates, location and nature of his service. From these a rough estimate of the likelihood of exposure to TCDD was made without knowledge of the assay results.

The extraction and assay of all samples for TCDD were conducted at the University of Nebraska, Midwest Center for Mass Spectrometry, Lincoln, Nebraska. Approximately 5 to 10 grams of tissue were used, when available, for each analysis. A known amount, generally 2 ng, of internal standard (either

^{37} C-2,3,7,8-TCDD or ^{13} C-2,3,7,8-TCDD) was added to the adipose tissue. The sample was digested in alcoholic potassium hydroxide followed by extraction with hexane to remove TCDD. The hexane extract was washed with concentrated sulfuric acid, neutralized, dried and concentrated. The final stage of preparation involved the use of three short-column liquid chromatography steps (silica gel, alumina and Florisel). Gas chromatography/high resolution mass spectrometry was employed for quantifation of 2,3,7,8-TCDD and coeluting isomers. Signal profiles were obtained at a mass resolution of 10,000 for m/z 321.8936, the most abundant molecular ion for TCDD, and for the internal standard mass by signal averaging for approximately 100 sec. commencing with the appearance of the co-eluting internal standard 2,3,7,8-TCDD.

Extracts which contained materials giving signals greater than 2.5 times noise at the exact mass of TCDD (i.e. 321.8936 \ 0.0020) over the integration period discussed above were reanalyzed. For the second analysis, signal profiles of m/z 321.8936 and m/z 319.8965 were monitored over the elution period of 2,3,7,8-TCDD (determined by injection of standard solutions). A positive detection was reported if signals were observed above the detection limit (2.5 times noise) and if their intensity ration was 1.0:0.78 + 0.10, which is consistent with the presence of four chlorine atoms in the molecule. Samples meeting all criteria except the correct isotope intensity ration have been considered to contain "not detectable" levels of TCDD. For these samples, it was judged that the presence of TCDD is not disproved by the observation of an incorrect isotope ration at these low concentrations; rather, the presence of TCDD is not confirmed.

Results: Of the twenty veterans who served in Vietnam, seven had no detectible TCDD with the limit of detection at 2 to 6 part per trillion (ppt). Another two had detectible material that could not be validated as TCDD and the results for one was considered equivocal because the measured value was only questionable above the detection limit. Five of the ten remaining Vietnam veterans had TCDD in amounts from 5 ppt to 7 ppt. Three Vietnam veterans had TCDD in the amounts from 9 ppt to 13 ppt. One individual had 63 and 99 ppt (average = 81 ppt) and another had 23 and 35 ppt (average = 29 ppt).

Of the ten control ("unexposed") veternas, four had TCDD indentified in their fat (6,7,7 and 14 ppt). Two other veterans had values low enough to be considered equivocal and in three instances the detected material was not validated as TCDD. The remaining veteran had no detectible TCDD.

One of the three Air Force officers with known exposure had no identified TCDD in his fat. The unidentified substance in his case and the TCDD measured in the other two officers was never more than 3 ppt above the limit of detection.

The clinical data were reviewed, seeking correlations between complaints or diagnoses and the assay results. Within the group of twenty Vietnam veterans, seven of them reported some health problems beginning in or during a tour of duty in Vietnam. No two, however, reported the same symptoms. The remaining thirteen veterans reported no illness in Vietnam although only one reported good health at all times. Among the nineteen veterans with current medical complaints, symptoms and diagnoses varied widely without any common pattern. Five had mental problems ranging from nervousness to schizophrenia and three had experienced difficulties of reporduction, namely, spontaneous abortions by the wives of two and congenital heart disease in the sone of a third.

Four of the seven veterans who reported difficulties while in Vietnam had no TCDD detected. The five veterans with mental problems included three without detectible TCDD and three whose assays results were 5 to 7 ppt. One of three veterans reporting reproductive problems had no detected TCDD, one had doubtful level and one had 7 ppt. Detectible TCDD in the body fat could not be correlated with clinical chemistry findings. Similar results applied to the controls i.e., veterans who did not serve in Vietnam.

The highest values for TCDD in adipose were found in two Vietnam veterans. Both men had military records substantiating duties that involved the use of TCDD-contaminated herbicides in Vietnam. Other individuals, however, who claimed extensive handling of herbicide in base perimeter operations had little or no TCDD in their adipose.

Discussion: TCDD was found in some persons who reported exposure to Agent Orange and in others who were never in Vietnam. On the other hand, some veterans from Vietnam had no detectible TCDD and the same is true of veterans who were never in Vietnam. The low level of TCDD in two Air Force officers and its absence in another is of special interest since their exposure to TCDD is certain and more recent than that of the veterans.

Among the twenty Vietnam veterans there was no uniformity of symptoms, either immediately after exposure, at the time of biopsy, or during the intervening period. No one symptom or group of symptoms was common to veterans with detectible TCDD in their fat. The presence of TCDD did not mean ill health nor did its absence indicate good health. No detailed statistical analysis was attempted of this small pilot series.

The TCDD content in the fat was very small, not exceeding 100 ppt. Since TCDD accumulates in fat more than in other tissue, the average body concentration would be much lower. Even if all tissues contained 100 ppt, however, the concentration would be less than 0.1 mircogram per kilogram of body weight, a value below the demonstrated toxic dose to threaten health.

Conclusions: The present assay for TCDD in fat tissue does not offer a satisfactory routine test for exposure to Agent Orange, providing neither clear evidence of contact with that specific defoliant nor of absence of such contact. Moveover, the technique cannot be used to correlate body levels of TCDD and medical conditions. The assay technique is difficult and not readily available, fat samples must be obtained by surgical biopsy and the source of any TCDD detected is uncertain. The current assay method does provide a research tool under proper conditions and for specific purposes, e.g., for determining the rate of disappearance of TCDD after known exposure.

PART III

A REVIEW OF ON-GOING EPIDEMIOLOGIC RESEARCH IN THE UNITED STATES ON THE PHENOXY HERBICIDES AND CHLORINATED DIOXIN CONTAMINANTS

The use of herbicides to control vegetation has been one of the most persistent controversial subjects arising from the Vietnam Conflict. The United States Air Force applied most of these herbicides in dense jungle areas to uncover and expose hidden enemy staging areas, clear vegetation from the vicinity of military bases and along lines of communication, and to destroy enemy crops. The objectives were to provide defoliated zones that would reduce ambushes and disrupt enemy attacks. The most commonly used "defoliant" was "Agent Orange", a mixture of the two commercial herbicides, 2,4-D and 2,4,5-T, widely employed for many years for brush control in forestry programs throughout the United States and for weed control in agriculture.

During a 5-year period from 1965 to 1970, the U.S. Air Force applied more than 40 million liters of Agent Orange containing approximately 92 kg TCDD (2,3,7,8-TCDD) in South Vietnam. Some two million American military personnel served 1-year tours during the same period. Recently, many veterans of that era have reported medical problems that possibly stem from exposure to Agent Orange during their military assignments. Their complaints have ranged from tingling in the extremities to rare forms of cancer. Some veterans have fathered children with birth defects and have suggested that the TCDD in Agent Orange is the culprit. The basis for resolving the TCDD controversy must in large measure stem from the results of scientific inquiry.

Accordingly, the Veterans Administration (VA) and the Centers for Disease Control have initiated extensive health studies of veterans exposed to Agent Orange and its dioxin contaminant during the Vietnam Conflict. Other federal agencies have initiated studies of populations potentially exposed to chlorophenols and TCDD during manufacturing processes, pesticide applications or as a consequence or improper hazardous waste disposal. Rationale for the more than twenty human studies underway is the need to determine the long-term health consequences of such exposures. Hence, studies are required on mortality, morbidity, reproduction and body burden levels of TCDD. Numerous efforts are underway to clarify the relationship of soft tissue sarcoma and phenoxy herbicides and chlorophenol exposure.

Early Federal Efforts: The United States Environmental Protection Agency, the United States Department of Agriculture and the National Institute for Occupational Safety and Health have all conducted limited studies on the potential associations of phenoxy herbicides and human spontaneous abortions and birth defects. These studies and those conducted in the industrial setting suggest that there is very little epidemiologic data associating TCDD or the phenoxy herbicides with any long-term health effects in humans other than chloracne. However, neither is there strong evidence to validate the absence of such effects. Most studies have not included sufficient numbers of subjects to detect an increased risk of an uncommon condition. Also the period of observation in many studies has been inadequate to detect an illness with long latency period between exposure and illness.

Agent Orange Registry: What has been the response of the Veterans Administration and other governmental agencies to the critical issues raised by Agent Orange? Early action by the VA included the development of a communication process for gathering scientific and medical information which would bear directly on the problem. In addition, early in 1978, the Veterans Administration established the Agent Orange Registry in order to accomplish four objectives:

(1) To identify all Vietnam veterans expressing a concern about the adverse health effects from their exposure to Agent Orange.

(2) To provide a mechanism for Vietnam veterans to voice their concerns to a physician, receive a physical examination and obtain responsible answers to some of their questions.

(3) To serve as a mechanism for follow-up of these individuals if, at a later date, new information develops as a result of the various research efforts underway or being planned.

(4) To obtain, as a by-product, some preliminary information on the current health status of the veternas who have participated in the registry.

The Agent Orange Registry provides us essentially with a listing of names and addresses, together with some background data on a self-reported group of veterans with service in Vietnam. A complete physical examination and a group of baseline laboratory tests are provided to each veteran. The information from this effort is currently being placed into a computer data bank. There are almost 100,000 Vietnam veteran who have been examined by the Veterans Administration. We are in the process of collating the data to determine whether or not there are any significant health trends in these individuals which might suggest areas for further investigation.

TCDD Assay of Human Adipose Tissue: The Veterans Administration, in cooperation with the Environmental Protection Agency (EPA) has initiated an interagency agreement to study levels of 2,3,7,8-TCDD in adipose tissue from a selected group of U.S. males.

The Environmental Protection Agency has been demographically collecting adipose tissue from the U.S. general population (i.e., by region, age, sex and race). This National Adipose Tissue Bank was initiated in 1968 and now contains specimens from over 12,000 individuals. Represented within this bank is available adipose tissue from approximately 500 males born between 1937 and 1952. Many of these individuals would have served in the military during the Vietnam-era and it is reasonable to assume that some actually served in Vietnam during the period of Agent Orange use. A retrospective study of selected chlorinated dioxins and furans may establish data on background levels of 2,3,7,8-TCDD in the U.S. male population as well as whether service in the military and especially in Vietnam has had an effect on the levels of TCDD in adipose tissue.

The study will be conducted in three phases. Phase I will be to obtain the name and social security number for the approximately 500 males noted above. This information will be used to determine military service status. Phase II will be the development of analytic methods for the determination of selected dioxins (especially the 2,3,7,8-TCDD) and furans in human adipose tissue. The method will be subjected to rigorous interlaboratory validation by an independent Analytic Referee, e.g., the Association of Official Analytical Chemists. Phase III will be the analysis of the adipose tissue and the preparation of a final report. Phases I and II should be completed within fiscal year 1983, and the report from Phase III should be available in early 1985.

Vietnam Experience Twin Study: The Vietnam Experience Twin Study was initiated by a group of research staff members at the Veterans Administration Medical Center, St. Louis, Missouri. The study will identify pairs of twin veterans where one twin served in Vietnam during the period of Agent Orange spraying and the other served in an area other than Southeast Asia. Approximately 600 pairs of twins will be examined using a series of psychologic, physiologic and biochemical tests.

The development of a protocol for the study and the establishment of a Twin Registry from which to solicit participants are now underway. The study will focus primarily on the total Vietnam experience rather than any single factor such as Agent Orange. If sufficient numbers of identical twin pairs are identified, there may be an opportunity to draw some conclusions regarding the effects of specific aspects of the Vietnam experience such as combat, herbicide exposure or use of insecticides.

Birth Defects and Military Service in Vietnam Study: In 1981 the Centers for Disease Control (CDC) in Atlanta, Georgia initiated a study designed to determine if Vietnam veterans are at an increased risk of having children with birth defects. Since 1968 CDC has maintained a registry of all babies born with defects in the greater metropolitan Atlanta area. Of the more than 15,000 children in this registry, approximately 7,500 had significant anatomical defects at birth. The investigators will attempt to locate and interview both parents of 5,400 of the children in this group. In addition, the parents of 3,000 control normal babies born during the same time period will be interviewed. Since the major objective of this study will be to determine whether an unusually high proportion of fathers of babies born with defects served in Vietnam, information will be gathered about Vietnam service as well as other factors which may be associated with the occurrence of birth defects. If the study demonstrates that a Vietnam veteran has an increased risk of fathering a child with a defect it may be desirable to attempt to determine if the increase is associated with Agent Orange exposure or with some other factor(s). The study is scheduled to be completed by the end of 1983.

The Air Force Health Study: In 1979 the Air Force initiated the protocol for a comprehensive epidemiologic study of the RANCH HAND personnel, a group of approximately 1,250 men who actually conducted the aerial, fixed-wing herbicide spraying missions in Vietnam from 1962 through 1971. The study is designed around the question, "Have there been, are there now, or will there be in the reasonably foreseeable future, any adverse health effects among RANCH HAND personnel caused by repeated exposure to Herbicide Orange?"

The investigation is composed of three integrated elements -- a mortality study of those individuals who have died since their exposure, a morbidity study to examine the current health status of the study subjects, and a follow-up study to look for delayed effects over the next 20 years. The mortality and morbidity study elements are being conducted simultaneously on the RANCH HAND personnel and a very carefully matched control group by the use of personnel tracking procedures coupled with an extensive review of military medical and personnel records; detailed face-to-face questionnaires to ascertain current and past health events as well as occupational and family data; and comprehensive physical examinations, psychological testing and diagnostic examinations are to be administered periodically during the follow-up phase. Results from the mortality analysis were reported in June 1983 and the results of the initial questionnaire and current health status analysis are to be released in October 1983.

Epidemiologic Study of Ground Troops Exposed to Agent Orange: Public Law 97-151 charged the Veterans Administration to "design a protocol for and conduct an epidemiological study of persons, who while serving in the Armed Forces of the United States during the period of the Vietnam conflict, were exposed to any of the class of chemicals known as "the dioxins" produced during the manufacture of the various phenoxy herbicides (including the herbicide known "Agent Orange") to determine if there may be long-term adverse health effects in such persons from such exposure. Efforts are underway by the Centers for Disease Control (through an interagency agreement with the VA) to complete a protocol and to identify suitable cohorts. This will be a huge research effort which will include interviews and examinations involving several thousands of veterans. A major problem in the design of such a study relates to the fact that there were very few records maintained that would link specific ground troops to herbicide exposure. Another problem encountered is that it is difficult to design data collecting instruments and techniques when the health outcome variables are so ill-defined. However, much design work has been accomplished, and it is hoped that the actual study will get underway early in 1984.

Conclusion: Neither the government nor the scientific community has resolved the numerous controversies (environmental, medical, or political) involving the use of Agent Orange in Vietnam from 1962 to 1970 nor the use of 2,4,5-T within the agricultural setting of the United States, nor the impact of TCDD from improper disposal of toxic wastes. Scientific data on the long-term health effects of exposure to TCDD are at present insufficient to form a concensus. Hence, the scientific community must continue to conduct valid research on this controversial environmental and health-related issue in order to provide a reliable basis for appropriate decision-making. The Veterans Administration stands firmly committed to working closely with other agencies of the Federal Government as well as with the private sector to obtain as many answers as quickly as possible consistent with sound scientific principles in order to resolve this perplexing issue. The resolution of the controversy, however, will be achieved only following the public's acceptance of the outcome of scientific investigations. To that end, scientists must accept the responsibility for not only conducting quality research, but also for translating the results of their efforts to legislators, to the courts, to the media and ultimately to the public at large.

Dr. Robert Neal, President
Chemical Industry Institute of Toxicology

The purpose of my presentation was to review what is known about the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Slide 1) to experimental animals and man. In regulating human exposure to TCDD, we are fortunate to have considerable information about the toxicity of this compound to humans. Information on human toxicity is generally not available on other compounds of environmental concern. However, in spite of the existence of a substantial data base, there are aspects of TCDD toxicity about which there is still considerable question. I will attempt to address some remarks to these data gaps.

In my presentation I will confine my remarks to those toxic effects in experimental animals and man which are supported by objective and scientifically sound data. As is generally the case with environmental contamination with toxic compounds, there have been numerous "scientific" opinions for the existence or absence of toxic effects as a result of exposure of experimental animals or man to TCDD which are not consistent with the existing scientific data. I believe it is essential that we differentiate between those concerns or lack or concern for human health or environmental degradation which are based on scientific fact from those based on speculation. With these distinctions in mind groups such as yours will be in a better position to make informed decisions concerning risks or potential risk resulting from human exposure to these compounds.

There have been a number of instances of human poisoning by TCDD. This slide (Slide 2) lists some of the more important ones. First of all there have been a number of accidents in industrial facilities which resulted in exposure of workers to TCDD. Most of these accidents have occurred during the synthesis of 2,4,5-trichlorophenol by alkaline hydrolysis of 2,4,5,6-tetrachlorobenzene. On July 10, 1976, an accident occurred in Seveso, Italy, which resulted in the exposure of a substantial nonindustrial population to TCDD. The importance of this particular accident is that it resulted in the first substantial exposure of women, particularly pregnant women, and children to TCDD under conditions in which unequivocal illness occurred. One minor incident resulted in the poisoning of two, or perhaps three, chemists involved in the synthesis of TCDD. The other major incident listed was the poisoning which occurred when used oil, contaminated with TCDD, was used for dust suppression on horse show rings in Missouri, an incident with which you are very familiar.

The acute toxicity of TCDD is quite variable depending on the animal species examined (Slide 3). The guinea pig is the most sensitive and the hamster the least sensitive of all the animal species that have so far been examined in detail. The acute toxicity in guinea pigs as compared to hamsters differs by approximately 5000-fold depending on the route of administration. It is frequently stated that TCDD is the most toxic chemical known. This is, of course, not true. Botulinum toxin and tetanus toxin are more toxic than TCDD in all species examined. Also, in some species (e.g. the hamster), there are a number of compounds, including some insecticides in commercial use, which are more acutely toxic than TCDD.

What is the acute toxicity of TCDD in humans? In spite of a number of incidents of human poisoning, there is no evidence that a human fatality has occurred solely as a result of exposure to TCDD. However, in all of these incidents of accidental exposure, we have no definitive data on the dose to which humans were exposed. The only reasonably accurate dose-response data for TCDD toxicity in humans comes from studies carried out in Halmesburg Prison in Pennsylvania in the mid-1960's. In these studies, Dr. Albert Klingman of the University of Pennsylvania exposed approximately 60 prisoners to dermal doses of 0.2 to 16 ug of TCDD. These doses were reported to have caused no toxic effects. Subsequently, a similar number of prisoners (approximately 10) were exposed to larger doses, some as high as 7500 ug applied dermally in smaller doses over a period of a few weeks. These doses were reported to cause chloracne. However, no other clinical symptoms were observed in the subjects of these studies. When one considers all of these data, it is reasonable to conclude that humans appear to be one of the less sensitive species to the acute and subacute toxic effects to TCDD.

A number of studies have been performed examining the potential for TCDD to cause reproductive toxicity in experimental animals. This slide (Slide 4) shows the minimum dose of TCDD causing reproductive toxicity in various species and in various studies. The data shown here for monkeys ignores the data generated in studies at the University of Wisconsin. There is considerable controversy surrounding the conduct of these studies. In my opinion, we must await replication of these studies by another laboratory.

It is important to note that the exposure of male mice (but not females) to high levels of TCDD for extended periods of time does not result in a decrease in reproductive capacity in that sex of that species. These data suggest that TCDD may exert its reproductive toxicity primarily in females. It will be important to examine males of other species to verify that this is the case.

There was no evidence of a statistically significant decrease in reporductive efficiency in the populations of women exposed to TCDD in the Seveso incident. I would again like to point to the particular value to toxicology of the data from Seveso. This unfortunate occurrence allows us to assess, although in a limited way, the potential for TCDD to cause reproductive toxicity in human females.

TCDD causes teratogenic effects in mice (Slide 5). The dose required is 1-3 ug/kg/day administered during the 7-15th day gestation. Exposure of pregnant rats to certain doses of TCDD caused kidney anomalies in the offspring. There has been considerable debate whether this effect is a true teratogenic effect or, alternatively, the result of the acute toxicity of TCDD to the fetus. In my opinion, the data more strongly supports the effect being the result of TCDD toxicity. To my knowledge no birth defects have been detected in monkeys born to or aborted from TCDD-treated mothers. Also, there was not a statistically significant increase in birth defects in the children born to or aborted from the pregnant women in Seveso who were exposed to TCDD. It was thought initially that an increase in birth defects may have occurred. However, a more careful examination of the data showed an obvious underreporting of birth defects in that region of Italy prior to the accident involving TCDD. When compared to a broader control base, there was not a statistically significant increase in birth defects observed in the exposed group in Seveso.

TCDD causes cancer in rats and mice (Slide 6). Tumors have been produced in mice whether the exposure is by way of the diet or applied to the shaven skin on the backs of the animals. The fibrosarcomas produced by dermal application of TCDD to mice is a particularly important observation when viewed in light of the suggestion that soft-tissue tumors may be increased in human populations exposed to TCDD.

A number of studies have been carried out looking for an increase in cancer, and in one study, mortality in humans who have been exposed or potentially exposed to TCDD (Slide 7). A study carried out in Sweden among forestry workers suggested a correlation between exposure to chlorophenols and chlorophenoxy acids and an increase in soft-tissue tumors. Subsequent studies in Finland among forestry workers and in New Zealand among agricultural workers (not shown) similarly exposed to chlorophenols and chlorophenoxy acids failed to show a similar correlation between exposure and soft-tissue tumors. Workers exposed to chlorophenols and chlorphenoxy acids are also potentially exposed to various chlorinated dioxins and in some cases to TCDD.

There are a number of confounding factors in the Swedish study which prevent the examination of the existence or absence of a cause-effect relationship between TCDD exposure and soft-tissue tumors in humans. In the first place, the chlorophenols themselves may have been responsible for the increase incidence of cancer. The compound 2,4,6-trichlorophenol is an animal carcinogen. In addition, the forestry workers were also exposed to other commercial chemicals. These chemicals may have been responsible for the increase in soft-tissue tumors. Finally, an increase in soft-tissue tumors was also observed in forestry workers not known to be exposed to preparations containing TCDD. Thus, the correlation between TCDD exposure and an increase in soft-tissue tumors is questionable at this time. However, because of these human data and because dermal exposure of mice to TCDD causes soft-tissue tumors, further study of this issue is clearly warranted. These studies should be carried out in humans which have developed illness as a result of exposure to TCDD (See Slide 2).

An increase in gastrointestinal cancer has been observed in a population of workers poisoned by TCDD in an industrial accident. However, the fact that populations examined was small (about 50 subjects) and an increase in gastrointestinal tumors have not been detected in other TCDD-poisoned populations, raises doubts whether there is a cause-effect relationship between gastrointestinal cancer and TCDD exposure. Finally, a population of U. S. workers (121 subjects) poisoned by TCDD in 1949 have been examined for an increase in mortality and various diseases (including cancer). No increases in mortality or morbidity, including incidence of cancer or death from cancer, was seen in this population.

TCDD can cause a number of toxic effects in experimental animals (Slide 8). In addition to acute lethality, reproductive toxicity and birth defects which have been discussed earlier, TCDD can cause liver damage in some species (rat, mouse), cause a decrease in immunocompetence (mice) and cause an induction of various enzymes in a number of species. The induction of enzymes is not known to have any clinical significance.

TCDD also causes a number of toxic effects in humans. These are shown in Slide 9. Of all the symptoms listed, the development of chlorachne is the most frequent and sensitive response of humans to TCDD exposure. A careful examination of all the available data on human toxicity resulting from exposure to TCDD and the closely related compounds, the chlorinated dibenzofurans, strongly suggest that if chloracne is not present, the individual or individuals being examined have not been exposed to a sufficient dose to TCDD to cause other symptoms of toxicity. Often chloracne is the only symptom of TCDD toxicity seen in exposed humans. However, in the more severely exposed populations, liver damage and neurological effects are also seen. Other symptoms such as weight loss, hyperlipidemia, anorexia, hirsutism and loss of libido are also seen. Another reported manifestation of TCDD toxicity is porphyria curanea tarda. However, because this condition is infrequently seen in humans poisoned by TCDD, it is questionable whether porphyria cutanea tarda is caused by exposure to TCDD.

In summary, the acute toxicity of TCDD varies widely dependent on the species under examination. The current date indicate that man is one of the lest sensitive species to the acute toxic effects of TCDD.

The most common and sensitive symptom of TCDD toxicity in humans is chloracne. In more severely poisoned subjects a number of other toxic effects are seen, the most important of which are liver damage and neurological effects.

TCDD causes cancer in rats when included in the diet and in mice when included in the diet or applied to the shaved backs of this species. The current data do not indicate there has been an increase in cancer in humans exposed to TCDD. However, there is a suggestion that there may be a correlation between soft-tissue tumors in humans and the exposure to TCDD. Whether or not there is a cause-effect relationship between soft-tissue tumors and TCDD exposure will require additional studies.

Stephen M. Ayres, M.D.
Department of Internal Medicine
St. Louis University Medical School

This summary had not been received at the time this report was published. For a brief description of Dr. Ayres' speech, please refer to page II-8.

Phillip C. Kearney, Ph.D.
Chief, Pesticide Degradation Lab
Agricultural Environmental Quality Inst.
Agricultural Research Center, USDA

This summary had not been received at the time this report was published. For a brief description of Dr. Kearney's speech, please refer to page II-10.

Jurgen H. Exner
IT Enviroscience

The properties, analysis, and fate of dioxin in soil were described. Data from the literature and from IT Enviroscience's internal research program were extrapolated to potential cleanup approaches. These data show that treatment of dioxin in soil is technically feasible. These treatments, in situ photolysis or chemical treatment and on- or off-site extraction or thermal destruction in incinerators or cement kilns, can be implemented within 2-4 years and are alternatives to long-term process were illustrated by describing the development and large scale implementation of a process that destroyed 14 lbs of dioxin in Nepacco waste contained in a storage tank at Verona, MO. The time from process conceptualization to startup of the chemical destruction plant was 20 months.

Important properties of dioxin that affect selection of treatment processes are low vapor pressure, extremely low solubility in water and organic solvents, and very high soil/water partitiion coefficient. Precise analyses of dioxin in soil are difficult because of non-homogeneity and the possibility of time-dependent adsorption. Therefore, information on the disappearance of dioxin in soil must take into account the validity of the analytical method.

The fate of dioxin in soil is determined by its strong sorption to soil, by vaporization in the ng-fg/m^2 concentrations from the surface of soil, and by photodegradation. Microbial degradation is insignificant, and plant uptake is small.

Field data from Seveso and ITE internal data support rapid photodegradation of dioxin in soil. Important parameters that must be known for an in situ soil treatment process are soil condition and preparation, reaction time and products, and environmental impact. The principle of in situ photodegradation is proven, but accurate economic information requires knowledge of the important process parameters. These parameters must be established in a demonstration project.

Similarly, the principle of extracting dioxin from soil is proven. Valid economics for such a process require understanding of the various methods of contacting solvents with soil; soil condition, consistency, and wettability; extraction efficiency; and solvent recovery and recycle.

Thermal destruction by incinerators or cement kilns requires proper feed preparation, appropriate feed rates and operating conditions, and acceptance of air, water, and ash discharges. The destruction and removal efficiency, which should be greater than 99.99%, depends on dioxin's thermal stability, the residence time, oxygen concentration, and flame configuration within the thermal oxidation system. The rate of thermal desorption of dioxin from soil and the destruction rate of dioxin adsorbed on particulate matter are other important parameters.

F. Jay Murray, Ph.D., D.A.B.T.
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HEALTH EFFECTS OF DIOXIN AND ACCEPTABLE LEVELS IN THE ENVIRONMENT

The Centers for Disease Control (CDC) has recommended one part per billion (ppb) as an acceptable level of dioxin (2,3,7,8-TCDD) in soil. Their primary consideration in recommending this level was a concern for children eating soil on a daily basis. CDC's one ppb recommendation is not designed to represent an "all purpose" standard, covering all situations of dioxin contamination. Rather, it is a conservative recommendation, designed for residential property and other areas where children may eat soil on a daily basis.

Levels of dioxin in soil above one ppb should be acceptable for situations where there is less potential for exposure. In fact, CDC has stated that higher levels of dioxin might be acceptable at some other sites such as roads and parking lots where less human contact is likely. In order for dioxin to pose a potential hazard to human health, people must actually be exposed to the dioxin. For example, little hazard to human health is posed by dioxin-contaminated soil buried under a paved road since there would be virtually no opportunity for human exposure.

In humans, no conclusive evidence exists that dioxin has caused cancer, reproductive effects or birth defects. Health effects in humans include: chloracne, liver effects, neuromuscular symptoms, altered porphyrin metabolism and a number of subjective symptoms. Chloracne is considered the hallmark of overexposure to dioxin.

Pure dioxin is extremely toxic in most animal species. Dioxin has produced cancer in laboratory animals (rats and mice) given the compound over the animals' lifetime. In animals, dioxin-related cancer has only been seen at dose levels great enough to cause other serious toxic effects. No effect levels have been established in laboratory animals. These no-effect levels are important because they are used to predict acceptable levels for humans.

Factors which may affect the potential hazard of dioxin in soil include: concentration of dioxin, location (e.g., surface versus buried), accessibility (e.g., restricted area), type of human activity (e.g., play area for children), presence of grass or ground cover, opportunity to accumulate in food chain, proximity to ground water/streams, potential for erosion, and the presence of oils or solvents. In conclusion, standards for sites must be determined on a case-by-case basis since the circumstances and potential for exposure will vary.

Henry Falk, M.D.
Chief, Special Studies Branch
Chronic Disease Division
Center for Environmental Health
Centers for Disease Control

This summary had not been received at the time this report was published. For a brief description of Dr. Falk's speech, please refer to Page II-11.

CHAPTER 3

OPTIONS FOR REMEDIAL ACTION

Introduction

A number of different options for remedial action have been proposed. This chapter examines in detail the current technology available to stabilize, treat, or dispose of the dioxin contamination in the state.

It must be stressed that certain options may be more suited to some sites than others. Therefore, each option must be compared on a site by site basis. This chapter attempts to review all possible means of remedial action, but does not recommend one option over another. Also, several of the technologies discussed may not be developed enough for immediate, full scale use. And while every attempt was made to include all possible technologies, exclusion of any technology does not necessarily mean that it is not a viable technology.

A great part of the information in this chapter was supplied by CH_2M-Hill, the Environmental Protection Agency's feasibility contractor for the Minker/Stout site. A summary of their remarks before the Task Force is available in Appendix VIII.

Stabilization In Place

Possibly one of the simplest means of dealing with the situation in terms of actual handling of the contaminated soil is to stabilize or secure it in place. This can be accomplished in the following manner.

In order to insure that the contaminated soil is unable to move, and that the dioxin cannot be leached from the soil, some sort of impermeable barrier must be installed. Once the extent of the contaminated area has been determined, a wall of impermeable material is installed around the site perimeter to a depth which penetrates bedrock. This wall or curtain can be made in several different ways. A grout curtain of cement can be installed by drilling a double wall of large diameter holes around the site down to bedrock and filling them with the cement, which will then spread to form the seal. Or a trench can be dug around the site and filled with a slurry of bentonite mixed with soil. The bentonite will then expand to fill any cracks or crevices in the rock and form a seal.

Along with sealing the perimeter, a clay cap at least 4 feet thick must be placed over the site to seal it from above. It is imperative that no groundwater movement occurs through the site.

It is important to note that the success of this method of containing the the contamination is highly dependent on site geology. If the site is unstable geologically, for example with large cracks and fissures in the rock, complete containment of the soil cannot be insured. Also, this method requires a great deal of onsite construction, and once completed, must be monitored indefinitely.

Another, somewhat simpler means of fixation in place is the addition of a fixative directly to the top layer of soil. Either an inorganic fixative or an organic polymer can be used. Examples of inorganic fixatives include cement or flyash, and organics include polyurethane, polyvinyl acetate and urea formaldehyde. The fixative is sprinkled onto the soil surface and tilled in, forming an impermeable cap over the contamination. Disadvantages of this method include the tendency of the fixative to break down over long periods of time.

The great advantage of this method is that contact with the soil itself is minimized in that the soil remains undisturbed. Of course, this method does not remove or destroy the dioxin, but minimizes its availability to the environment.

On Site Storage

Onsite storage essentially involves creating a landfill directly on the site. Some type of structure is constructed either above or below ground to hold the contaminated soil, and prevent exposure to the environment.

With storage below ground, some type of impermeable liner such as clay or a synthetic sheet must be used in order to prevent possible groundwater contamination. Some type of leachate collection system and monitoring wells are also necessary.

Above ground storage can be accomplished with either a concrete structure or an above ground cell with an impermeable clay or synthetic liner. In either case long term monitoring is required.

This method involves a slightly increased risk of exposure in that the soil must be excavated, causing possible dust formation. One advantage is that contaminated material from all the sites can be consolidated on a single site with proper geologic and geographic requirements. Of course, this would involve some highway transport of the wastes, with the attendant increase in risk.

Transport To Secure Landfill

Transport to a secure landfill involves many of the same principles as on site storage. This option includes excavation of the contaminated soil, and removal from the site by truck to a properly permitted hazardous waste landfill.

Since highway transport would be necessary with this option, the vehicles actually transporting the waste would have to comply with the Department of Transportation (DOT) regulations. Up to this point, the only contaminated soil which has been shipped has been for samples. These samples were classified as a Poison B. If bulk shipment of the contaminated soil stays under the same classification, then the soil would have to either be packed into drums or other containers which met DOT specifications for Poison B, or shipped in a tanker which would also have to meet DOT specifications for Poison B.

Once the contaminated soil reaches the landfill, it is treated in much the same manner as in on site storage. Either an above or below ground cell is constructed to hold the material, and all monitoring requirements become the responsibility of the landfill operator. Two landfills exist within a 600 mile radius which are capable of taking the soil.

A major advantage of this option is that with replacement of the excavated soil, the site once again becomes habitable. Some of the disadvantages include the large cost of transport for the distance required, as well as the increased risk of exposure, both from dust formation on site during excavation, and accidental spillage during highway transport. And like on site storage, this method does not destroy dioxin, but minimizes its availability to the atmosphere.

Incineration

One of the most commercially available means of destroying the dioxin in the contaminated soil is by incineration of the soil. Incineration is widely used in the destruction of other hazardous wastes, and can be considered a proven technology.

The basic principle of incineration is to destroy the dioxin molecule by thermal degradation, and several different incineration techniques are available to accomplish this. The first is by direct incineration of the contaminated soil. Although no test burn data on dioxin is available, by comparison with other highly chlorinated organics it is felt that a temperature of 2300°F and a residence time of 2 seconds would be sufficient to destroy the dioxin. Some of the problems with direct incineration of the soil include the thermal inefficiency of heating that large a quantity of soil to 2300°F, and the slagging and other problems which would occur inside the incinerator.

Another incineration technique which would avoid the problems mentioned above is pyrolytic incineration. This is a two-step process in which the contaminated soil is first placed in some type of rotary hearth or other kiln and baked at 1600°F to drive off the organics, including the dioxin. The organics rich gas stream is then fed to an afterburner operating at 2300°F with a 2 second residence time to destroy the dioxin. In this way, the soil itself is not subjected to the higher temperatures.

No matter what basic incineration techniques are used, certain requirements must be met in regards to site preparation, emission controls, and the like. Of course, the soil must be excavated, and some type of size reduction facility will be needed, since the excavated material will probably contain trees, rocks, concrete, road materials, and other materials unsuitable to feed directly into an incinerator. For this size reduction facility, the feed will either have to be dried out or slurried to facilitate handling. The rocks and other large objects will be separated from the soil, screen graded, and crushed in some type of crusher such as a ball mill. If the feed is slurried in, then some type of concentrating step must be performed and the resultant feed put in some type of form suitable for feed into the incinerator, such as pellets. All soil handling should be done by enclosed conveyor, with sprays, splash guards and other suitable dust suppression equipment on all handling equipment.

The actual incineration of the materials can be accomplished by either of the techniques outlined above, in addition to the following emission control requirements. Some type of dust collector such as a bag house is needed to trap particulates from the rotary kiln. The trapped particulate can then be fed back into the rotary kiln. Emissions from the afterburner can be trapped by a venturi scrubber in series with a packed scrubber to trap any organics in the effluent. Process water from the packed tower must then be treated, possibly by reintroduction to the kiln of the solid residues removed.

One possibility not yet mentioned would be to convert an existing area cement kiln to burn the contaminated soil. Several such kilns exist in the eastern part of the state and are not currently in use. Most if not all of the kilns have size reduction equipment already in place. However, the kiln would have to be retrofitted extensively with adequate pollution control devices as outlined above to ensure containment of the dioxin.

Although the technology to incinerate the contaminated soil is currently available, several questions remain to be answered about matters such as ash disposal. Estimates place the amount of ash which will remain at 80% of the original volume of incinerated soil. Since no test burn data is available for dioxin in soil, the residual amount of dioxin in the ash has yet to be determined. And this ash must still be considered a hazardous waste by definition, and would have to be disposed of in a secure landfill. However, if the residual level of contamination in the ash is within acceptable levels, the possibility exists that the ash can be delisted, making disposal much easier. In the case of the cement kiln, the ash may possibly even be used as a component of cement.

The major advantage of incineration is the fact that it is an accepted, proven technology which is already in use on similar compounds. The major disadvantage is the increased risk of exposure, not only from dust caused by excavation of the soil, but from volatilization of the dioxin from the incineration process. Another disadvantage is that the ash is, by definition, still a hazardous waste.

Solvent Extraction

Extraction is a process whereby the dioxin is stripped from the contaminated soil with some type of solvent, which is then treated to remove or destroy the dioxin. The purpose of this method is to get the dioxin into a medium more suitable to work with, and to leave decontaminated, and possibly usable, soil.

A number of different common solvents exist which would be capable of extracting the dioxin from the soil. Examples of such solvents include toluene, hexane, and freon. The solvent that works the best will have to be determined by testing. A less common extraction technique is the use of supercritical fluids. A supercritical fluid is a gas such as carbon dioxide which has been compressed into its liquid phase. Supercritical fluids have shown promise as extracting solvents, and hold one advantage over common solvents. Most of the common solvents which would be suitable as extracting solvents are also highly toxic in and of themselves. And even if the soil is put through a dryer, a certain amount of the solvent will remain with the soil, usually 1-2% by weight. Use of a solvent such as liquid carbon dioxide would not pose a risk to human health. However, the use of a supercritical solvent has some major drawbacks. First, the technology has not yet been developed to a commercial scale. Secondly, working with anything under extreme pressure always increases the risk of explosion or other mishap.

Once a suitable solvent is chosen, the actual extraction process can be examined. First, size reduction equipment will be necessary as described in the section on incineration. The actual extraction will require some type of multi stage contactors. The soil is washed with solvent in the contactor, the solvent is then drained off, and additional fresh solvent is added. This process is repeated until the dioxin in the soil is reduced to an acceptable level. Current estimates place the aproximate number of washes necessary at 10 to 15. When the soil is deemed clean, then it must be dried to remove as much solvent as possible. The contaminated solvent from the various washes is concentrated by means of distillation, or cleaned by some type of adsorption process using activated charcoal or resin. All of the steps described above must be done under vacuum, to insure that no contamination is leaked to the environment.

The last step would be to treat the concentrated solvent in some manner to remove or destroy the dioxin. This can be done in a number of ways. One process which has already been performed is that of U.V. photolysis. Ultraviolet light is shone on the contaminated solvent, ultimately breaking the bonds in the dioxin molecule. Gamma radiation works in a similar manner. Another process is the addition of a chemical reagent to the solvent to dechlorinate the dioxin molecule. Still another option would be to incinerate the contaminated solvent.

Ideally, the major advantage of this method is that the clean soil can be returned to the site, and the contaminated solvent is more easily treated. Also there are a variety of commercially available solvents capable of extracting the dioxin. Unfortunately, there are a number of disadvantages associated with this method. First, no doubt exists that dioxin can be extracted from soils. This is how samples are tested for dioxin. However, no attempt has been made to demonstrate this process on a commercial or even a pilot scale for the treatment of dioxin. Another major drawback is the toxicity of most solvents which are capable of extracting the dioxin. If 1 or 2% by weight of the solvent is left in the soil, chances are the soil would be more toxic than it was prior to removal of the dioxin. Also, liberating the dioxin from the soil with a solvent increases the availability of that dioxin to the environment, increasing the risk of exposure.

Biodegradation

One of the least expensive and most environmentally desirable techniques for breaking down large organic molecules is to subject the molecules to the action of microorganisms. Numerous studies have examined the susceptibility of dioxin to microbial decomposition, mostly in the uncontrolled environment.

Early studies appeared to indicate that dioxin in soil in the environment was disappearing, possibly due to biodegradation. Unfortunately, later studies showed that the disappearance was due to the increasing difficulty of recovering the dioxin which bound more tightly to the soil with time, rather than any microbial action on the dioxin molecule.

More recently, research programs are being conducted to establish organisms which will ultimately degrade dioxin and other chlorinated ring structures directly in the environment. These projects seek either to modify naturally occuring organisms or to apply recent advances in recombinant DNA technology to genetically engineer organisms which are capable of degrading dioxin.

Many of the studies now being done are focusing on the genetic modification of a number of organisms such as anaerobic and aerobic bacteria, yeasts, and algae. Ideally, the organism created would be able to survive in the environment with direct application to the contaminated soil. A more realistic approach may be to engineer an environment more suitable to the organism, and place the contaminated soil within the artificial environment. This approach would allow the control of specific conditions such as temperature, light, oxygen, and pH to insure the survival of the organism.

Following is a sampling of the research projects under way to study the development of organisms capable of degrading dioxin and other chlorinated ring structures.

At the University of Illinois, a bacterial culture capable of degrading 2,4,5-T has been developed by gradually exposing a mixed culture to increasing strengths of the compound. Research is currently underway to develop a variant of the 2,4,5-T bacteria capable of digesting dioxins by use of the same gradual acclimation method. At the University of Helsinki in Finland, researchers are studying the degradation of pentachlorophenol by organisms either anaerobically or aerobically in an effort to understand and apply the mechanisms involved to dioxins and other chlorinated ring structures. And at the University of Cincinnati, studies are being conducted on yeasts to determine if mammalian genes for chemical degradation can be successfully implanted or transferred.

If some type of organism can be discovered which can indeed biodegrade dioxin, the advantages are obvious, both economically and with respect to handling of contaminated materials. However, it must be stressed that at this point, no organism has been discovered, and such a discovery could be many years in the future.

In Place Treatment

The option of in place teatment is an attractive one in that the contaminated soil could be left in place and treated in some manner to destroy the dioxin. There have been a number of different methods proposed to accomplish this. However, most of the proposed methods have not been developed to the extent that they can be considered as viable technology. Some of these methods are described below.

Perhaps the most technologically developed and the most familiar method is that of ultraviolet (uv) degradation, also know as photolysis. Uv degradation was discussed briefly in the extraction section of this chapter in connection with destruction of dioxin in a solvent medium. When used for the decontamination of soils, the method works as follows. Some type of hydrogen donor such as olive or vegetable oil is applied to the surface of the contaminated soil. Then some type of uv light emitting unit is passed over the soil surface, and the dioxin molecules within reach of the uv radiation are destroyed. Then the surface is tilled to expose fresh soil and the procedure is repeated. This method is most suited to areas where the contaminated soil is shallow. However, some uncertainty exists as to the efficiency of this method, because uv radiation cannot penetrate the soil to any depth, and no estimates are available as to the number of passes necessary to appreciably reduce the dioxin level in the soil.

Another possible method of in place treatment which is similar to uv degradation is the use of gamma radiation. Like uv radiation, gamma radiation destroys the chlorine bonds within the molecule. However, gamma radiation is still in the experimental stage, and will need further developement before it can be considered a viable technology.

Chemical treatments have also been researched in regards to in place treatment. Work has been done on the addition of a sodium containing compound such as sodium polyethylene glycol to the soil along with a hydrogen donor. The sodium compound would then act to dechlorinate the dioxin molecule. But once again, this method is still experimental, and needs further work before it can be added to the list of viable alternatives.

Biodegradation can be considered as an in place treatment method, because of the possibility at some point in the future of simply adding the proper organism directly to the soil to decontaminate it. However, as discussed in the previous section on biodegradation, no organism has been isolated or created which is capable of degrading dioxin yet. Current estimates place the length of time to possibly develop such an organism at 5 years or more.

The advantage of in place treatment is obvious in that soil handling and site disturbance are minimal. However, the disadvantage is the lack of a currently available means of treating the contaminated soil in place.

Stabilization In Place Until New Technology Develops

Throughout all the sections in this chapter, certain technologies have been discussed as having merit, but not being available at this time because of a lack of technological or commercial development. The purpose of this last potential remedial action is to provide an opportunity to allow these and other emerging technologies a chance to develop so that they can be considered as viable options in solving Missouri's dioxin problem.

Initially, some type of interim stabilization measures would be implemented, such as capping or sodding to combat erosion, and other measures to insure that no movement of dioxin is occuring. Site access would be restricted to reduce exposure to the contaminant, and a monitoring program would be implemented to see if any movement is occurring. Concurrently, the development of new technologies for the destruction of dioxin would be observed closely and encouraged.

Some of the emerging technologies as mentioned in previous chapters are as follows. Extraction, especially with supercritical solvents, although a proven technology at the bench scale, has yet to be proven commercially for this type of contaminated soil. Biodegradation holds eventual promise as a simple, effective method, but as yet no organism has been discovered that can successfully degrade dioxin. Gamma radiation and chemical treatment with sodium reagents are two possible methods of in situ treatment which require further study.

Still further emerging technologies which were not mentioned in previous sections include lasers, wet air oxidation, and plasma arcs. Treatment with lasers involves the use of a beam of even intensity to destroy the dioxin molecule. Wet air oxidation involves suspending the contaminant in an aqueous solution at elevated temperature and high pressure, and bubbling air or oxygen through the solution, oxidizing the dioxin molecule. The plasma arc utilizes high energy radiation to almost instantly decompose the dioxin into elemental components.

The possibility exists that even more methods of destroying or containing dioxin will be developing in the near future. The option of stabilizing in place and waiting for new technology will provide a wider range of final remedial actions from which to choose.

CHAPTER 4

SUMMARIES OF REMEDIAL ACTION PROPOSALS FROM INTERESTED FIRMS

This chapter contains summaries of remedial action proposals which were presented to the Task Force by various waste disposal and management firms. These summaries were prepared by the firms, and illustrate the commercial stage of development of the various technologies.

Acurex Waste Technology, Inc.

Acurex Waste Technology, Inc. (AWT) is a wholly-owned subsidiary of Acurex Corporation, a diversified technology company of about $50M sales headquartered in Mountain View, California. Acurex activities include environmental and waste-treatment technologies for U.S. EPA and commercial processing of PCB oils for industry and utilities. Dr. Weitzman is a chemical engineer, chief scientist of AWT, and Manager of the Cincinnati laboratory. Dr. Schraub is Manager of Market Development for Acurex Energy and Environmental Division.

AWT is utilizing a proprietary EPA-approved commercial process to treat PCB oils. The PCB molecule is destroyed by chemically combining the chlorine atoms on the PCB molecule with metallic sodium, forming NaCl (table salt) and a small amount of long-chain polymer which is not acutely toxic and is disposable. This novel process also destroys the chemically similar dioxin compound, 2,3,7,8-TCDD, by the same sodium reduction reaction yielding salt and polymers. The destruction process is combined with solvent extraction soil cleaning methods using a special blend of AWT solvents to make up the Acurex Dioxin-Soil Cleanup Process described in this testimony.

The Acurex Dioxin-Soil Portable Cleanup Process consists of these steps:

1) Obtain permits and approvals.
2) Move portable equipment to contaminated site. Equipment includes enclosed soil moving front loader; required number of soil washing modules (Tanks in which soil, rocks, etc, are washed in solvent with agitation); Solvent Reclamation Unit; Clean Solvent Tank; Analytical Laboratory and Records Office for verification and process quality control Sample analysis; Personnel Trailer: special equipment; toilet and shower and clothes dry-cleaning unit; Water truck.
3) Prepare site including security, wetting of soil for dust control, size reduction to fit into about 20 cu yd washing modules.
4) Scoop soils, etc. into washing modules.
5) Fill with clean solvent and agitate at room temperature and pressure for about one-half hour

6) Pump out solvent and refill with clean solvent and repeat washing process until solvent shows soil to be below desired contamination level (such as 1ppb). Note that a unique relationship exists between dioxin left in soil and that extracted into the solvent.
7) The contaminated solvent is then purified for recycle in a low-power standard distillation process.
8). The remaining small volume of solvent containing all dioxins is transferred to a reactor where Acurex reagents and sodium metal dispersion are added to destroy the toxic dioxin by removing chlorine atoms.
9) Dispose of the small volume of dioxin-free sludge (e.g., 100,000 cu yds soil produces less than 1 cu yd of sludge).
10) The safe soil is returned to its original location or placed in suitable location as regulations allow.
11) Equipment is removed and site regreened.

The AWT process has been verified in Missouri by an actual cleanup of a sample of Minker-Stout soil provided by the Department of Natural Resources at the University of Missouri Environmental Trace Substance Center in Columbia during February, March and April of 1983. Some analytical problems (interference peaks in the gas chromatograph analysis) were encountered but overcome by the combined efforts of DNR, U. of Missouri and Acurex staff.

The Acurex Dioxin-Soil Portable Cleanup Process is estimated to cost about $250 per cubic yard of soil for a 10,000 yard size cleanup job of which $100/yd is the variable cost.

The R&D phase and the initial laboratory demonstration phase are now complete. We believe that AWT process to be a viable technology and a serious candidate for large-scale dioxin soil cleanup and destruction. The next step is a pilot-scale (e.g., one full size washing module) field demonstration affecting a small dioxin site cleanup. Such a program was suggested at a cost of between one-half and one million dollars and a one-year schedule.

In summary, AWT has a novel process to destroy dioxins in soil which is already demonstrated to work on the Minker-Stout soil. The Acurex Dioxin Soil Portable Cleanup Process has these benefits: cleans dioxins, PCB's and other halogenated organics from soil down to a specified level; destoys the dioxin molecules; is safe using non-flammable, non-toxic solvents; is controllable and verifiable, creates no secondary pollutants or migration of existing pollutants; and is already demonstrated in Missouri.

We believe this techology merits thoughtful consideration in the solution to the Missouri dioxin soil problem.

Chemical Waste Management

Late in 1982, then the dioxin problem in Missouri began to receive a great deal of attention, Chemical Waste Management, by virtue of its extensive experience in hazardous waste site mitigation, began a search for a solution to the problem.

In the selection of a project approach, nine key factors were considered:

1. Public health and safety during project implementation and final completion.
2. Technical feasibility.

3. Operational soundness.
4. Provision of an environmentally sound and defensible solution.
5. Compliance with regulations.
6. Timing.
7. Cost.
8. Public reaction/opinion/perception (in Missouri).
9. Public reaction/opinion/perception (in other states).

Using these key factors, four potential treatment/disposal options were evaluated, breaking each option into positive and negative factors. After evaluating incineration, secure burial, treatment, and site capping/encapsulation, the following conclusions were reached:

1. Due to the large volume of soil, destruction of dioxin with the soil is cost prohibitive;
2. Since dioxin is the major concern, it should be extracted from the soil in order to reduce volume;
3. Dioxin which is excavated/extracted should be destroyed through incineration;
4. Again, due to the volume of "low level" dioxin-contaminated soil, removal and destruction of dioxin with the soil is cost prohibitive;
5. Site capping/encapsulation should be an alternative solution for select "low level" sites;
6. Because of public reaction/sensitivity by other states to receiving dioxin, as much of the dioxin processing/disposal should be done within the State of Missouri as is possible.

In light of these conclusions, Chemical Waste Management recommends the following primary operations:

1. Excavation of highly contaminated soils and replacement with clean fill;
2. Site capping/encapsulation of "low level" dioxin contaminated sites;
3. Extraction of the dioxin from the soil;
4. Disposal of treated soil in a secure landfill;
5. Destruction of the dioxin in the extract medium via the ultraviolet light process;
6. Incineration of the treated extract medium at sea via one of CWM's ocean incineration vessels.

Based upon the information at hand, Chemical Waste Management estimates time required for completion of the project to be from 14 to 24 months, with costs ranging from $22 million to $65 million, depending upon the establishment of acceptable levels of contamination and the number of sites to be cleaned up.

Chemical Waste Management, in conjunction with Battelle Memorial Laboratories, is currently conducting tests on dioxin contaminated soils. When those tests are complete, the preliminary estimates will be further refined.

In terms of the key factors and the evaluation of potential treatment/disposal options, Chemical Waste Management believes that the primary operations outlined above represent the most environmentally sound and cost-effective solution to the dioxin problem in Missouri.

Critical Fluid Systems Inc.

Critical Fluid Systems, Inc., is a chemical process engineering company that designs and sells a variety of proprietary extraction systems based on critical fluid technology. These new extraction techniques fill a range of processing needs in such industries as pharmaceuticals, food and organic chemicals as well as waste treatment. Critical Fluid Systems, Inc., was formed in January 1980 as a subsidiary of Arthur D. Little, Inc., to commercialize industrial applications of the patented technology. The company is headed by Dr. Richard P. de Filippi, who initiated the technical development work in the international consulting firm's laboratories in 1976; Critical Fluid Systems' offices, laboratories and pilot plant are located in Cambridge, Massachusetts.

Critical Fluid System's verstile technology, is based on the use of condensed gases as extracting solvents, which provide more economical, and more efficient, alternatives to distillatin, conventional solvent extraction and various incineration and landfill techniques for the management of hazardous wastes. For example, one critical fluid process can be used to clean up soils contaminated with toxic materials.

The State of Missouri faces a massive cleanup of dioxin contamination in Times Beach and other communities. The removal of dioxin presents some very special economic and technical problems - problems which widely used, conventional solvent technologies for waste treatment have not yet succeeded in solving.

The use of critical fluid extraction to strip soils of contaminated material is technically feasible and, in the view of Critical Fluid Systems' engineers, worth trying at Times Beach. Although never applied specifically to dioxin, the technology has been successful in extracting other chemicals and materials from soils. For example, in laboratory tests, Critical Fluid Systems' engineers have separated lubricating oils from steel-making mill scale, diesel oil from cuttings produced in oil well drilling operations, and vegetable oil from spent bleaching clays in food processing. These test results provide reason to believe that the technology might succeed in removing dioxin from soil substrates.

Company officials emphasize, however, that success in such special problems can never be assumed. They propose, therefore, that the State of Missouri underwrite a small scale preliminary test of the efficacy of critical fluids in Times Beach dioxin/soil samples. The company would work with an approved EPA contractor to process samples using a variety of condensed gas solvents and operating conditions. At the completion of the test program, which, if appropriate, would include preliminary estimates of capital and operating costs for a commercial scale facility, a recommendation would be made on the feasibility of proceeding with subsequent phases leading to construction of a full-scale processing plant to be operated by a major waste management company.

The special properties of critical fluids in separating substances have been known to chemists for many years but have only recently been applied on a commercial scale. The first commercial applications have been to extract hops for brewing and to decaffeinate coffee.

Critical Fluid Systems' patented extraction method utilizes fluids that are liquefied gases. At a certain combination of temperature and pressure, these gases reach what is called their critical point. At this point the gases are neither liquid nor vapor but have many of the solvent properties of both. These properties make extraction more rapid and efficient than processes using distillation and coventional solvent extraction methods.

Critical Fluid Systems is currently working under contract to a number of government agencies on critical fluid development projects that include applications for treating and decontaminating solid wastes, extracting chemicals, including pesticides, from waste water, and regenerating activated carbon and other adsorbents used in manufacturing.

SCA Chemical Services, Inc.

Decontamination or destruction of the dioxin contaminant in large volumes of material (soil, construction debris, and furniture) presents challenges that exceed current proven state-of-the-art destruction or decontamination techology. One method has been burial of the contaminated material in specially constructed cells. When material contaminated with dioxin is buried, the problem has been placed in storage for others to solve at a later date. Decontamination or destruction of the contaminant dioxin found in small quantities in the large volumes of contaminated soil located at the various sites in Missouri poses a technical and waste management challenge of unprecedented magnitude. SCA Chemical Services, Inc., one of the nations's largest hazardous waste treatment and disposal companies is pleased to accept that challenge.

SCA recognizes that there is no proven contamination or disposal technology capable of decontaminating large volumes of solid contaminated with dioxin levels ranging from 1 to over 300 parts per billion. The actual level of decontamination to be reached in order to certify the soil free of dioxin and suitable for unrestricted use has yet to be established. Therefore, SCA assumes that any acceptable decontamination or destruction process must achieve average levels of dioxin significantly less than 1 part per billion in the resulting decontaminated material. To date, no large-scale treatment or disposal process has been operated with documented destruction of a contaminant, especially one as persistent as dioxin, bonded to soil particles or soaked into concrete block, to achieve concentration levels significantly below 1 part per billion. It is tempting to make assumptions that certain technologies will work; however, a much more scientific approach is required in order to select candidate treatment processes. Initial evaluation by SCA for the preparation of this proposal has determined that there is no proven off-the-shelf system available to meet the dioxin decontamination challenge.

The existing situation is further clouded by the fact that dioxin is currently regulated by the Toxic Substance Control Act (TSCA). EPA proposed new regulations that would transfer dioxin from the TSCA regulation to the Resource Conservation and Recovery Act (RCRA) regulation in accordance with 40 CFR 261-265 and 40 CFR 122. After the public comment phase, the regulations will be revised to reflect public comment and concern. At this time, it is difficult to project whether or not dioxin will be transferred from TSCA to RCRA and, if so, what the content of the final regulations will be. In either case, any material contaminated with dioxin will be subjected to specific controls and regulations. Such specific controls and regulations will limit the number of commercially available treatment facilities permitted to receive or treat dioxin contaminated material. In addition, the public reaction in host states to the import of large amounts of dioxin contaminated soils must be considered. Because of these many unknowns, it is prudent to assume that the disposal or decontamination of dioxin contaminated materials must be accomplished within the boundaries of Missouri.

In order to meet the technical challenges associated with the successful design, construction and operation of a dioxin contaminated materials decontamination and disposal facility, a multi-disciplined technical and management team will be required. Such a team must have access to the most advanced technologies available in the world, be highly skilled in the evaluation and testing of all types of candidate decontamination systems, be capable of translating conceptual design to a final facility design and supervising the construction of such a facility, be knowledgeable of all regulations and hazardous waste management techniques, and be capable of operating the facility in the most effective and safe manner possible. SCA has assembled such a team to respond to the challenge issued by the Governor's task force on dioxin and the Missouri Department of Natural Resources. The team is comprised of the best technical engineering, construction management, and operational personnel available in the United States today. With the exception of Battelle Columbus Laboratories, all team members are residents of Missouri.

The initial phase of the project will be screening and evaluation of candidate technologies. Laboratory tests may be required in order to complete evaluation of these technologies. The selected technology must be verified by a full-scale pilot operation prior to commitment of the process to final engineering design. Battelle Columbus Laboratories has been selected as the technology team member because of their unequaled capability and knowledge of all types of chemical processes, their extensive laboratories and small-scale test facilities, their current experience as a leading laboratory performing analysis of dioxin contaminated soil samples from Missouri, and their extensive experience in evaluating similar types of problems for the United States Environmental Protection Agency and the United States Army. Battelle has recently completed a $250,000 technology evaluation study for the U.S. Army to determine the best means of decontaminating large volumes of toxic materials, chemical agents and chemical munitions. Battelle Laboratories, Inc. is a not-for-profit research and development center with revenues exceeding $400,000,000, much of which were directly applicable to waste management. Battelle is a recognized authority on various types of hazardous waste treatment technologies including incineration, solvent extraction, and genetic engineering. Battelle has current experience in all types of processes suitable for consideration for decontamination of dioxin contaminated materials.

Once the decontamination technology has been thoroughly tested and confirmed it must be translated into a facility design suitable for construction. SCA has chosen the Fru-Con Corporation, with World Headquarters in Ballwin, Missouri. Fru-Con is a full service engineering and construction corporation. Two elements of Fru-Con are participating in this project. Fruco Engineers, Inc. is an experienced engineering firm with an international reputation for excellence specializing in the design and construction management of cement plants, chemical plants, refineries, industrial facilities, and major buildings. Fruin-Colnon Corporation Contracting Company will provide construction of the facilities. Fruin-Colnon are experienced constructors who have constructed numerous cement plants, chemical plants, refineries, wastewater treatment facilities, and major buildings throughout the State of Missouri, the United States, and internationally. SCA Chemical Services, Inc., a wholly-owned subsidiary of SCA Services, Inc. is one of the major solid waste and hazardous waste corporations in the United States. SCA Chemical Services, Inc. (SCA) operates six major hazardous waste treatment and disposal facilities throughout the United States, has major research and development laboratories in Buffalo, New York. One of its six operational facilities owns and operates the largest commercial hazardous waste incinerator in the United States located near Chicago, Illinois. SCA is an experienced hazardous waste management company that has placed its emphasis on high technology treatment of hazardous waste. It is SCA's established philosophy that land burial of hazardous waste is the option of last resort and should only be utilized when no other treatment technologies are available. The SCA project will be directed by Mr. James L. Boyland who is currently Director of Business Development located in St. Louis, Missouri. Mr. Boyland served on the Department of Natural Resources ad hoc committee for hazardous waste and is experienced in hazardous waste regulations and hazardous waste management.

The SCA team does not approach the decontamination of dioxin contaminated material with preconceived ideas or proprietary processes. Rather, it will evaluate the situation, all relevant data, and proceed to identify those solutions that appear practical. It is the purpose of this proposal to provide the State of Missouri with the option to go forward with the technology development, design, construction, and operation of a dioxin decontamination facility. Such a facility may prove to be necessary in the event that other licensed hazardous waste disposal or treatment facilities are unable or unwilling to accept the large volumes of dioxin contaminated soils from Missouri. The high cost of transportation of such large volumes of soil to remote facilities is also a major cost consideration. With a proper plan and qualified contractors, the State of Missouri should be able to request and obtain federal funding for the required technology development, design, construction, and operation of a dioxin contaminated facility located within the State of Missouri. The SCA team stands ready to support the State of Missouri in any way possible in order to achieve such a goal.

Rollins Environmental Services, Inc.

Presently, there exists many options for the destruction or disposal of extremely hazardous waste (i.e., PCB's, radioactive waste, organic pesticides and herbicides) and many of the same methodologies are being suggested for the treatment of dioxin waste. Unfortunately, there is very little data, if any, on the effectiveness of these methods to treat dioxin materials. Compounding this problem is the large volume of dioxin waste material requiring treatment in Missouri. Most of the present options are not applicable to a large scale problem such as this, except at extreme costs.

Rollins Environmental Services (TX) Inc. (RES) presents the following discussion of thermal oxidative destruction, solvent extraction and land disposal methods that may have application to the dioxin problem. Suitability of these options is based on economics, scale of operation and practicality of the solution. To minimize the number of cost options, we assumed the establishment of a local treatment facility in the St. Louis area for on-site versus off-site (out-of-state transportation cost) treatment.

Thermal Destruction

Our best estimate of the cost of off-site incineration is $234,806,000.00. This number includes excavation, transportation and incineration at a permitted facility. The project time for off-site incineration is estimated to be 3-5 years. On-site incineration with a portable or temporary unit would also be a time intensive option costing $411,100,000.00 and taking greater than 20 years to complete the incineration of the waste with available technology.

Equally important is the disposal of large quantities of incineration ash generated by this disposal method. If the waste ash can not be delisted, it will require disposal in a Class I landfill, further increasing the total cost of incineration.

Solvent Extraction/Oxidative Destruction

Both solvent extracton and oxidative destruction methodologies, in theory, are viable options for dioxin removal, but the technological-operating problems to apply these methods would be more cost intensive than incineration.

Land Disposal

Land disposal, as an option for the treatment of extremely hazardous waste is quickly becoming obsolete with respect to other treatment technologies. The issue of securing a hazardous waste below grade is being questioned because of the uncertainties of these facilities.

Rollins Environmental Services (TX) Inc. is engineering (is developing) an above ground closure method that addresses the problems associated with the below grade secure landfill. The advantages of our above ground closure method include:

1. Only one handling is required and this is bulk handling. Therefore, dissemination of the material is greatly reduced.
2. If the contaminated soil is land-disposed in a secure, aboveground, well monitored site, the problem of future contamination is eliminated.
3. The time of removal from public area is rapid.
4. Transport costs and hazards of shipment to a centrally located above ground closure are minimized.

5. Dioxin has an extremely low solubility in water. If protected from rainfall and surface runoff, it is non-migratory.
6. There is a half life to dioxin - but this may depend on matrix and environmental conditions.
7. There are reports of 5 strains of bacteria which are active with dioxin - leading to the interesting possibility that the material may be inoculated to speed dioxin destruction.
8. Naturally, costs would be appreciably lower for local land disposal.

Above ground land disposal is an environmentally sound method for this waste because it is protected from rainfall and runoff contamination. The construction of a central storage for all dioxin waste, if monitored by a responsible organization, will give the best remedy for the available funds until a final solution is found.

Cost Estimates for Above Ground Closure

Past project experience would indicate that the entire contamination (as presently known) in the St. Louis area could be contained in a twenty acre site with a twenty foot high above ground closure. Assuming the same transportation and excavation costs outlined earlier and assuming one centrally located site, the estimated costs are as follows:

Excavation	$12,500,000
Transportation	$ 1,600,000
Construction & Waste Placement	$12,500,000
Total Estimated Cost	$26,600,000

Project time could be very flexible. Individual sites could be excavated at the rate of 1000 to 3000 cubic yards per day. The limiting factor would be at the central closure site. However, this site could be designed to handle 5,000-10,000 cubic yards per day. Based on these handling rates, the project culd be executed in 2-3 months at an estimated cost of approximately $50.00 per cubic yard.

The above ground closure approach obviously satisfies the economic, scale of operation and practical considerations.

Summary

In summary, Rollins Environmental Services (TX) Inc. recommends a land disposal/above ground closure methodology for the dioxin problem in Missouri because:

1. It places the dioxin contamination in a secure position, out of the public domain, within the fastest time frame,

2. present technologies of incineration and solvent extraction may create additional hazards due to multiple handling of the waste material, and

3. an above ground closure facility will insure secure storage of the dioxin waste until such time as a viable treatment method is developed (i.e., chemical or biological degradation).

IT Enviroscience

It Enviroscience (ITE), a division of It Corporation, has been engaged in dioxin decontamination and analysis activities for the last six years. We are familiar with the literature and much of the on-going research in this field. Also, we have expended considerable internal resources in testing destruction techniques in the laboratory and in evaluating various engineering concepts. We believe that there are several technically feasible dioxin treatment methods that have been proved in principle. These methods are thermal destruction, soil extraction and treatment of the extract, in-situ soil treatment, and soil stabilization.

ITE does not recommend any single treatment technology at this time but proposes technical evaluation for comparison purposes. After the final decision, IT is prepared to design and/or operate the disposal process.

Selection of the appropriate treatment technique for Missouri soil should await technical demonstration of the method. In this manner, important scientific, engineering, and economic parameters can be defined. In the proof-of-principle stage of any process, differences in engineering assumptions commonly lead to variations in cost estimates. Only after technology demonstrations in engineering scale equipment can appropriate comparisons be carried out.

Thermal oxidation evaluation requires two parts, an engineering study to define the ITE-cement kiln process, described below, and a demonstration of thermal destruction efficiency using the U.S. EPA mobile incinerator. The engineering study is estimated at $50,000 in 3 months, the demonstration burn at $1,000,000 within one year.

Technical demonstration of solvent extraction can be carried out for about $250,000 in 9 months, demonstration of in situ treatment can be provided for $100,000 to 250,000 in 9 months, and demonstration of soil stabilization for about $200,000 in 9 months.

ITE has facilities suitable for handling large quantities of dioxin and the personnel knowledgeable in dioxin chemistry, engineering, waste incineration, and plant operation. We propose an unbiased technology evaluation of processes that the Governor's Task Force wishes to pursue by weighing toxicological, technical, economic, and social factors.

Thermal Oxidation

At present, thermal oxidation of dioxin-contaminated soil has the highest probability of any process for achieving complete destruction of dioxin. Technical parameters that must be defined are the destruction and removal efficiency (DRE) for dioxin, which is a function of dioxin's stability and rate of volatilization from soil. Other important parameters are soil variability, handling, and feed rate to a rotary kiln. These and other questions must be addressed before the public will accept thermal destruction as an appropriate technology.

Suitable demonstration of this technique may be carried out by using the U.S. EPA's mobile incinerator.

Economics of thermal destruction depend on the equipment used for that purpose. A new rotary kiln incinerator, shown schematically in Fig. 1, can be designed and built within 2 to 4 years at a capital cost of about $7-20,000,000.

A potentially more economical solution involves modification of an existing cement kiln. Normal considerations for using cement kilns for hazardous waste destruction require adding a secondary combustion chamber, as in Fig. 1, and air pollution equipment to the cement kiln. Use of a secondary combustion chamber adds capital and large fuel costs to normal costs of operating a cement kiln. Fuel costs for such a facility range from $50-200/ton of soil.

Cement kilns provide large residence times for solids at temperatures equivalent to those found in rotary kilns of hazardous waste incinerators. If dioxin-contaminated soil is introduced at the burner end of the cement kiln, there is adequate time to obtain burnout of dioxin at 2,000°F before the ash is dripped into the clinker cooler. The gases will be exposed to 2200°F for more than 2.2 seconds, normal requirements for destruction of PCB, a compound of similar stability as dioxin. This concept is illustrated in Fig. 2. The recycle of decontaminated soil is for purposes of heat and material balance and for gas cooling and cleaning.

If this ITE concept can be demonstrated and an existing kiln can be modified, low costs relative to other thermal processes can be achieved. This cost is presently estimated at $180-300/ton based on the above concept. This figure includes fuel costs, equipment modifications, a rental fee, and other operating costs.

Extraction/Treatment

Extraction of dioxin-contaminated soil with appropriate solvents can remove dioxin from soil and allow concentration of dioxin in small quantities of extracts. The dioxin in these extracts can be destroyed by incineration, chemical treatment, or ultraviolet light irradiation.

Experiments by ITE show that three successive batch extractions using three volumes of solvent to one weight of soil reduces the dioxin concentration in soil from 360 ng/g to 4 ng/g, a reduction of greater than 98%. The third extract indicated <1 ng/g, residual dioxin in soil. However, rigorous Soxhlett extraction revealed the 4 ng/g residual concentration. These data confirm literature indications that a fraction of dioxin in soil is bound very tightly.

Important technical parameters that must be addressed are soil variability, selection of solvent, type of extraction equipment, phase separation, and solvent recovery. Estimated costs for a 100 ton/day fixed extraction/photolysis plant are $150-300/ton These costs include excavation, 4-year depreciation of capital, maintenance, utilities, raw materials, labor, and miscellaneous items such as overhead, insurance, etc. Cost does not include final soil disposal.

A major concern about soil extraction is the ability to achieve residual dioxin levels of less than 1 ppb. If that level is unachievable for technical or economic reasons, is there value in reducing the concentration range of dioxin in soil to 1 to 10 ppb?

In Situ Treatment

Destruction of dioxin in soil without soil removal is attractive because there is no need for final soil disposal. Two techniques appear technically feasible.

The first, UV irradiation of soil sprayed with a solubilizing liquid, has been demonstrated at Seveso with sunlight (H. K. Wipf et al., in "Dioxin, Toxicological and Chemical Aspects," F. Cattabeni, A. Cavallaro, G. Galli, eds., Spectrum Publications, Inc., NY, 1978, p. 201-217). ITE is carring out experiments with synthetic UV light. The major questions are rate of reduction, degree of reduction, and environmental impact of reaction products.

If soil contamination is very deep, provisions must be made to plow up or excavate, disk, rake, and spread the soil. Preliminary cost estimates for in situ photo-chemical treatment are $50-250/ton. This estimate includes capital depreciation, chemical, labor, and maintenance costs.

Chemical in-situ treatment is being explored by the U. S. EPA-Cincinnati. The proposed reaction involves displacement of chlorine atoms by alkali-metal alkoxides.

Soil Stabilization

Soil stabilization may be achieved by a number of proprietary procedures. The intent of these methods is to fix the dioxin and reduce its potential for migration. Since dioxin in soil does not appear to migrate, additional reduction in soil permeability and dioxin availability must be defined. At that point, decisions of relative risks and costs will decide the suitability of this procedure.

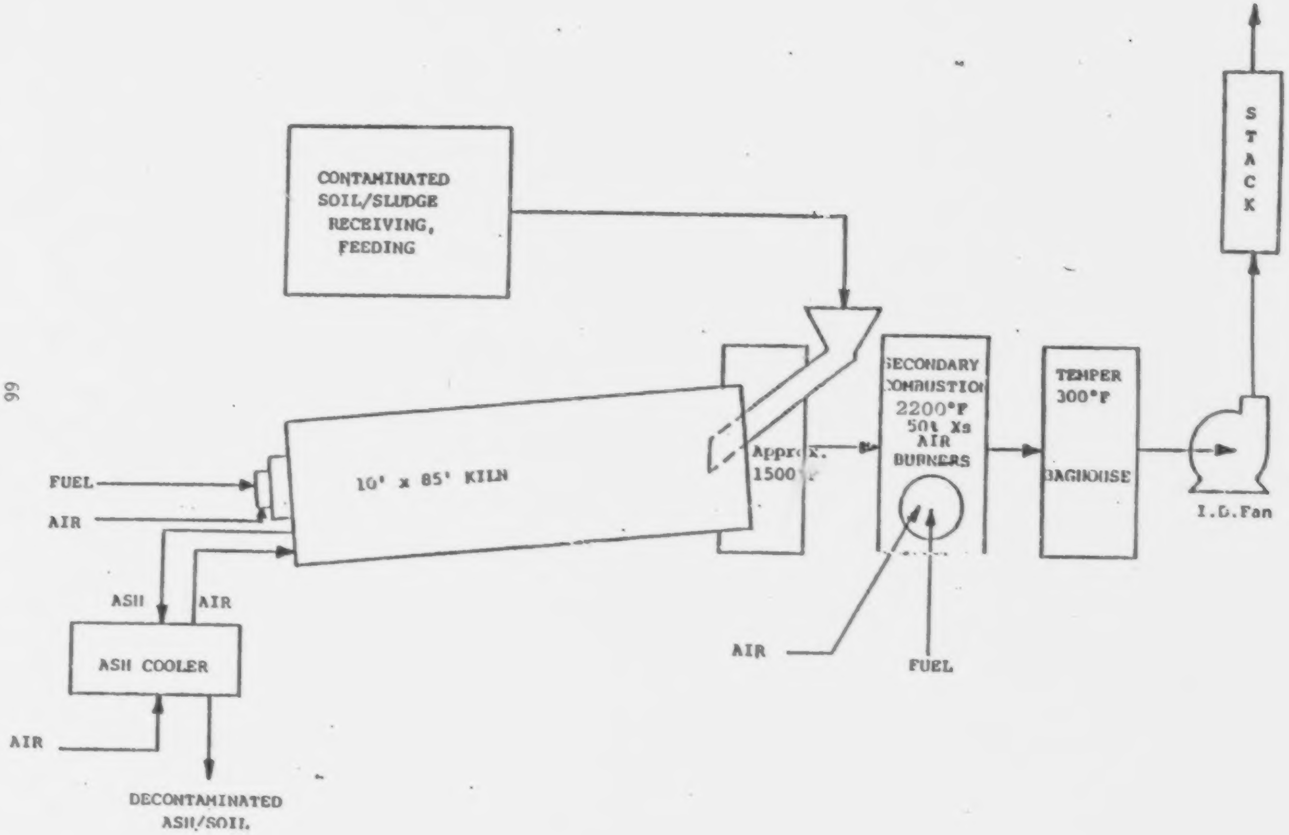
CONTAMINATED SOIL/SLUDGE RECEIVING, FEEDING
10' x 85' KILN
FUEL
AIR
ASH
AIR
ASH COOLER
AIR
DECONTAMINATED ASH/SOIL
Approx. 1500°F
SECONDARY COMBUSTION 2200°F 50% Xs AIR BURNERS
AIR
FUEL
TEMPER 300°F
BAGHOUSE
I.D.Fan
STACK

Fig. 1. New Rotary Kiln Thermal Oxidation System

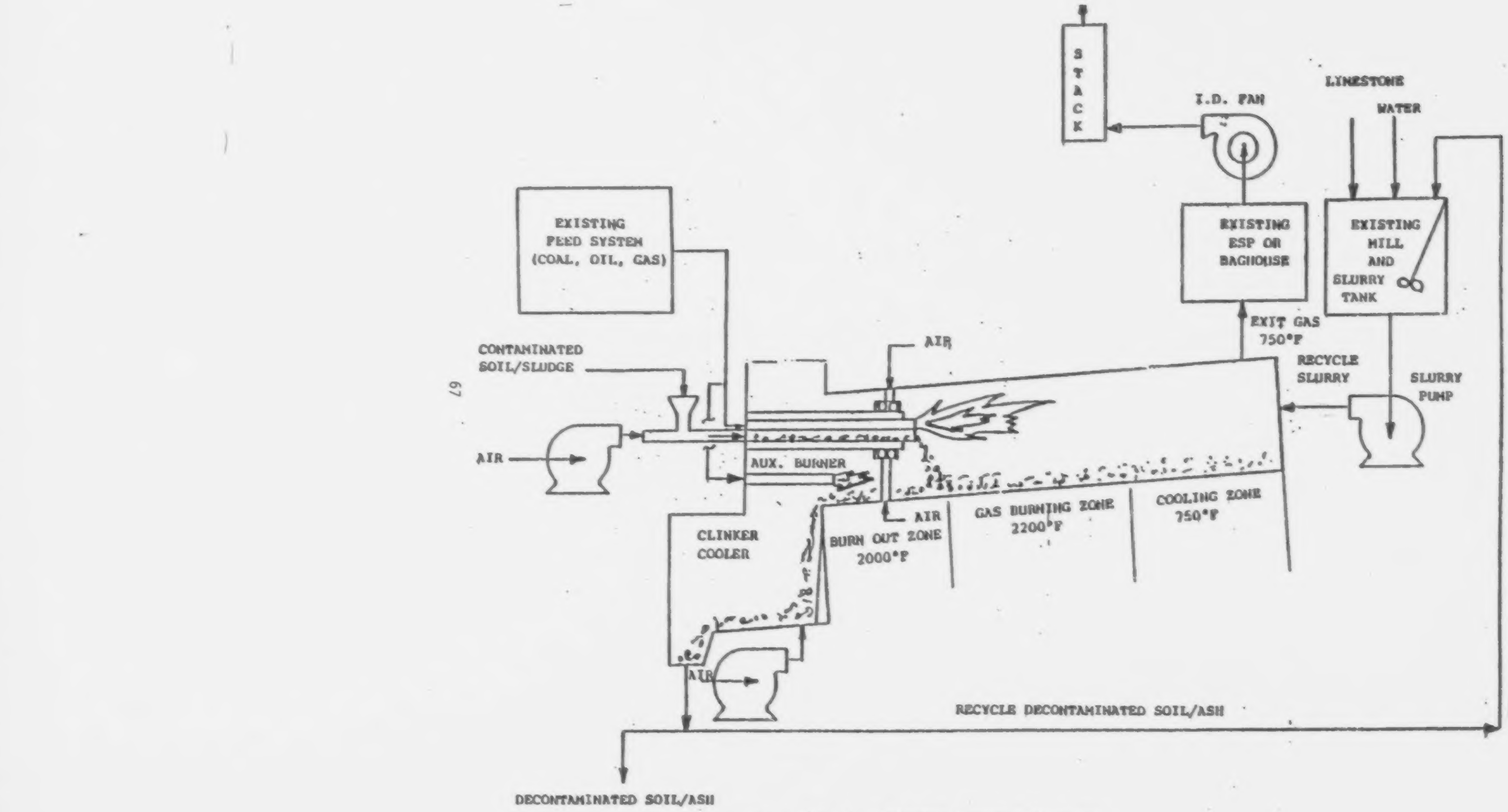
STACK
I.D. FAN
LIMESTONE
WATER
EXISTING FEED SYSTEM (COAL, OIL, GAS)
EXISTING ESP OR BAGHOUSE
EXISTING MILL AND SLURRY TANK
EXIT GAS 750°F
CONTAMINATED SOIL/SLUDGE
AIR
RECYCLE SLURRY
SLURRY PUMP
AIR
AUX. BURNER
AIR
CLINKER COOLER
BURN OUT ZONE 2000°F
GAS BURNING ZONE 2200°F
COOLING ZONE 750°F
AIR
RECYCLE DECONTAMINATED SOIL/ASH
DECONTAMINATED SOIL/ASH

Fig. 2. Existing Cement Kiln System

CHAPTER 5

HEALTH RISK ASSESSMENT

The Division of Health and CDC have been working cooperatively from the early years of work with the dioxin issue and most intensively in the months since July 1982. The primary resource person at CDC has been Dr. Renate Kimbrough. Directly or indirectly, Dr. Kimbrough has been providing the guidance and the scientific aspects of health assessment. A preliminary health risk assessment was provided in December of 1982 to the Missouri Division of Health which concluded that levels greater than 1 ppb in soil pose an unacceptable health risk.

An expert group of consultants has been invited to discuss the theory and process of health risk assessment with CDC and a formal statement will be issued by CDC during the summer of 1983. The final report of the Task Force will have this new material for consideration or inclusion. The Task Force believes that the final health risk assessment methodology should be reserved for that report.

CHAPTER 6

ASSESSMENT OF AVAILABLE TECHNOLOGY

The purpose of this chapter is to provide an in-depth assessment of the various disposal technologies outlined in Chapter 3. Included are recommendations as to which disposal technologies are most likely to be viable and which ones require further study. It must be kept in mind that all of the technologies were described in Chapter 3 strictly on a descriptive basis, with no attempt at relative comparison. This chapter will evaluate those technologies on the basis of applicability to the specific sites, primarily Times Beach and Minker/Stout, and on practicality with respect to degree of development. It should be noted that, at this time, no technology has been demonstrated to destroy dioxin in soil to levels significantly below 1 ppb.

Currently Available Technologies

Following is an assessment of those technologies in which the Task Force considers the process or method of disposal to be available on a commercial scale, and deemed potentially capable of either detoxifying the dioxin contaminated soil, or preventing the release of the contaminant to the environment.

Incineration is considered to be the technology which probably offers a relatively short term permanent solution for dioxin contaminated soil. While data demonstrating destruction to sub-ppb levels is not yet available, it would appear that selection of proper temperatures and residence times would provide a reliable system for detoxification. As with all methods of destruction, it becomes practical only if EPA regulations will permit the treated soil to be treated as non-hazardous waste. Incineration of hazardous waste is common, equipment is commercially available, and controls to ensure environmental safety are well understood. It is recommended that trial burns, in facilities described below, should be made as soon as possible to demonstrate feasibility.

The EPA has at least two facilities which would be capable of performing the required trial burn. One is their mobile incineration system, which would be brought in and set up directly on the chosen site. Several problems exist with this approach, primarily because of the length of time it would take to set up the unit and arrange the necessary permits. Siting requirements such as electricity, water, and fuel would be difficult and expensive to set up. The mobile incineration unit is not recommended as a means of conducting the test burn, nor is it recommended as an end solution to the problem.

The second EPA facility is the newly constructed Pine Bluff incineration system in Arkansas. This incinerator is designated as a research facility, and has recently been granted the necessary permits to operate. It is understood that this unit would be capable of conducting a test burn for dioxin destruction. It is recommended that an attempt be made to obtain the highest priority on the test list for the facility and, once placed on list, all necessary research permits be expedited.

The Task Force believes that information resulting from the test burns, using various soils having different concentrations of contamination, will be useful in determining the degree of dioxin destruction that incineration can provide, and the residual dioxin left in the resultant ash. This data would permit negotiation with EPA as to whether this ash must be landfilled as a hazardous waste, or can be considered a non-hazardous material.

Ultra violet destruction of dioxin has been demonstrated in certain applications on a commercial scale. However, it has also been demonstrated that irradiation time becomes excessive as concentration of dioxin decreases into the ppb range. Therefore, it is not considered to be a viable solution to the problem of dioxin in soil at low concentrations, but may be useful in destroying dioxin in extracts.

Solvent extraction followed by incineration or other methods of destruction remains a possible solution to removal of dioxin from soil. Exhaustive continuous extraction is used in laboratory analysis of dioxin in soil. However, large scale extractions would probably require batch treatment with each stage of extraction removing a fixed fraction of the remaining dioxin, presenting the same problem as UV destruction as concentration of dioxin decreases. It is recommended that further study be done, and this technology is discussed further on page 71.

In place stabilization using grout curtains and organic or inorganic fixatives may be an acceptable final solution for some Missouri sites, but is not recommended for the Times Beach and Minker/Stout sites for the following reasons. In the case of the Minker/Stout site, the geology of the site is poorly suited to this type of containment effort. A layer of highly fractured limestone above sandstone makes complete elimination of groundwater movement through the contaminated area difficult to guarantee. In the case of Times Beach, the contamination is predominantly on and along the roads. But in order to ensure containment of the contaminated soil, the entire town would have to be encircled by the grout curtain, making this an inefficient means of containment. However, the concept of grout curtain containment is in use today, and may be a viable alternative at sites other than Times Beach and Minker/Stout, depending on site specific geology.

Although organic and inorganic fixatives are considered methods of in place stabilization, further study is needed. These methods will be discussed in further detail on page 71.

On-site storage, either in below ground cells or above ground vaults, was evaluated. Below ground cells do not provide an appropriate option. This option would involve on-site excavation and subsequent reburial of the contaminated soil as well as long term monitoring at each of the sites.

Above ground concrete storage vaults have some advantages over below ground storage in that monitoring requirements are more easily met, and any leaks or faults in the structure are more easily detected and repaired. This is not a recommended option, except under special circumstances, because of logistical problems in permitting, monitoring and maintaining 30 or more individual sites. Special circumstances, as mentioned above, could involve the consolidation and storage of contaminated soils from a number of smaller sites at one of the larger, centrally located sites in the area, probably as interim storage in conjunction with a treatment scheme.

The Task Force discussed the possibility of transport to a secure landfill offsite as a remedial action for the Times Beach and Minker/Stout sites. There are existing outstate landfills which could possibly be considered, but landfilling at any of them is not a viable option at this time, due to the large volumes of soil involved, and the distance that this volume of soil would have to be transported. The Task Force wishes to emphasize that landfilling in this manner should only be considered as a last resort for these sites, if all other options for remedial action have failed.

The final option which seemed to be viable was that of in place treatment of the contaminated soil. Most of the technologies grouped into this option are still in the developmental stages, and will be discussed on page 72.

Implementation Considerations

The Task Force believes that in order to implement many of the above mentioned technologies, a centrally-located hazardous waste storage-treatment facility should be considered. A study should be done to resolve where such a facility could be located and whether it should be state owned.

Recommendations for Further Study

Certain of the technologies discussed in Chapter 3 are considered to have merit, but have not been developed to the point where they can be considered viable disposal techniques. Some are functioning at lab scale. Some are operating commercially for wastes other than dioxins, and some have not yet been tested. However, each of these technologies is worth pursuing. Further research and testing should be done on each of the following technologies.

Fixatives

Under the option of in place stabilization, two types of surface fixatives exist: inorganic substances and organic polymers. Further studies on impermeability and long-term stability of these types of fixatives should be conducted.

Solvent Extraction

The concept of solvent extraction is well established and in wide use. However, the feasibility of this process to remove dioxin from contaminated soils to sub-ppb levels has yet to be demonstrated. In an effort to determine if solvent extraction can reduce the level of contamination to environmentally acceptable levels, the following studies are recommended.

An appropriate solvent should be identified. The ideal solvent must have a large solubility for dioxin and a large solvent-soil distribution coefficient, must be easily removable from the soil, and must be non-toxic because a portion of the solvent will probably remain trapped with the soil even after drying. The solvent studies should include supercritical fluids as well as more common solvents.

Additional studies should address the destruction of dioxin in the solvent after the extraction process is completed. Some of the methods which have been proposed are destruction by the addition of a chemical reagent, incineration of the solvent, and destruction of the dioxin using UV photolysis.

In-Place Treatment

The option of in-place treatment is important if a method of decontamination can be developed whereby the contaminated soil would not need to be excavated or moved. In-place treatment offers the advantage of minimal risk to workers and the environment and probably would be the least expensive method. Specific study of the following forms of in-place treatment is recommended.

Biodegradation is already in use on a commercial scale for the detoxification of certain wastes. However, researchers have not yet been able to isolate or genetically engineer organisms which are capable of degrading dioxin. The possibility also exists that an organism may be found which can metabolize dioxin in a controlled environment, in which case the contaminated soil could be excavated and placed in the controlled environment. This research is of the utmost importance and should continue.

Other methods of in-place treatment which merit further study are ionizing radiation, chemical treatment, and any other method which shows possibility of destroying or stabilizing the dioxin molecule.

Recognizing that the methods listed above may not be the only ones which should be considered as possible future options for remedial action, the Task Force welcomes and encourages any new technology which holds the promise of solving Missouri's dioxin problems.

CHAPTER 7

RECOMMENDATIONS FOR FURTHER RESEARCH

This chapter attempts to focus on research needs which are necessary to make informed decisions about remedial action. The two major areas of concern in which data is lacking are health studies and the behavior of dioxin in the environment.

Many animal studies have been done which indicate that widely varying levels of toxicity demonstrated by different animal species. Much debate has been heard on how to extrapolate the animal studies on toxicity to humans. Research on humans has not clearly indicated what level of exposure can cause toxic effects.

Research and accidental exposure of humans has shown definite acute effects of dioxin such as chloracne, liver disease and porphyria cutanea tarda. Birth defects have been reported in some studies but not confirmed in others. Long term effects such as cancer are still debatable, although limited data on soft tissue sarcomas are suggestive. Therefore, continuing research on the human effects of dioxin is recommended.

A related issue is that of the health of residents who have lived at contaminated sites for a number of years. Since many questions remain about the long term health effects that dioxin may have on these people, the Task Force recommends consideration of continued and broadened health studies be conducted on these people.

Further studies on the behavior of dioxin in the environment are needed. Research should be conducted to determine the possibility of dioxin leaching from contaminated soil into groundwater over a period of years. Related work is necessary to determine if dioxin found in aquatic organisms in surface waters near contaminated sites is due to leaching of dioxin directly into surface waters, or to the transport of contaminated soils as sediment into the water. Since the presence of dioxin has been impossible to detect in water, some type of concentrator should be developed, such as a resin or charcoal, or a quantitative bioindicator. Further research into background levels of dioxin in aquatic organisms of surface waters away from the contaminated sites is recommended.

Research should be conducted regarding the persistence of dioxin in the environment, particularly as a contaminant in soil. The use of the term "half life" with respect to dioxin in the environment is erroneous, and a systematic attempt should be made to clarify the mechanisms of dioxin disappearance in the environment.

Possibly the most important topic for additional study is that of the bioavailability of dioxin in soil. Research data available has indicated that dioxin in soil exhibits a binding phenomena, in that the dioxin molecules adhere very tightly to the soil particles. Further research may indicate whether or not the dioxin is so tightly bound to the soil that it is not available to the organism that comes in contact with the soil, whether that contact is dermal or by ingestion.

The Task Force recognizes the need for research and resources to help solve the problems that Missouri faces. This research also will assist in addressing and solving national problems. There already exists in Missouri the necessary expertise and research facilities that could be equipped to perform the necessary studies, if properly funded.

CHAPTER 8

SPECIFIC SITE RECOMMENDATIONS

This chapter deals with recommendations of the Task Force for the Times Beach and Minker/Stout sites, the two sites which have had the highest priority. The Task Force will submit recommendations for other sites in its final report. Certain of the comments to follow are general in nature, and could be applied to both Times Beach and Minker/Stout. These comments will be addressed first. Comments more specific to the individual sites will be discussed in separate sections.

Although one cannot be absolutely sure what actual exposure or risk is experienced by any individual Missouri citizen, the public health recommendations of separating the people from the soil contaminated with greater than 1 ppb appear to be reasonable and prudent measures. It must also be assured that the sites themselves are stable and that no significant movement of either dioxin or contaminated soil is occurring. Actions already taken at the two sites, namely the temporary relocation and buyout, will serve to alleviate one of the main concerns of the Task Force, namely human exposure at the sites. These measures have provided more time to study the remedial actions and to make informed decisions about those remedial actions. It is of prime importance that the buyout be expedited. Once that is accomplished, the Task Force recommends that access to the sites be completely restricted by posting signs, erecting barriers, and any other measures necessary to keep unauthorized persons away from the contaminated soil. This would come under the heading of initial remedial action.

The Task Force recommends a program of additional sampling to determine the nature and extent of the problem. This sampling should include both surface water and ground water monitoring to determine whether the contamination is migrating, either by direct leaching from the soil, or by sediment transport. A concentrator such as charcoal, resin, or a quantitative bioindicator, should be used to detect any low levels of dioxin in the water.

In studying the information available about the two sites, the Task Force concludes that the Minker/Stout site needs priority over Times Beach, at least in implementing initial remedial action, because Minker/Stout is an unstable site with respect to migration of contaminated soils whereas Times Beach appears to be more stable. These concerns are addressed in greater detail in the following sections.

The adequacy of the health studies performed to date should be evaluated.

Minker/Stout

The Task Force recommends that the buyout for Minker/Stout be expedited. Information indicates that this site is unstable with respect to migration of soils, hence the priority status for Minker/Stout. Immediate action should be taken to stop the erosion which is occurring in the fill area and washing across the site into Romaine Creek. Short term measures such as extending the drain pipe out from the culvert and past the fill area, along with covering eroded areas with sod or other natural cover may be sufficient to stop most or all of the erosion of contaminated soil from the fill area. Thereafter, monitoring of surface and groundwater should be done in the site area to determine if erosion of contaminated soil has been eliminated.

While the short term measures outlined above should reduce soil erosion, the site must still be considered an unstable one because of extremely poor site geology, consisting of a layer of highly fractured limestone over sandstone. The Task Force recommends that planning for final remedial action for this site be continued with all deliberate speed.

Times Beach

The buyout should be expedited in order to remove the residents from the site and reduce exposure to the contaminated soil. Access to the site should then be limited to further reduce exposure. Evidence indicates that Times Beach is a relatively stable site. Therefore, no further immediate remedial actions are recommended at this time. The Task Force recommends that additional soil sampling be done to determine more specifically the areas and levels of contamination within the site. Sampling of surface and groundwater should be done to determine if the contamination is spreading due to leaching or sediment transport. If the results of these studies should indicate that significant contaminant migration is occurring, then additional interim remedial action should be undertaken. In any event, the results of the sampling can be used to aid in planning for final remedial action at the site.

APPENDIX 1

AGENDA

DIOXIN TASK FORCE MEETING

February 23, 1983

Conference Room, Department of Natural Resources
1915 Southridge
Jefferson City, Missouri

1. 10:00 Introduction of Task Force Members........Fred Lafser, DNR
Governor's Remarks to Task Force

2. 10:30 Discussion, Scope and Magnitude of Dioxin Problems in Missouri:

(a) Location and Levels of Contamination......Fred Lafser, DNR

(b) Health Effects......Robert Hotchkiss, DOH

12:00 Lunch

3. 1:30 Summary of Solutions Proposed by Interested Companies......DNR Staff

4. 1:45 EPA Role

5. 2:15 Discussion of Task Force Activities and Assignment of Sub-Tasks

6. 3:00 Discussion of Schedule of Meetings

7. 3:15 Adjourn

DIOXIN TASK FORCE SITE TOUR 3-17-83

ITINERARY

8:00 a.m.	Arrive - lobby of Airport Marriott
8:15 a.m.	Leave Marriott
9:00 a.m.	Arrive at Shenadoah Stables
9:30 a.m.	Leave Shenadoah Stables
10:30 a.m.	Arrive at Methodist Church (drive by)
11:00 a.m.	Arrive at Times Beach
11:45 a.m.	Leave Times Beach
12:00-1:30 p.m.	Lunch at Stratford House - Fenton
2:00 p.m.	Bubbling Springs Stables (drive by) (Cashel Site)
2:15 p.m.	Arrive Minker & Stout Sites (Sullens Site)
3:00 p.m.	Leave Minker & Stout Sites
3:30 p.m.	Jones Truck Lines (drive by)
3:40 p.m.	Overnite Transfer, Inc. (drive by)
4:00 p.m.	Southern Cross Lumber (drive by)
4:30 p.m.	Return to Marriott parking lot

AGENDA
(Revised)

DIOXIN TASK FORCE MEETING

March 30, 1983

Conference Room, Department of Natural Resources
1915 Southridge
Jefferson City, Missouri

1. 10:00 Approval of previous meeting minutes....February 23 Meeting

2. 10:15 Explanation of Cattle Deaths on Bill Davis Farm....Dr. Thomas Satalowich, DVM

3. 10:30 Evaluation of Dioxin Disposal Technologies and Costs....CH_2M - Greg Peterson, John W. Lee, Bill Byers

1:00 Lunch

4. 2:00 Discussion of Superfund Procedures - EPA

5. 3:00 Summary of proposals received to date by the Waste Management Program from firms interested in dioxin cleanup....Beth Rice, WMP

6. 5:00 Future Meeting Agendas....April 14 and 28 meetings

7. 5:30 Adjourn

AGENDA
(Revised)
DIOXIN TASK FORCE MEETING

April 13, 1983

Second Floor Conference Room
Division of Health-Laboratory Building
307 West McCarty
Jefferson City, Missouri

1. 3:00 - VIDEO TAPE SERIES

   Tape 1
   "Introductory Discussion of Dioxin Problem in Missouri"
   by Henry Falk, M.D., CDC, Atlanta, Georgia

   Tape 2
   "Animal and Human Health Effects of 2,3,7,8 Tetrachlorodibenzo Dioxin - An Overview"
   by Renate Kimbrough, M.D., CDC, Atlanta, Georgia

2. 5:00 - Dinner

   Conclusion of Video Tape Series (Optional)

April 14, 1983

Department of Natural Resources Conference Room
1915 Southridge Drive
Jefferson City, Missouri

1. Approval of previous meeting minutes....March 30 meeting

2. 8:30 - William G. Dunagin, M.D.
   Division of Dermatology M173
   University of Missouri
   Health Science Center
   Columbia, Missouri 65212

3. 10:00 - Jay Murray, Ph.D.
   Syntex Corporation
   3401 Hillview Avenue
   Palo Alto, California 94304

4. 11:00 - David Stallings, Ph.D.
   Chief Chemist
   Columbia National Fisheries
   Research Lab
   U.S. Fish and Wildlife Service
   Route 1
   Columbia, Missouri 65201

5. 12:00 - Lunch

6. 1:30 - Alvin L. Young, Ph.D.
Major, U.S. Air Force
Acting Chief of Research Section
Agent Orange Project Office
810 Vermont Ave., N.W.
Washington, D.C. 20420

7. 3:00 - Dr. Robert Neal, President
Chemical Industry Institute of Toxicology
Research Triangle Park
Raleigh-Durham, North Carolina 27709

8. 4:00 - Stephen M. Ayres, M.D.
Department of Internal Medicine
St. Louis University Medical School
1325 South Grand Boulevard
St. Louis, Missouri 63104

9. 5:00 - Future Meeting Agendas....April 28 and May 12 Meetings

10. 5:15 - Adjourn

AGENDA

DIOXIN TASK FORCE MEETING

April 28, 1983

Department of Natural Resources Conference Room
1915 Southridge Drive
Jefferson City, Missouri

I. 9:00 a.m. Appoval of previous meeting minutes...March 30 and April 14 meetings

II. 9:15 a.m. Speakers:

(1)	Phillip C. Kearney, Ph.D. Chief, Pesticide Degradation Lab Agricultural Environmental Quality Inst. Agricultural Research Center, USDA Beltsville, Maryland	Fate of dioxin in the environment; plant, fish and animal studies
(2)	Jurgen H. Exner, Ph.D. Manager, Engineering/Research and Development IT Enviroscience Knoxville, Tennessee	Behavior of dioxin in soils-degradation
(3)	Henry Falk, M.D. Chief, Special Studies Branch Chronic Disease Division Center for Environmental Health Centers for Disease Control Atlanta, Georgia	Human health effects of dioxin

III. 12:00 Lunch - catered in by Nick's Homestead

IV. 1:00 p.m. Slide presentation/discussion of southwest sites:
- (1) Neosho site - Dwight Douglas
- (2) Denny Farm site - Ray Forrester
- (3) Spring River - Ron Crunkilton
  Dr. Jim Whitley
  Department of Conservation

V. 2:30 p.m. Discussion with EPA Representative
- (1) New RCRA rule on dioxin
- (2) Coordination with CH2M-Hill feasibility study

VI. 3:00 p.m. Discussion of interim report due June 1, 1983

VII. 5:00 p.m. Future meeting agendas...May 12 and May 26 meetings

VIII. 5:30 p.m. Adjourn

TENTATIVE AGENDA

DIOXIN TASK FORCE MEETING

May 12, 1983

Ramada Inn - Roanoke Room
1510 Jefferson Street
Jefferson City, Missouri

I. 9:00 a.m. Approval of minutes...April 28 meeting

II. 9:15 a.m. Presentations by firms submitting preliminary remedial action proposals

(1).	Midland Ross Corporation 2375 Dorr Street Toledo, Ohio	incineration
(2).	Chemical Waste Management Waste Management, Inc. 3003 Butterfield Road Oak Brook, Illinois	extraction, incineration of solvent, landfilling of soil
(3).	Critical Fluids Systems, Inc. Subsidiary, Arthur D. Little, Inc. 25 Acorn Park Cambridge, Mass.	extraction by supercritical solvent

15 Minute Break

(4).	Acurex Waste Technologies, Inc. Subsidiary, Acurex Corp. 8074 Beechmont Avenue Cincinnati, Ohio	extraction, chemical treatment of solvent
(5).	SCA Chemical Services, Inc. 1836 North Broadway St. Louis, Missouri	SCA doesn't wish to make a presentation at this time, but would like to make themselves available to answer questions about their proposal

12:00 Lunch to be arranged by Ramada Inn

II. 1:00 p.m. (Continued)

(6).	IT Enviroscience 312 Directors Drive Knoxville, Tennessee	thermal destruction; extraction; in situ treatment
(7)	Rollins Environmental Services, Inc. P. O. Box 609 Deer Park, Texas	comparison of incineration, extraction, and landfilling

III. 2:00 p.m. Discussion of Initial Draft Interim Report

3:00 p.m. 15 Minute Break

IV. 5:00 p.m. Adjourn

May 13, 1983

I. 9:00 a.m. Further Discussion of Initial Draft Interim Report

10:30 a.m. 15 Minute break

12:00 p.m. Lunch to be arranged by Ramada Inn

II. 1:00 p.m. Further Discussion of Initial Draft Interim Report

III. 4:00 p.m. Future meeting agendas...May 26 and June 16 meetings

IV. 4:30 p.m. Adjourn

AGENDA

DIOXIN TASK FORCE MEETING

May 26, 1983
Department of Natural Resources
Conference Room
1915 Southridge Drive
Jefferson City, Missouri

I.	9:00 a.m.	Approval of Previous Meeting Minutes...May 12 and 13 Meeting	
II.	9:15 a.m.	Discussion of Final Draft Interim Report	
III.	10:30 a.m.	15 Minute Break	
IV.	11:45 a.m.	Lunch at Nicks Homestead	
V.	1:00 p.m.	Renate Kimbrough, MD. Medical Officer Center of Environmental Health Centers for Disease Control Atlanta, Ga.	Health risk assessments
VI.	2:30 p.m.	Further Discussion of Final Draft Interim Report	
VII.	4:30 p.m.	Discussion of Proposed RCRA Rule on Dioxin	
VIII.	5:00 p.m.	Future Meeting Agendas...June 16 Meeting	
IX.	5:30 p.m.	Adjourn	

APPENDIX II

DIOXIN TASK FORCE MEETING

February 23, 1983

Department of Natural Resources' Conference Room
Jefferson City, Missouri

MEMBERS PRESENT

James Finch, Chairman
Dwight Douglas
Ray Forrester
Perry King
Carl Marienfeld
Sharon Rogers
George Roush
James Shaddy
Betty Wilson

Robert Powell was not present. Claudia Spener represented Dr. Powell.

ORDER OF BUSINESS

The meeting came to order at 10:10 a.m.

Missouri Department of Natural Resources' Director, Fred Lafser, introduced each Dioxin Task Force member. He also introduced Dr. Robert Hotchkiss, Division of Health; Bill Rice, U.S. Environmental Protection Agency; and several DNR staff members.

Governor Christopher S. Bond then welcomed the task force members and thanked them for serving on the task force. Governor Bond presented some background information on what has happened and various steps taken by him since the dioxin issue began last year. The Governor directed the task force to study all angles of the problem and to study any alternatives for eliminating the danger of dioxin and to recommend the most safe, practical and technologically sound solutions. He also presented a five-point dioxin program which he has recommended to the Missouri General Assembly (please refer to the transcript of the Governor's remarks previously sent to you).

Dr. David Bedan, Director of the Waste Management Program, then gave a brief history of the contamination episode. He presented a chart, produced by EPA, that showed the origination of the waste from the old NEPACCO plant in Verona, Missouri and showed how the waste got to the different sites. Dr. Bedan then presented a map of the 22 confirmed sites and gave a brief summary description of each site. He noted there were three errors in the summary description.

1. Minker Site - The range of concentration should be from .3-300 ppb instead of 900 ppb.
2. Times Beach Site - It should be 300 ppb instead of 100 ppb.
3. IML Lines Site - This should be changed to the Overnite Transfer Company.

Dr. Robert Hotchkiss, Director of the Division of Health, then provided a summary of the health effects that dioxin has had on experimental animals and situations of human exposure. He also provided a summary of different studies relating to the cancer-causing potential of human exposure to dioxin. He stated there is a study of the possible health effects associated with the actual real exposure to dioxin here in Missouri that is in the process of being carried out. There have been 146 dermatological exams, 112 comprehensive medical exams, and over 850 extensive medical questionnaires involved in this study.

Dr. Denny Donnell, also of the Division of Health, summarized a talk given by Dr. Marilyn Fingerhut to the physicians at the St. Louis Medical Society which referred to Swedish and American studies concerning questions of the cancer-causing potential of human exposure to dioxin.

Representative Bob Feigenbaum then discussed three bills that have come out of public hearings and meetings held since October. One of the bills is the Superfund which would provide funding in the amount of $6.8 million for an ongoing cleanup measure, not only for the dioxin sites but also for future hazardous waste cleanups. Of the $6.8 million, $5 million would come from generators, $1.5 million from transporters, and the remainder from landfill operators. This Superfund money would only be used when the responsible party could not be found. The second bill is the Cancer Registry which will allow the state to correlate any high levels of different types of cancer to the hazardous waste sites. This information could then be used to convince EPA and federal agencies that there is a problem at a particular site. The third bill is the Workers' Right to Know which doesn't deal just with the dioxin problem.

Joe Jansen of the Waste Management Program then gave a summary of some of the disposal methods and proposals that the department has received from various companies in the waste management business indicating their desire to help with the dioxin problem. Mr. Jansen then presented a chart which gave some very preliminary and very general cost estimates of the different alternatives that the department thinks might work for clean up at the various sites. These were very rough estimates.

Bill Rice, U.S. Environmental Protection Agency, then discussed EPA's role and activities with the dioxin problem. First EPA or the Missouri Department of Natural Resources investigates the sites to determine whether there is a likelihood that dioxin is present. They would send out sampling teams and have the samples analyzed either through their own laboratories or through contracted laboratories. They would then decide from this initial sampling if additional sampling is required. After the problem has been characterized, they can then take legal action against property owners or companies may propose to clean up the site themselves or the sites can be put on the National Priority List.

FUTURE MEETINGS

It was decided that the next meeting would include visits to some of the dioxin sites in the St. Louis area. The task force members are to meet in the lobby of the airport Marriott at 8:00 a.m. on Thursday, March 17. From there they would travel by bus to various dioxin sites in the area. After this trip it would then be decided whether trips to sites in southwestern Missouri would be beneficial.

The following meeting dates were also decided upon:

Wednesday, March 30
Thursday, April 14
Thursday, April 28
Thursday, May 12
Thursday, May 26

These meetings will be held in the Department of Natural Resources' conference room and will begin at 9:00 a.m. and run until 4:00 p.m. with an hour for lunch from 12:00 to 1:00.

OTHER BUSINESS

There was some discussion on task force activities and assignment of subtasks. It was suggested that after the site visits and gathering of some background information on various sites that an attempt be made to group the sites by some criteria and prioritize them for the first report and have various people work on various aspects (landfilling, incineration, health subtask, disposal sub-task). It was suggested that the members think about this and give suggestions on who would be particularly desirable and set up subtasks at the next meeting.

Joe Jansen then distributed expense account forms and explained to the members how to fill out the forms for reimbursement.

Some suggestions were then made on various speakers to hear at future task force meetings. It was decided that the members should write DNR about any suggestions they have and have the department make arrangements for the speakers. The contact person is to be Dr. David Bedan, Department of Natural Resources, Waste Management Program, P.O. Box 1368, Jefferson City, MO 65102 (phone: 314-751-3241). Regarding speakers on health related issues the contact person is to be Dr. Denny Donnell, Department of Social Services, Division of Health, P.O. Box 570, Jefferson City, MO 65102 (phone: 314-751-2713).

The meeting was adjourned at 3:35 p.m.

Respectfully submitted,

Dianne Luebbert
Dianne Luebbert
Secretary

Approved,

James A. Finch Jr
James Finch
Chairman

April 1, 1983
Date

DIOXIN TASK FORCE MEETING

March 30, 1983

Department of Natural Resources' Conference Room
Jefferson City, Missouri

MEMBERS PRESENT

James Finch, Chairman
Dwight Douglas
Ray Forrester
Perry King
Carl Marienfeld
Robert Powell
Sharon Rogers
George Roush
James Shaddy
Betty Wilson

ORDER OF BUSINESS

The meeting was called to order at 10:00 a.m.

There was a correction to the February 23, 1983 minutes. Where the Minker Site and Times Beach Site were listed at the bottom of page 1, ppm should have been ppb. Dwight Douglas then made a motion to approve the minutes with corrections. They were approved.

Dr. Thomas Satalowich, DVM, Missouri Division of Health, discussed the analyses of the cattle deaths on the Bill Davis farm. He stated the milk had been tested for dioxin approximately four times and turned out negative each time. Mr. Davis also had brought cattle that had died on his farm to the lab to be tested, and these tests also turned out negative. The cattle had a form of infectious bovine leukemia that could not be detected as dioxin related.

Mr. Robert Morby, U.S. Environmental Protection Agency, then discussed Superfund procedures. He discussed the three principal types of action: immediate removal, planned removal, and remedial action. He also discussed the difference between Cooperative Agreement and State Contract.

David Bedan then briefly talked about the "Report on Potential and Confirmed Uncontrolled Hazardous Waste Sites in Missouri" that had been released the previous day, March 29.

Dr. Denny Donnell, Missouri Division of Health, discussed the tentative plan for speakers for the April 14, 1983 meeting. The five speakers that are available are:

1. Bill Dunagin - UMC Dermatologist
2. Jay Murray - Syntex, Palo Alto, California
3. Alvin Young - Veteran's Administration, Washington, D.C.
4. Robert Neal - Chemical Industrial Institute of Toxicology, North Carolina
5. Stephen Ayres - Chief of Internal Medicine, St. Louis University Hospital

Some of the other suggested speakers who could not come until a later date or could not come at all are:

1. Samuel Epstein - University of Illinois at Chicago - Is not willing to come.
2. Ellen Silbergeld - Environmental Defense Fund, Washington, D.C. - Would be available on April 27 or 28.
3. Ray Susskind - Institute of Environmental Health, University of Cincinnati - Would not be available until early July.
4. Dr. Kimbrough - CDC - Would not be available for several weeks.

Dr. Donnell suggested the Task Force might want to see the video tapes of presentations to the St. Louis Medical Society, including a talk given by Dr. Marilyn Fingerhut concerning questions of the cancer-causing potential of human exposure to dioxin.

It was then suggested that the Task Force view the video tapes on the afternoon of April 13 starting at 3:00, breaking for dinner around 5:00 or 5:30, and reconvening again in the evening around 7:30 or 8:00 p.m.

Greg Peterson and Bill Byers from CH_2M Hill then gave a presentation on the evaluation of dioxin disposal technologies and costs for the Minker/Stout Site. They discussed the advantages and disadvantages of six different options. The six options are:

1. Securing in place
2. Consolidation on site
3. Securing off site
4. Incineration
5. Extraction
6. Storage until new technology is developed.

Beth Rice, Waste Management Program, then gave a summary of the proposals received to date by the Waste Management Program from firms interested in dioxin cleanup. Preliminary proposals were requested from all the firms that contacted us. At the present time the program has received two proposals.

A sixth person was then added to the list of speakers for April 14. Dr. Carnow of the University of Illinois at Chicago will be speaking for Dr. Epstein. It was decided since there would be a sixth speaker to start the April 14 meeting at 8:30 a.m. with three speakers in the morning and three in the afternoon. There would be an hour for task force discussion at the end of the day.

Some suggestions were made by Dave Bedan and Robert Schreiber for topics for the April 28 meeting:

1. Presentations from companies interested in dioxin cleanup.
2. Presentations from labs involved in testing for dioxin.
3. Presentations from people involved in studies concerning the effects of dioxin on animals
4. Someone from Fish & Wildlife Research Center in Columbia.

There was then some discussion on whether the Task Force should hear from specific companies about their proposals.

Dwight Douglas then questioned whether the Task Force wanted to see the video presentation from the wastewater school at Neosho. It was decided to see these slides and video presentation at the April 28 meeting. The Task Force would then decide whether they should visit the sites in southwestern Missouri, since complaints had been received saying the dioxin problem was being addressed more in the St. Louis area than in the southwestern Missouri area.

The meeting was adjourned at 4:20 p.m.

Respectfully submitted,

Dianne Luebbert

Dianne Luebbert
Secretary

Approved,

James A. Finch

James Finch
Chairman

4/28/83
Date

DIOXIN TASK FORCE MEETING

April 14, 1983

Department of Natural Resources' Conference Room
Jefferson City, Missouri

MEMBERS PRESENT

James Finch, Chairman
Dwight Douglas
Ray Forrester
Perry King
Carl Marienfeld
Robert Powell
Sharon Rogers
George Roush
James Shaddy
Betty Wilson

ORDER OF BUSINESS

The Task Force met the previous afternoon, April 13, 1983, at 3:00 p.m. at the Division of Health Laboratory Building to view three video tapes. One of the tapes was titled "Introductory Discussion of Dioxin Problem in Missouri" by Henry Falk, M.D., CDC, Atlanta, Georgia. The second tape was titled "Animal and Human Health Effects of 2,3,7,8 Tetrachlorodibenzo Dioxin - An Overview" by Renate Kimbrough, M.D., CDC, Atlanta, Georgia. The third tape was titled "Epidemiology of Sarcomas" by Marilyn Fingerhut, Ph.D.

The meeting on April 14 was called to order at 8:40 a.m. at the Department of Natural Resources.

The first speaker was Dr. William G. Dunagin, M.D., Division of Dermatology, University of Missouri Health Science Center, Columbia, Missouri. Dr. Dunagin made the following conclusions:

1. Chloracne is the most sensitive indicator of dioxin exposure in humans.
2. There do not appear to be any cases of chloracne in Missouri.
3. Dermal absorption of dioxin from soil in Missouri is about one million times less than the amount necessary to induce toxicity.
4. In animal studies the dose of dioxin necessary to cause cancer is near or about the same dose which causes direct toxicity.

The next speaker was Dr. Jay Murray, Ph.D., Syntex Corporation, Palo Alto, California. Dr. Murray discussed some of the reproductive and other toxic effects of dioxin seen in laboratory animals. He also summarized some of the human health effects of dioxin including chloracne, liver effects, neuromuscular symptoms, altered porphyrin metabolism, and a number of subjective symptoms. Dr. Murray also discussed a study of an industrial accident that occurred at a Monsanto Plant in 1949 involving overexposure to TCDD and another industrial accident that occurred in Seveso, Italy.

The next speaker was Dr. David Stalling, Ph.D., Chief Chemist, Columbia National Fisheries Research Lab, U.S. Fish and Wildlife Service, Columbia, Missouri. Dr. Stalling summarized some of the work being done in various parts of the country on analysis for dioxin. He also discussed dibenzofurans which are very similar in structure and behavior to dioxins and are often found along with dioxins.

At lunch the Task Force discussed possible agenda items for the future task force meetings.

The next speaker was Dr. Robert Neal, President, Chemical Industry Institute of Toxicology, Research Triangle Park, Raleigh-Durham, North Carolina. Dr. Neal discussed different situations involving human poisoning with TCDD. He also presented a table showing the results of studies in Sweden, Finland, United States and Germany of humans exposed to TCDD. Dr. Neal stated that studies show that TCDD does produce cancer in mice and rats; however, there is no cause and effect relation between TCDD exposure and cancer yet shown in man but this needs to be investigated further.

The next speaker was Dr. Alvin L. Young, Ph.D., Major, U.S. Air Force, Acting Chief of Research Section, Agent Orange Project Office, Washington, D.C. Dr. Young discussed a series of studies conducted at the Eglin Air Force Base in Florida where a herbicide containing TCDD was sprayed. Studies were done on plants at the site and on deer, rabbits, opossums, beach mice, insects, snakes and birds at the site. TCDD was not found in the deer, opossums or rabbits. However, TCDD was found in beach mice that were around the edges of the water site. TCDD was also found in the insects, in the snakes, in the meadowlark birds and in fish at the bottom of the pond on site. Studies are also being conducted on herbicide levels on Johnston Island and at Gulfport, Mississippi. Dr. Young also discussed other studies being conducted including a study in St. Louis, Missouri on identical twins where one twin is a Vietnam veteran and the other twin served in the military but not in Vietnam.

The final speaker was Dr. Stephen M. Ayres, M.D., Department of Internal Medicine, St. Louis University Medical School, St. Louis, Missouri. Dr. Ayres discussed some of the major and minor criteria for the diagnosis of dioxin associated illnesses. He also recommended that the state pass legislation dealing with hazardous waste, particularly Representative Bob Feigenbaum's bill dealing with funding.

Sharon Rogers and Betty Wilson reported they had both received calls from a Mr. Murray Abrahamson who lives across from the Minker site. He expressed concern for the health and welfare of his family.

The meeting was adjourned at approximately 5:00 p.m.

Respectfully submitted,

Dianne Luebbert
Dianne Luebbert
Secretary

Approved,

James A. Finch Jr.
James Finch
Chairman

4/28/83
Date

DIOXIN TASK FORCE MEETING

April 28, 1983

Department of Natural Resources' Conference Room
Jefferson City, Missouri

MEMBERS PRESENT

James Finch, Chairman
Dwight Douglas
Ray Forrester
Perry King
Carl Marienfeld
Robert Powell
Sharon Rogers
George Roush
James Shaddy
Betty Wilson

ORDER OF BUSINESS

The meeting was called to order at 9:10 a.m.

Dwight Douglas moved to approve the March 30, 1983 and the April 14, 1983 minutes as submitted. Robert Powell seconded the motion. Both sets of minutes were approved as submitted.

The first speaker was Dr. Phillip C. Kearney, Chief, Pesticide Degradation Lab, Agricultural Environmental Quality Institute, Agricultural Research Center, USDA, Beltsville, Maryland. Dr. Kearney first discussed different processes which affect pesticides in the environment, but more specifically the molecule TCDD. The different processes discussed were: sunlight or photochemical degradation, chemical degradation, microbiological degradation or microbial metabolism, leaching, adsorption of a pesticide or TCDD to a soil particle, uptake by plants, and volatility. Dr. Kearney then discussed studies that have been done on a terrestrial ecosystem. This is a way of looking at the life history of a molecule such as TCDD and what happens to it. He then discussed bioaccumulation of TCDD in the aquatic environment, bioaccumulation in farm animals and bioaccumulation from oral and dermal exposure to TCDD in rats. Dr. Kearney referred to a paper by Poiger and Schlatter, Swiss Federal Institute at Zurich, which discussed different types of studies done on rats and compared the different levels of accumulation of TCDD in the livers of these animals. Dr. Kearney then discussed his experiences in Seveso, Italy during the summer of 1976. He was asked to devise a decontamination plan for the Seveso area.

The next speaker was Dr. Jurgen H. Exner, Manager, Engineering/Research and Development, IT Enviroscience, Knoxville, Tennessee. Dr. Exner discussed some of the dioxin treatment techniques and the kinds of problems faced in trying to develop a treatment process. He illustrated some of these problems by giving a brief description of their efforts at Verona. Dr. Exner then discussed the chemistry of dioxin in the soil. He also discussed some of the options that exist for treating dioxin in the soil (e.g. extraction, incineration, etc.).

The next speaker was Dr. Henry Falk, Chief, Special Studies Branch, Chronic Disease Division, Center for Environmental Health, Centers for Disease Control, Atlanta, Georgia. Dr. Falk discussed CDC's roles in the dioxin episode. CDC has been working with EPA in terms of evaluation and interpretation of EPA environmental data as it becomes available and also working with them on the quality assurance aspects of the environmental data. CDC has also worked closely with the Missouri Division of Health in evaluating situations and making public health decisions. Three other points that Dr. Falk also discussed were:

1. The extreme potency of dioxin in a variety of animal species.
2. There is a variability within species and there is a variability between the dose that will cause an acute effect, the dose that will cause a chronic effect, and the dose that one would consider safe.
3. There is a body of knowledge about the effects of dioxin in people that comes mostly from occupational settings.

Dr. Falk also outlined the risk assessment process and the analysis that led to the selection of the 1 ppb level of dioxin in the soil as a level which could be used publically in terms of making decisions about different sites.

Dwight Douglas then gave a slide presentation on some of the dioxin sites in southwest Missouri. He briefly explained the geography of the area including the Neosho site, the community of Verona and the Spring River. The three locations of the contaminated sites are: (1) the digester at the sewage treatment plant, (2) the Water and Wastewater Technical School site, and (3) the Bunker site.

Ray Forrester then provided a 17-minute video tape titled "Toxic Cleanup" that gave a brief synopsis of the Denney Farm Site action which was conducted by Syntex under the supervision of EPA.

Dr. Jim Whitley, Chief of Fisheries Research, Missouri Department of Conservation, briefly discussed studies done in the 1950's and 1960's on aquatic life in the Spring River. The Department of Conservation has also recently collected fish from the Spring River and sampled them for dioxin. Ron Crunkilton, Water Quality Biologist, then discussed the economic importance of the Spring River in southwest Missouri. He stated the river contains one of the most diverse fish populations in the state. He also stated that the Food and Drug Administration advisory levels on dioxin have been set. Mr. Crunkilton discussed averages and ranges of dioxin found in different kinds of fish in the Spring River. As a result of some of these values, the State of Missouri has issued an advisory for people to limit their consumption of fish from the entire Spring River.

There was then some brief discussion about the upcoming meetings in May. It was decided that there would be a two-day session on May 12 and 13. The second day would be for the purpose of working on the interim report due June 1. It was also decided to change the May 26 meeting to May 23 if Dr. Renate Kimbrough would be able to speak on the 23rd.

Robert Morby, U.S. Environmental Protection Agency, then discussed a new rule proposed by EPA that was published in the Federal Register on April 4, 1983. It was EPA's intent to provide a way to regulate dioxin as it would be defined as a hazardous waste. As proposed the rule would prohibit interim status facilities from managing dioxins until they are fully permitted. Three exemptions are: (1) wastewater treatment sludges that were generated in surface impoundments that contained dioxin, (2) tank and container facilities, and (3) wastepiles that were enclosed in some type of facility. This proposed rule would regulate wastes generated from specific processes that could contain dioxins. The term "generate" would also apply to wastes that were moved, picked up or moved about which would also be applicable to the dioxin situation in Missouri. This proposed rule would also have jurisdiction over the permitting requirements for the management of dioxin contaminated wastes. This proposed rule is open for comments until June 3, 1983.

Dick Smith, U.S. Environmental Protection Agency, then discussed the procedures that will be used in selecting a remedial action at a hazardous waste site that is on EPA's National Priority List. He also discussed requirements under the National Contingency Plan which spells out the requirements for feasibility studies.

There was then some discussion among the task force about meeting dates for the months of June, July and August. The meeting dates of June 16 and July 21 were set. There was also some discussion on the May 12 agenda.

Ron Kucera then announced that Congressman Volkmer's office had invited the department to testify before its Science and Technology Subcommittee in Washington. He planned to tell them that: (1) R & D monies in the EPA budget should not be cut at this time, and (2) there should be an extension of time on the Superfund.

There was then some discussion on what should be included in the interim report due June 1. It was decided that the program staff would prepare a draft report up to Chapter 4. The task force members are to bring any ideas or conclusions they would like to see in the interim report to the next meeting. The members are also to send any comments or suggestions they have on the proposed regulation to Dave Bedan before the May 12 meeting.

Respectfully submitted,

Dianne Luebbert
Dianne Luebbert
Secretary

Approved,

James A. Finch Jr
James Finch
Chairman

5/13/83
Date

DIOXIN TASK FORCE MEETING

May 12, 1983

Ramada Inn, Roanoke Room
Jefferson City, Missouri

MEMBERS PRESENT

James Finch, Chairman
Dwight Douglas
Ray Forrester
Perry King
Carl Marienfeld
Robert Powell
Sharon Rogers
George Roush
James Shaddy
Betty Wilson

ORDER OF BUSINESS

The meeting on Thursday, May 12, 1983, was called to order at 9:20 a.m.

The minutes from the April 28, 1983 task force meeting were approved as submitted.

Beth Rice, Waste Management Program, Department of Natural Resources, stated that the first company on the agenda, Midland Ross Corporation, did not wish to make a presentation. She passed out a statement from Irving Williams that stated the reasons why they did not wish to make a presentation.

The next presentation was by George VanderVelde, Technical Director for Chemical Waste Management, and Darrell Schimeck, a project administrator for Superfund activities, also of Chemical Waste Management. Mr. Schimeck stated there were nine key elements or factors they evaluated in determining a successful project. They are:

1. Public health and safety during operations and in resulting solution
2. Technically feasible
3. Operationally sound
4. Provides a solution that is environmentally sound and defensible
5. Compliance with regulations
6. Timing
7. Cost
8. Public reaction/opinion/perception in Missouri
9. Public reaction/opinion/perception in other states.

Chemical Waste Management then evaluated four potential treatment or disposal options in terms of these nine factors, breaking each down in positives and negatives and then made a recommendation for each option. The first option, incineration of dioxin contaminated soil, was recommended as a good option. The second option, secure burial, was recommended as a poor option. The third option, treatment of the dioxin contaminated soil by either physically or chemically neutralizing the dioxin contaminants, was recommended as a good option. The fourth option, site capping/encapsulation, was recommended as a good option.

Chemical Waste Management made the following conclusions:

1. Due to the volume of soil, destruction of dioxin with the soil is cost prohibitive.
2. Since dioxin is a major concern, dioxin should be extracted from the soil in order to again reduce the volume.
3. The dioxin which is excavated or extracted should be destroyed chemically and/or incinerated.
4. Again due to the volume of low level dioxin contaminated soil, removal and destruction of the dioxin with the soil is cost prohibitive.
5. Site capping and encapsulation should be considered as an alternative solution for select low level contaminated sites.
6. Because of public reaction and sensitivity by other states to receiving dioxin, as much of the dioxin processing and disposal should be done within the State of Missouri as is possible.

Considering those conclusions Chemical Waste Management's recommendations for primary operation are as follows:

1. Excavation and recapping of high level dioxin sites.
2. Concurrent capping and encapsulation of the low level dioxin sites.
3. Extraction of the dioxin from the excavated soils.
4. Disposal via a secure burial of soil following the dioxin extraction.
5. Treatment/destruction of dioxin contaminated soil extract medium via the ultraviolet light process.
6. Ocean incineration of the treated soil extract medium.

Based upon the current level of information, Chemical Waste Management estimated the total cost to be between $22-$65 million and the total time of the project completion to be 14-24 months.

George VanderVelde then discussed a proposal to build a facility within the state that would involve chemical separation. The process would remove the dioxin from the soil, concentrate that dioxin into a liquid medium which could then be reduced in terms of dioxin that is present through the means of a UV process. The UV process would reduce the level of dioxin. The liquid material which contains the bulk of the dioxin would be incinerated and the very low level material left in the soil would then be buried in a secure landfill.

The next presentation was by Tom Cody, Vice-President of Critical Fluids Systems, Inc. Mr. Cody discussed Critical Fluids Systems' technology which is based on the use of condensed gases as extracting solvents. The technology has never been applied specifically to dioxin, but has been successful in extracting other chemicals and materials from the soil. Critical Fluids Systems proposes that a small-scale test program be done to determine the feasibility of utilizing critical fluid extraction technology. After the test program, a recommendation would be made on the feasibility of proceeding with subsequent phases leading to construction of a full-scale processing plant to be operated by a waste management company. The cost of the proposal would be as follows:

Set-Up (6 months)	$ 350,000
Run	500,000
Pre-Design Engineering Package (3 months)	50,000
Extraction Plant (process 3.5-7.0 tons/hour)	3,000,000

The next presentation was by Tony Schraub, Mechanical Engineer, and Leo Weitzman, Chief Scientist, from Acurex Waste Technologies, Inc. Mr. Schraub discussed their process which destroys dioxin by chemical transformation of the dioxin molecule and produces a safe polymer. The Acurex dioxin soil cleanup process would involve the following steps:

1. Move portable equipment to contaminated site
2. Preparing the soil (wetting it thoroughly so that dust is eliminated or reduced)
3. Scoop dioxin soil directly into soil washing machines
4. Wash soil with agitation in special Acurex solvent
5. Remove solvent from soil by vacuum extraction
6. Return safe soil to original location
7. Purify solvent and concentrate pollutant into small-volume sludge
8. Treat sludge with Acurex dioxin and PCB destruction process rendering harmless
9. Remove equipment and sludge, leaving clean site
10. Regreen site as appropriate.

Some of the benefits of the Acurex soil cleanup solution are: cleans dioxin or PCB from soil to specified level; destroys the dioxins; safe (nonflammable, etc.); process already demonstrated in Missouri; no transport of contaminated soil from site; controllable and verifiable; creates no secondary pollutants or migration; and returns the site to an environmentally safe and clean condition. The preliminary cost, assuming 10,000 cubic yard cleanup at a site which is relatively accessible, would be $200-$500 per cubic yard.

The next presentation was by John Schofield and Jurgen Exner of IT Enviroscience. Mr. Schofield discussed two solutions for destroying dioxin: (1) destruction by thermal oxidation or incineration and (2) destruction by a chemical means. Dioxin could be destroyed by a chemical process such as the ultraviolet process. This would be a potential solution for a site that is lightly contaminated with dioxin that is fairly close to the surface. The cost for such an in situ chemical process would be approximately $50 per ton. The other solution would be destruction of the material by incineration in a cement kiln. Cement kilns could handle thousands of tons per day. Two things would be required: (1) to prove to the public that incineration can destroy dioxin (this demonstration could be done by using the EPA mobile incinerator) and (2) carry out engineering feasibility of converting a cement kiln to a soil incinerator. This process would cost approximately $200 per ton.

The next presentation was by James L. Boyland, Director of Business Development for the Midwest Region for SCA Services. Mr. Boyland stated that SCA Services believes incineration is the best proven technology for destruction of dioxin and that a cement kiln would offer the best solution. They have not estimated any costs as of yet. Mr. Boyland also discussed a team that SCA Services had formed to look into the problem. Battelle Columbus Laboratories is on this team to assess, to evalute and to recommend technologies. Fru-Con Corporation is also on this team as specialists in the design and construction of any kinds of facilities that would be anticipated. SCA Services would be the prime contractor and operator for the project.

The next presentation was by Ed Hillier, Field Services Group of Rollins Environmental Services, Inc. and Don Matter, Field Services Manager. Mr. Hillier proposed that off-site incineration may be feasible for small amounts of dioxin, assuming you would want to move the material as fast as possible. Rollins Environmental recommends above ground storage of the material until new technlogies are developed and proved to be effective. The estimated cost for off site incineration for 500,000 cubic yards would be $234,000,000 which includes removal and transportation. The time period would be approximately 5600 days. The estimated cost for above ground storage for 500,000 cubic yards would be $26,600,000 which includes excavating, transportation, construction and waste placement. Don Matter discussed the option of incineration. He stated that the ash from incineration would still have to be made secure in a licensed permitted facility.

Ron Kucera, Department of Natural Resources, informed the task force of the results of his trip to Washington, D.C. and his testimony before the Science and Technology Committee that was requested by Congressman Volkmer. He indicated to the committee that Missouri would benefit by an extension of the Superfund Act and that Missouri would like to have some additional R & D monies for research needs.

Greg Peterson, CH2M Hill, the firm contracted to do the Minker/Stout feasibility study, provided additional information on temporary stabilization of the material at the Minker/Stout site until new technology is developed. The firm looked at approximately 20 alternatives, including a polyurethane spray foam. The cost for this, including monitoring wells, would be between $1-$2.5 million.

The task force then brought up the question to Dick Smith, U.S. Environmental Protection Agency, that if a site on the National Priority List did some type of interim action such as temporary cover, if that would cause the site to be removed from the list since there would no longer be an immediate risk?

Dave Bedan, Waste Management Program, Department of Natural Resources, then went over the different chapters in the draft of the interim report. The task force then discussed whether Chapter 5, The Theory of and Procedures for Health Risks Assessment, should be included in the interim report. It was decided that the title of Chapter 5 should be changed and should include what the Division of Health has been doing and what their interaction with CDC has been. It was also decided that Chapter 6 would be discussed in further detail at Friday's meeting and if time permitted, there would be discussion on proposed revisions to EPA's new regulation, otherwise this item would be put on the May 26 agenda. A suggestion was also made that there should be a statement in Chapter 6 making it perfectly clear that the recommendations were site specific and are not recommendations for other sites which have not yet been adequately addressed.

The meeting was adjourned at approximately 5:00 p.m.

Respectfully submitted,

Dianne Luebbert
Dianne Luebbert
Secretary

Approved,

James G. Finch
James Finch
Chairman

5/26/83
Date

DIOXIN TASK FORCE MEETING

May 13, 1983

Ramada Inn, Roanoke Room
Jefferson City, Missouri

MEMBERS PRESENT

James Finch, Chairman
Dwight Douglas
Ray Forrester
Perry King
Carl Marienfeld
Robert Powell
Sharon Rogers
George Roush
James Shaddy
Betty Wilson

ORDER OF BUSINESS

The meeting on Friday, May 13, 1983, was called to order at 8:10 a.m.

The task force discussed several recommendations and suggestions for the interim report due on June 1, 1983. These suggestions and recommendations were as follows:

1. The report should address only the Minker/Stout and Times Beach sites. The remaining dioxin sites should be listed in the appendix with a statement that they are being included for information only.
2. On page 3 of the "Historical Summary" (3rd paragraph), there should be a note to refer to the body of the report for further clarification of half-life.
3. Would like to see additional sampling at various points in Times Beach to determine levels of contamination and to determine the extent of the problem.
4. Would like to see surface water and ground water sampling by use of bio-indicators and activated carbons and resins to determine if contamination exists and then to determine if that contamination is a result of leaching or sediment transport.
5. The report should outline the effects of the buyout at Times Beach. It should state that actions already taken have alleviated the problem and have removed the immediate risk.

6. The report needs to state that interim stabilization of the soil at the Minker/Stout site needs to be done. For instance attach a pipe to the drain pipe that runs under the road and run that pipe down off the hill. The report then should state that remedial work needs to be done at both the Minker/Stout and Times Beach sites.

7. Should assign priorities in the report. Minker/Stout should be given first priority and state the reasons why it should be first. Minker/Stout is an unstable site, whereas Times Beach is more stable.

8. Specific conclusions and recommendations for Times Beach:

   a. Buyout should be expedited. Should include removal of people. Access should be limited.

   b. With reduced exposure there is no need for further immediate action. Testing will continue.

   c. Health risks:

      (1) Has been reduced because of removal of exposure.

      (2) Study of health effects to residents should be broadened and continued.

      (3) Will reassess health effects.

   d. Initial remedial action - Taking measures to limit access. When state obtains title, then can totally restrict access.

   e. Planning for final remedial action should be continued based upon further study.

9. Specific conclusions and recommendations for Minker/Stout:

   a. Buyout should be expedited.

   b. Immediate remedial action is called for. Short term action is in order such as culvert, sod, and grass to stop erosion.

   c. Planning for final remedial action should be continued. Should have statement about geology at the site. Should proceed with all deliberate speed.

10. Should select three or four of the options for remedial action the task force heard about and place emphasis on them.

11. State or federal government should consider a centralized location for a treatment facility or storage area.

12. Small sites should not use portable equipment but should take material to state or federally owned area.

13. Monitoring should be done at Minker/Stout site after drain pipe installed and other initial action completed.

14. Soil from one of the sites should be sent to the Pine Bluff, Arkansas incinerator for a test burn.

The task force then discussed the different options for remedial action. A summary of the discussion for each option is given below:

Stabilization In Place - A grout curtain is not a recommended action for the Minker/Stout or Times Beach sites, but may be used for other sites. Discuss further under further studies. Also recommended long-time research into organic polymers as fixatives.

On-Site Storage - Do not recommend on-site storage at each and every site. Portable equipment would be burdensome to move, would cost a large amount of money, would require individual permits, and would have to set up such things as generators, water, power, etc. Recommend one centrally located facility and bring all the material to that facility to store, treat, etc. Recommend a preliminary study that would look into possible sites where a facility and storage space could be constructed. In most cases on site storage would appear not to be feasible, but there may be some special situations where it could be used.

Transport to Secure Landfill - Do not recommend as an option to pursue unless all other high technology fails.

Biodegradation - Recommend further research.

Incineration - Recommend a pilot burn at Pine Bluff to see if ash residue will be acceptable for non-hazardous disposal. Will have to determine if the dioxin is destroyed or if it is sent into the atmosphere. Request priority treatment to be placed on test list. Request research permit so this test can be done. Suggested someone might be able to visit Pine Bluff.

Extraction - Recommended further study in the field of extraction. Make a distinction that this is not an end solution.

In-Place Treatment - Recommended further research. This is not an immediate available solution. Photolysis process is not an end solution to problem. Recommended further research on ultraviolet radiation, gamma radiation, and lasers.

Temporary Storage or Stabilization Until New Technology Develops - Do not recommend as an option.

A summary list of those recommendations for further research are: organic polymers, incineration to a certain extent, extraction, biodegradation, in-place treatment such as ultraviolet radiation, gamma radiation, lasers, etc. and bioavailability.

Dick Smith, U.S. Environmental Protection Agency, also discussed a study that will begin in the near future by the Division of Geology and Land Survey on Romaine Creek. They will be looking at the sediment transport issue and will be determining general ground water movements in the Minker/Stout area. DGLS will be installing stream gauges and will be injecting dye at various points in the stream basins. They will also be surveying wells in the area.

Dick Smith also addressed a question from the previous day about whether a site that is on the National Priority List and has completed interim action that has reduced a risk, whether that site would be taken off the list because of the interim action. Mr. Smith stated that once a site has received a hazard ranking score sufficient to put it on the National Priority List, that site is not re-scored at any point. It remains on the list until final action is completed.

It was also decided that the next meeting would remain on May 26 instead of May 23. There had been discussion at the previous meeting to change the date to May 23.

James Shaddy suggested that since the task force had mentioned several topics that need further study or further research, that they could possibly solicit research needs from the University of Missouri - Columbia coordinator.

The meeting was adjourned at 2:40 p.m.

Respectfully submitted,

Dianne Luebbert
Dianne Luebbert
Secretary

Approved,

James A. Finch
James Finch
Chairman

5/26/83
Date

APPENDIX III

LIST OF REFERENCES

The following is a partial list of the multitude of documents available on the topic of dioxin.

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APPENDIX IV

PROCEDURES FOR SELECTING A REMEDIAL ACTION

Background

Section 104(a)(1) of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) authorizes the President to provide remedial actions which are necessary to protect the public health, welfare, or the environment at waste sites. The National Contingency Plan (NCP) provides the regulations necessary to implement CERCLA and outlines the basic procedures for selecting a final remedial action. Pursuant to a delegation of authority, the EPA Assistant Administrator for Solid Waste and Emergency Response is empowered to approve the selection of remedies for hazardous waste sites. The statutory and regulatory requirements for documenting the remedy selection are discussed in the following sections.

CERCLA Requirements

The selection of the final remedial action occurs after the completion of the feasibility study and is subject to fundamental statutory requirements contained in CERCLA. The key declarations that are necessary for every remedial action recommendation are:

1. EPA has consulted with the State before determining the appropriate remedial action [Section 104(c)(2)];

2. The action being taken is (a) a cost-effective solution and (b) appropriate when balanced against the need to use Trust Fund money at other sites [Section 104(c)(4)];

3. The proposed remedy effectively mitigates and minimizes damage to and provides adequate protection of public health, welfare and the environment; and

4. When the selected action involves off-site disposal, these additional declarations are, also, satisfied [Section 101(24)]:

   a. The action is the most cost-effective when compared to other possible remedial actions;
   b. The action will create new capacity to manage, in compliance with the Solid Waste Disposal Act, hazardous substances in addition to those located at the site in question; and
   c. The action is necessary to protect the public health, welfare, or the environment from a present or potential risk which may be created by further exposure to the continued presence of such substances.

NCP Requirements

The NCP regulations outline certain procedures for identifying, evaluating and selecting a remedial action which provides a permanent remedy to prevent or mitigate the migration of a release of hazardous substances into the environment. These regulatory procedures are based upon the statutory requirements contained in CERCLA and are summarized below.

1. Development of Alternatives - A limited number of alternatives is developed for either (1) source control actions where wastes remain at or near the area where they were originally located; (2) off-site remedial actions when wastes have migrated off-site; or (3) both types of actions.

2. Initial Screening of Alternatives - The alternatives developed are subject to an initial screening to narrow the list of potential remedial actions for further detailed analysis. The alternatives are evaluated according to these factors: (1) costs; (2) environmental effects and overall effectiveness; and (3) acceptable engineering practices.

3. Detailed Analysis of Alternatives - A more detailed evaluation is conducted of the limited number of alternatives that remain after the initial screening.

4. Selection of Remedial Alternative - The lead agency (either EPA or the State) selects the remedial alternative which the agency determines is cost effective (i.e., the lowest cost alternative that is technologically feasible and reliable and which effectively mitigates and minimizes damage to and provides adequate protection of public health, welfare or the environment).

Development of the Record of Decision (ROD)

The mechanism that has been developed to document the remedial action selected is the ROD. This decision document is prepared by EPA with assistance from the State whether EPA is the lead agency or not. The ROD, also, insures that the selected remedy is based upon appropriate data and is in accordance with the CERCLA requirements and Superfund policy.

The following activities provide an outline of the review and approval process necessary to select and implement a remedial action. Deviations from this outline of activities may occur depending on the nature and complexity of the site. Also, the length of time necessary to accomplish these tasks will depend on these same factors.

1. The draft feasibility study is submitted for review.

2. The EPA Regional Office reviews the draft feasibility study for compliance with the NCP.

3. The Regional Office confers with the State on the results of the feasibility study to ensure that the State concerns are adequately addressed in recommending appropriate remedial actions.

4. The Regional Office prepares a draft ROD to obtain approval to initiate remedial actions.

5. The State assists the Regional Office in developing information to support the ROD recommendation and submits a letter indicating the State's support of the selected remedial action.

6. The Regional Office provides a three (3) week public comment period regarding the draft feasibility study and draft ROD followed by a public meeting to receive citizen comments.

7. The public comments are incorporated into the draft feasibility study and draft ROD and the Regional Office prepares a responsiveness summary outlining the public comments and providing EPA's responses to them.

8. The draft ROD and supporting documentation are submitted to EPA Headquarters for review.

9. The Regional Office in cooperation with the State then develops the final ROD after incorporation of the Headquarters' comments.

10. The Regional Office submits the final ROD to Headquarters for approval.

11. The Assistant Administrator for Solid Waste and Emergency Response approves the ROD after balancing the need for the remedial action at this site against the amount of money available in the Fund to respond to other sites.

12. Following approval of the ROD, funds for the remedial action will be obligated and the activity will be implemented through a Cooperative Agreement or a new or amended State Superfund Contract which provides all required State assurances, including assurance of the availability of an acceptable hazardous waste disposal facility.

April 1983
Superfund Section
U.S. Environmental Protection Agency

APPENDIX V

TESTIMONY OF
MISSOURI DEPARTMENT OF NATURAL RESOURCES
BEFORE COMMITTEE ON SCIENCE AND TECHNOLOGY
(U.S. CONGRESS)

May 4, 1983

Mr. Chairman, Members of the Committee.

My name is Ron Kucera. I would like to extend my thanks to the committee for the opportunity to appear here today to discuss the subject of alternatives to the landfilling of hazardous waste. I serve as deputy director for the Missouri Department of Natural Resources. Our Department has responsibility for parks management, historic preservation, geologic resources, energy, and environmental quality. Without a doubt the most difficult issue facing our agency has been the management of hazardous waste including wastes currently generated as well as those which did not receive proper or adequate disposal in the past.

I am proud to say that it is the policy of the State of Missouri, enunciated in statute, to discourage the landfilling of hazardous waste. In 1980 the Missouri General Assembly passed amendments to the State's hazardous waste law enacting more stringent standards for the permitting of landfills, placing a tax on the generation of hazardous waste, and requiring the use of practical alternatives to landfilling. The injection or landfilling of bulk liquid hazardous waste is entirely prohibited. Missouri is a leader among the states in reducing the dependence on landfilling for the management of hazardous waste. Currently, less than 5% of wastes generated in Missouri are destined for disposal by landfilling. We are now vigorously pursuing further amendments to our waste law which will place a very substantial tonnage tax on all hazardous waste that is disposed of through landfilling. We are confident that this new law will provide strong additional incentives to Missouri industries to invest in process changes, recycling, treatment, and incineration. The revenues from this new tax will be targeted to the correction of problems caused by poor waste management practices of the past.

As the members of this committee are most certainly aware, the public policy concerns over the use of landfills are not restricted to the matter of current and future waste generation. The clean-up of old inadequate or leaking waste sites often yields large volumes of waste that must be managed by legally approved hazardous waste facilities. If, as is frequently the case, large quantities of contaminated soil are involved, landfilling usually is thought to be the least-cost option when compared to treatment or incineration. Though we do not yet have good cost estimates, this relationship appears to hold for the remedial action alternatives available for many of Missouri's dioxin sites.

I would like to take just a minute to acquaint the committee with the numbing reality of the dioxin problem that we are facing in Missouri. It is a dilemma of almost unfathomable proportions, and there is no tonic cure, no easy solution. As of this week our Department's investigators working cooperatively with the staff of the Kansas City regional EPA office had confirmed through quality-assured sampling and analysis the existence of 30 separate dioxin sites in Missouri. Our investigators continue to find additional leads, and if the current trends persist, we will no doubt confirm more contaminated sites.

It is estimated that the total volume of contaminated soil with greater than one part per billion 2,3,7,8 tetrachlorodibenzo-p-dioxin could exceed 400,000 cubic yards or 500,000 tons. The oft-suggested solution of high temperature incineration could easily cost more than $2,000 per ton or more than $1 billion for all the dioxin contaminated material known to date in Missouri. Another alternative, solvent extraction with subsequent incineration or ultraviolet photolytic degradation would most likely involve even higher costs. Furthermore, investigations may reveal similar though perhaps smaller dioxin problems in Arkansas, California, Illinois, Kansas, Michigan, New Jersey, New York, Ohio, Oregon, and Pennsylvania. Obviously, the large potential costs for incineration and solvent extraction technologies could exceed the capability of the federal Superfund.

If we must decide between the less costly options of landfilling and some hybrid of on-site stabilization and land treatment, then it is abundantly clear that we must know more about the fate of dioxin in soil as a function of time. Unfortunately, we have found that there are crucial information deficiencies. The basic research that would allow us to make confident predictions about the behavior and half-life of dioxin in Missouri soils has not yet been initiated. Fundamental questions about the naturally occurring reduction of dioxin concentrations in soil through photolysis, biotic activity, volatilization, and solution have not been answered with adequate precision. Amazingly, after all the years of debate, we do not yet have a good handle on the half-life of dioxin in soil, nor do we know which degradation process predominates. Hopefully, through research we may learn that there are effective methods for enhancing and accelerating natural dioxin degradation in on-site applications. It should be noted that natural degradation processes might be severely reduced in the environment of a landfill thereby guaranteeing lengthy persistence of the dioxin.

It is vital to our efforts in Missouri that the research and development program within EPA provide accelerated funding to dioxin-related research activities. This is definitely not the appropriate time to diminish the EPA budgetary commitment to research. We cannot afford to base multimillion dollar remedial action decisions on nickel research.

APPENDIX VI

CONFIRMED DIOXIN SITES

Barry County

Denney Farm

Callaway County

Timberline Stables

Franklin County

Quail Run Mobile Home Park

Greene County

Syntex Facility - Springfield

Jefferson County

Bubbling Springs Ranch
Cashel Residence
Minker Residence
Romaine Creek
Ruth Sullins Property
Stout Residence
Saddle and Spur Club
Sandcut Road

Lawrence County

Bill Ray Farm
Erwin Farm
Rusha Farm
Syntex Facility
Spring River

Lincoln County

Shenandoah Stables

Newton County

Neosho Digester, Trench Tank, Spill Area, and Wastewater School

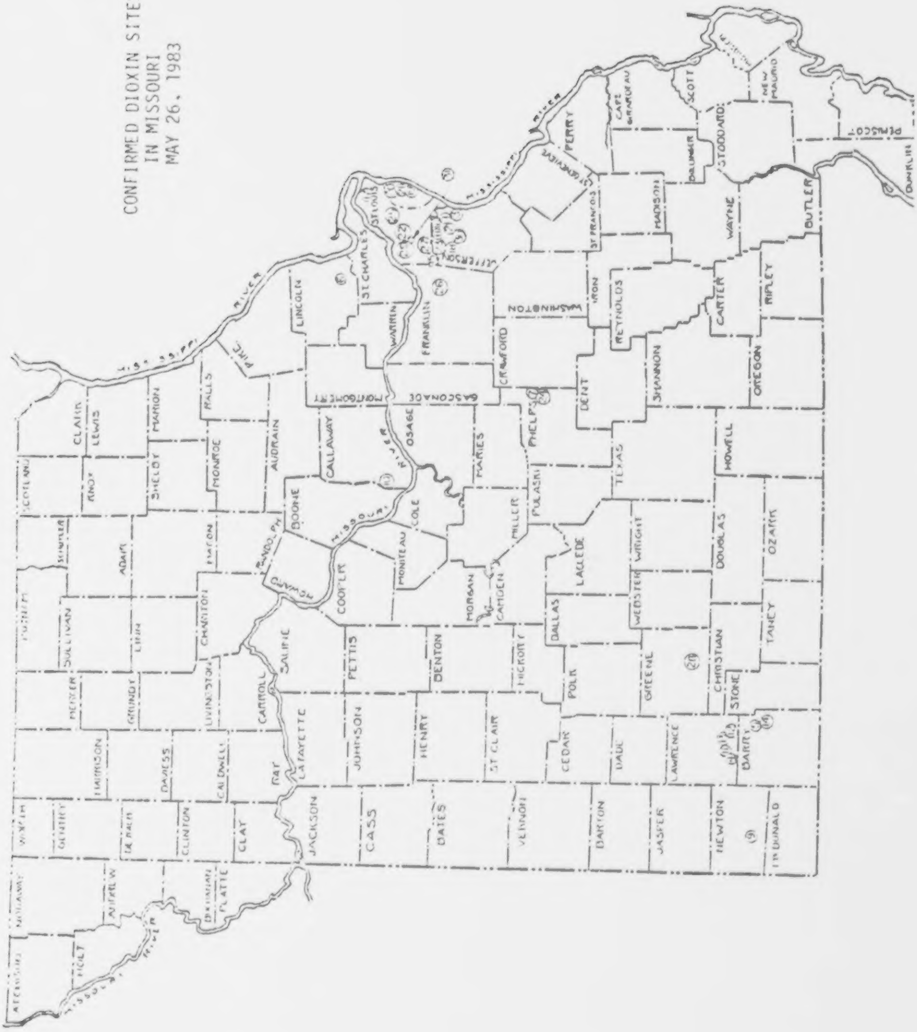
Phelps County

Bliss Farm Road
Walls Property and Piazza Road

St. Louis County

- Baxter Garden Center
- Hamill Transfer Company
- Jones Truck Line
- Methodist Church
- Mid-America Arena
- Overnite Transfer, Inc.
- Russell Bliss Oil Company (Frontenac)
- Sontag Road
- Southern Cross Lumber Company
- Times Beach

CONFIRMED DIOXIN SITES
IN MISSOURI
MAY 26, 1983


ATCHISON
NODAWAY
HOLT
ANDREW
WORTH
GENTRY
DE KALB
BUCHANAN
PLATTE
CLINTON
CLAY
HARRISON
DAVIESS
CALDWELL
RAY
MERCER
GRUNDY
LIVINGSTON
CARROLL
PUTNAM
SULLIVAN
LINN
CHARITON
SCHUYLER
ADAIR
MACON
RANDOLPH
HOWARD
SCOTLAND
KNOX
SHELBY
MONROE
CLARK
LEWIS
MARION
RALLS
AUDRAIN
PIKE
LINCOLN
ST CHARLES
ST LOUIS
WARREN
MONTGOMERY
CALLAWAY
BOONE
COOPER
SALINE
LAFAYETTE
JACKSON
CASS
JOHNSON
PETTIS
MONITEAU
COLE
OSAGE
GASCONADE
FRANKLIN
JEFFERSON
MARIES
MILLER
MORGAN
BENTON
HENRY
BATES
ST CLAIR
VERNON
HICKORY
CAMDEN
PULASKI
PHELPS
CRAWFORD
WASHINGTON
IRON
ST FRANCOIS
STE GENEVIEVE
PERRY
CAPE GIRARDEAU
SCOTT
MISSISSIPPI
NEW MADRID
PEMISCOT
DUNKLIN
STODDARD
BOLLINGER
MADISON
WAYNE
BUTLER
RIPLEY
CARTER
REYNOLDS
DENT
SHANNON
OREGON
TEXAS
HOWELL
LACLEDE
DALLAS
POLK
CEDAR
WEBSTER
WRIGHT
DOUGLAS
OZARK
GREENE
CHRISTIAN
TANEY
STONE
BARRY
LAWRENCE
DADE
BARTON
JASPER
NEWTON
MC DONALD
MISSISSIPPI RIVER
MISSOURI RIVER
OSAGE RIVER
9
14
21
26

May 26, 1983

SUMMARY OF SITES WHERE THE PRESENCE OF DIOXIN HAS BEEN CONFIRMED

1. Minker Residence Site:

   This site is located near Imperial. The known concentrations of dioxin at the site range from .3 to 300 ppb. The contaminated soil at this site originated at the Bubbling Springs Stables. MDNR geologists indicate that this site is in a very poor geological setting. EPA personnel have conducted intense sampling at the site. Contaminated soil from this site has eroded to three (3) adjacent properties. This site along with Romaine Creek and the Stout Residence Site is included on the National Priorities List. A State Superfund contract has been signed with the State for these three sites. The contract covers relocation and a Feasibility Study for the sites.

2. Romaine Creek:

   Drainage to Romaine Creek comes from Minker and Bubbling Springs areas. The known concentrations of dioxin in thee creek sediment range from .3 to 272 ppb. EPA is conducting future sampling and investigation. This site along with Minker and the Stout Residence Site is included on the National Priorities List. A State Superfund contract has been signed with the State for these three sites. The contract covers relocation and a Feasibility Study for the sites.

3. Stout Residence Site:

   This site is located near Imperial. The known concentrations of dioxin at this site range from 2.1 to 21 ppb. The contaminated soil at this site originated at the Bubbling Springs Stables. EPA is presently investigating this site and conducting additional sampling. This site along with Romaine Creek and the Minker Residence Site is included on the National Priorities List. A State Superfund contract has been signed with the State for these three sites. The contract covers relocation and a Feasibility Study for the sites.

4. Spring River:

   This river flows along the property line of the Syntex plants and runs westward. EPA, Missouri Department of Conservation (DOC) Division of Health, and MDNR have combined efforts to investigate the dioxin problem in the river. Sediment samples analysis indicate dioxin levels in the river sediment range from .004 to .087 ppb. Sampling of the fish in the river indicates dioxin levels in the fish flesh range from .019 to .04 ppb. The DOC has issued a warning to the people of the area to limit their fish consumption. A continued sampling effort is being conducted to determine if seasonal trends develope.

5. Denney Farm Site:

This site is located near Verona. This site originally contained levels ranging from 50,000 to 2,000,000 ppb. These high levels are from drum samples buried at the site. EPA and Syntex are working to complete the clean-up of the site. This site has been fenced and buried wastes have been dug up and secured above ground. Syntex has accepted responsibility for this site. The remedial work has been conducted by Syntex under the terms of a Consent Decree.

6. Shenandoah Stables Site:

This site is located near Moscow Mills. The known levels of dioxin at this site range from 1.4 to 1750 ppb. This is another site that was investigated by both EPA and CDC in 1974. The higher concentrations at the site are in the slough area adjacent to the arena. Some of the contaminated material was used as fill material along Highway 61 near the stables. Recent investigations have been unable to confirm dioxin in the fill material. The site is included on the National Priorities List. An Administrative Order to close the arena has been sent to the owner for signature.

7. Bliss Farm Road Site:

This site is located near St. James. The known levels of dioxin at this site range from .1 to 382 ppb. These concentrations are much lower than the concentrations of the samples taken by CDC in 1974.

8. Bubbling Springs Ranch Site:

This site is located near Fenton. The known concentrations of dioxin at this site range from 1.4 to 95 ppb. The site was originally investigated by both EPA and CDC in 1974. Contaminated soils have been excavated over the years and used as fill material at the Minker Stout, Sullins, and Cashel properties. An Administrative Order to close the arena has been sent to the owner for signature.

9. Neosho Digester, Trench, Tank, Spill Area, and Wastewater School:

This site is located near Neosho. The known levels of dioxin at the tank and spill area range from 62 to 1900 ppb. The known levels of dioxin at the digester and trench range from .49 to 60 ppb. EPA, the City of Neosho, and the Receiver of the defunct Wastewater School are negotiating the permanent capping and clean-up of the site. This capping will protect the public from contact until EPA determines an allowable amount of dioxin in the soil. Hopefully, action at this site will be completed by January 1, 1983.

10. Timberline Stables:

This site is located near New Bloomfield. The known levels of dioxin at this site range from .7 to 53 ppb. This is another site that was investigated by both EPA and CDC in 1974. Some of the contaminated soil was supposed to have been disposed of at the Jefferson City Landfill on Highway 94. Investigations thus far have not located any dioxin at the landfill. An Administrative Order to close the arena has been sent to the owner for signature.

11. Erwin Farm Site:

    This site is located near Verona. The known levels of dioxin at the site range from .006 to .83 ppb. The higher concentration is from a composite sample of drums being stored at the site. EPA has been working with Mr. Erwin & Syntex to accomplish the clean-up of the site.

12. Rusha Farm Site:

    This site is located near Aurora. The known levels of dioxin at the site range from .4 to 8 ppb. The higher concentration is from a sample of one drum of filter cake material being stored at the site.

13. Syntex Facility - Verona:

    This site is located near Verona. It consists of several disposal areas on the Syntex Plant property. EPA has been working with Syntex to clean up the site. Syntex has accepted responsibility for this site and is conducting a field investigation under the terms of an Administrative Order to determine the extent of contamination. The level of dioxin in wastes stored on site is about 500 ppb.

14. Bill Ray Farm Site:

    This site is located near Verona. It consisted of eighteen (18) drums removed from the NEPACCO (Syntex) property. Investigations indicate fifteen (15) drums are empty, one (1) drum contained no detectable amounts of dioxin, and two (2) drums with detectable amounts of dioxin. EPA has negotiated with Syntex to return the two (2) drums to the Syntex property.

15. Times Beach Site:

    This site consists of the twenty-eight (28) miles of roads in Times Beach and the city park area. The known levels of dioxin range up to 300 ppb. EPA and MDNR are investigating solutions to the problem. CDC has recommended evacuation of the town. The City is included on the National Priorities List.

16. Ruth Sullins Property:

    This site is located near Imperial. The known concentrations of dioxin at the site range up to 100 ppb. The contaminated soil at this site originated at the Bubbling Springs Stables and covers the corner of her three quarter acre lot. EPA expects to combine this site with the Minker/Stout/Romaine Creek site.

17. Southern Cross Lumber Company Site:

    This site is located in Hazelwood. The known concentrations of dioxin at the site range up to 27.3 ppb. The site was sprayed by Bliss from 1970-76. The site is now covered with gravel.

18. Saddle and Spur Club Site:

This site is located in northern Jefferson County. The known concentrations of dioxin range up to 15 ppb. Investigations indicate the area was oiled by Bliss late '71 or early '72. The stable property is approximately five (5) acres. The arena floor has been covered with sand. An Administrative Order to close the arena has been sent to the owner for signature.

19. Hamill Transfer Company Site:

This site is located in south St. Louis. The known concentrations of dioxin at the site range up to 15.6 ppb. The site was oiled by Bliss on May 27, 1972, using 8,000 gallons of waste oil. Since that time, the site has been "sealed" with a five (5) inch tar and gravel mixture.

20. Overnite Transfer Inc., Site:

This site is located in north St. Louis. The known concentrations of dioxin at the site range up to 9.2 ppb. The site was oiled by Bliss annually between 1962 and 1976. The site is now a five (5) acre graveled parking lot.

21. Jones Truck Lines Site:

This site is located in north St. Louis, The known concentrations of dioxin at the site range up to 22 ppb. The site was oiled by Bliss in the early '70's. Since that time, the site has been paved with asphalt and is now being used as a 4-5 acre parking lot.

22. Methodist Church Site:

This site is located near Ellisville. The known concentrations of dioxin range up to 3.5 ppb. The site was oiled by Bliss in the early 70's. Since that time the site has been paved with asphalt and is now being used as parking for the church.

23. Cashel Residence Site:

This site is located across Romaine Creek Road from Bubbling Springs Stables. The known concentrations of dioxin range between 10 and 100 ppb. The contaminated soil at this site originated at the Bubbling Springs Stables. EPA expects to combine this site with the Minker/Stout/Romaine Creek site.

24. Wall Property and Piazza Road Site:

This site is located near St. James. The known concentrations of dioxin range between 5 and 100 ppb. This site is adjacent to the Bliss Farm Road Site. Piazza Road was oiled by Bliss at the same time Bliss oiled his own road.

25. Russell Bliss Oil Company (Frontenac) Site:

This site is located in Frontenac. The known concentrations of dioxin range up to 100 ppb. This site was used by Bliss to store and mix waste oil until 1977.

26. Quail Run Mobile Home Park:

This site is located near Gray Summit. The known concentrations of dioxin range up to 1100 ppb. EPA's investigation indicates that Bliss oiled the roads at the Mobile Home Park.

27. Sontag Road Site:

This site is located in Castlewood. The known concentrations of dioxin at the site range from 1 to 60 ppb. This site was oiled by Bliss to control dust. This road has since been paved.

28. Syntex Facility - Springfield:

This site is located in Springfield. The known concentrations of dioxin range up to 8 ppb. This site consists of a holding lagoon which is being closed by Syntex under RCRA. EPA is working with Syntex to accomplish the clean up of the site.

29. Mid-America Arena Site:

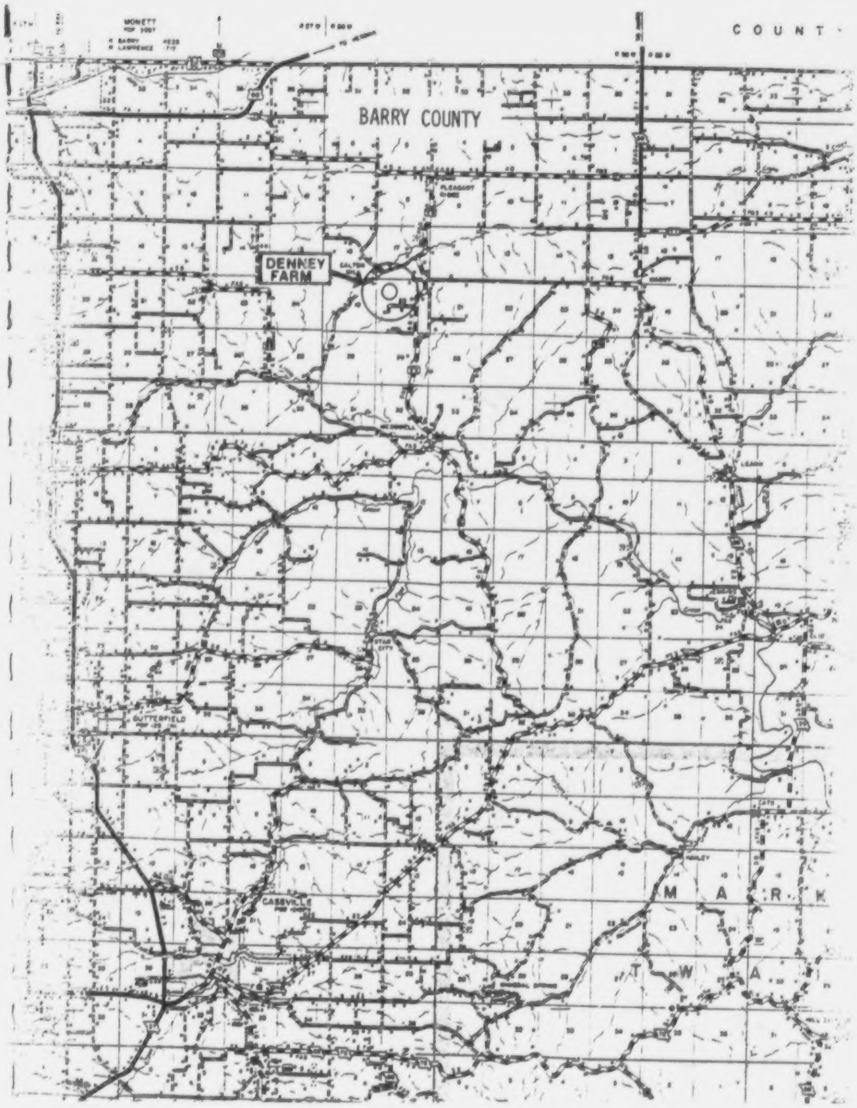
This site is located near Ellisville. The known concentrations of dioxin range up to 78 ppb. This arena is part of the "Ellisville Area Site" which appears on the National Priority List. A Superfund Cooperative Agreement has been signed for the clean up of this site.

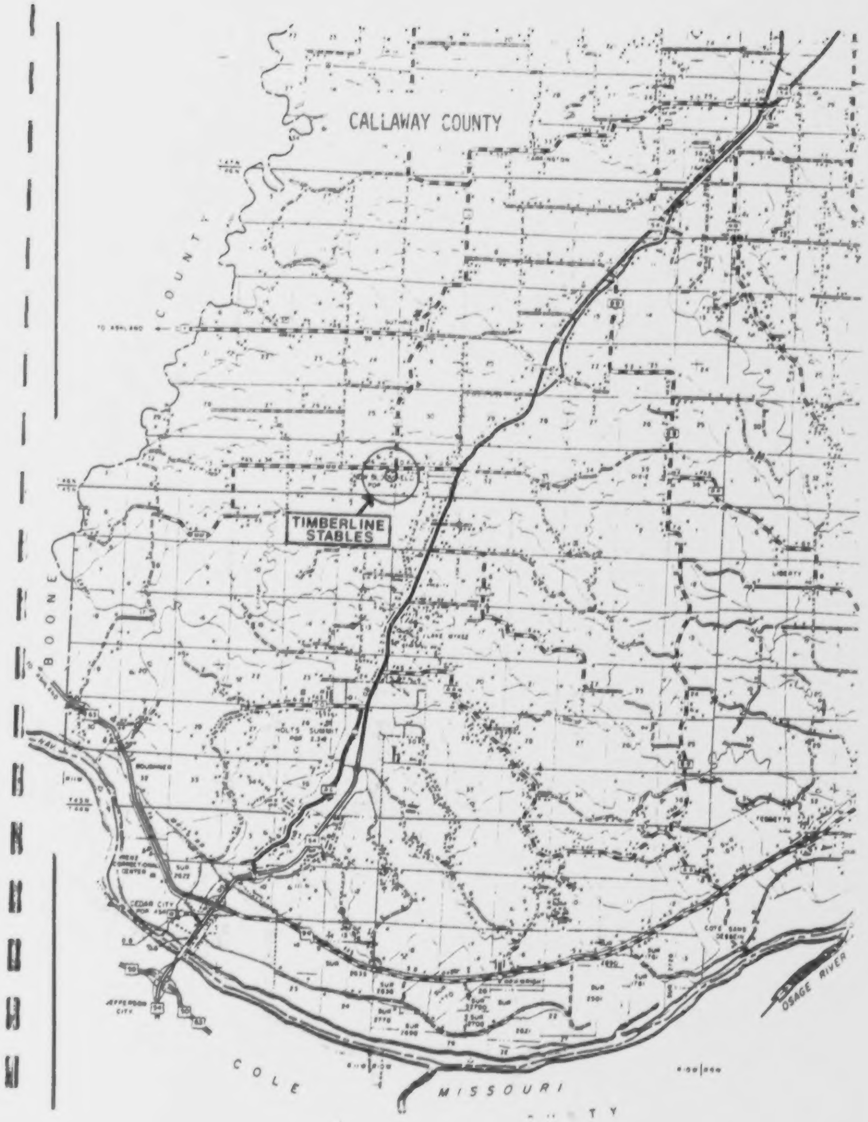
30. Sandcut Road Site:

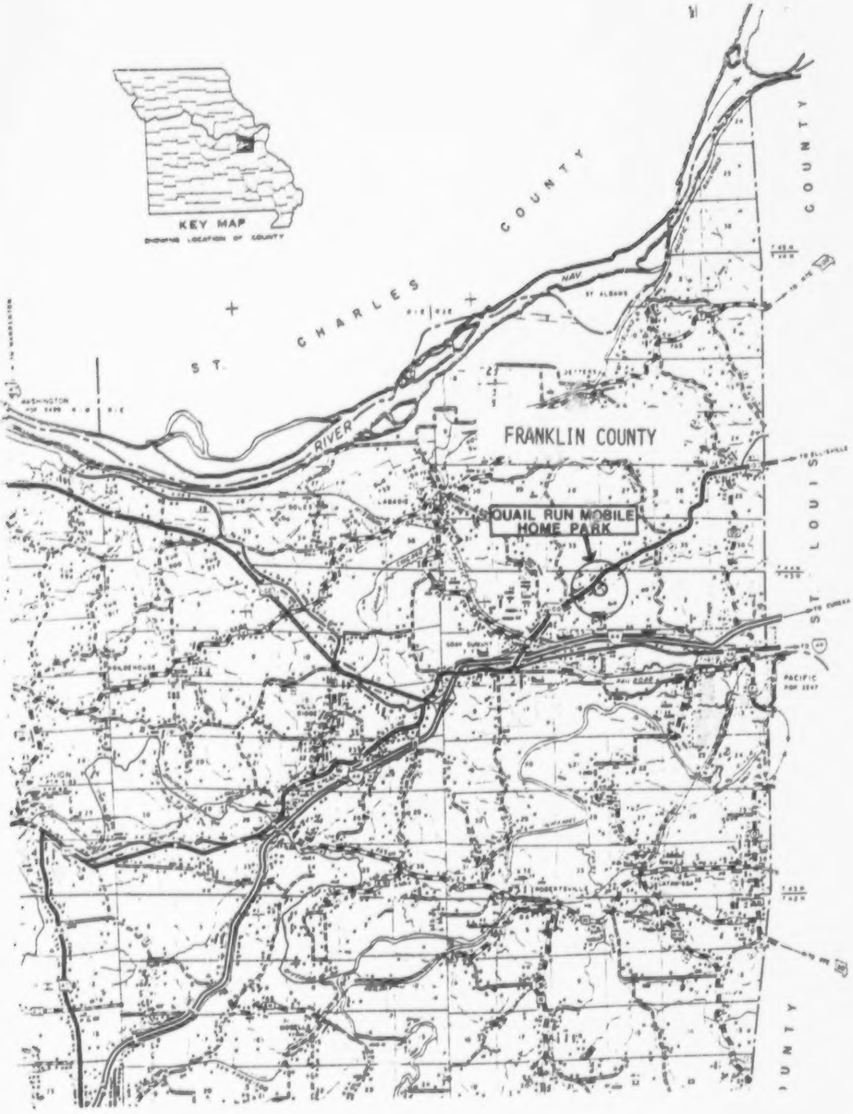
This site is located near Cedar Hill. The known concentrations of dioxin range up to 24 ppb. A former driver for Bliss Oil lived in a trailer along this road and routinely "oiled" the road to control dust.

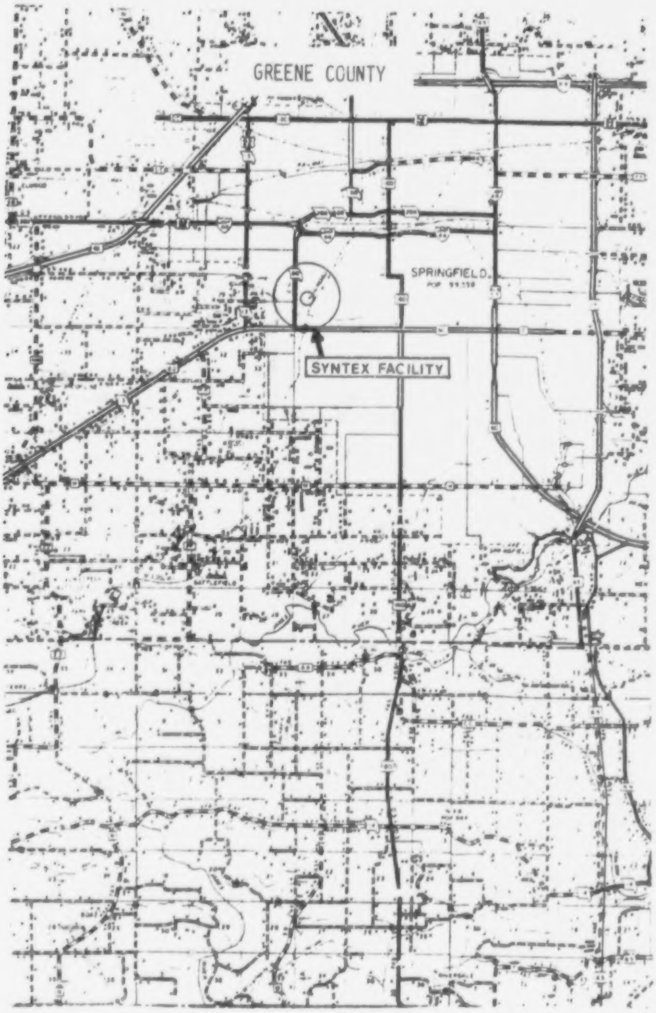
31. Baxter Garden Center:

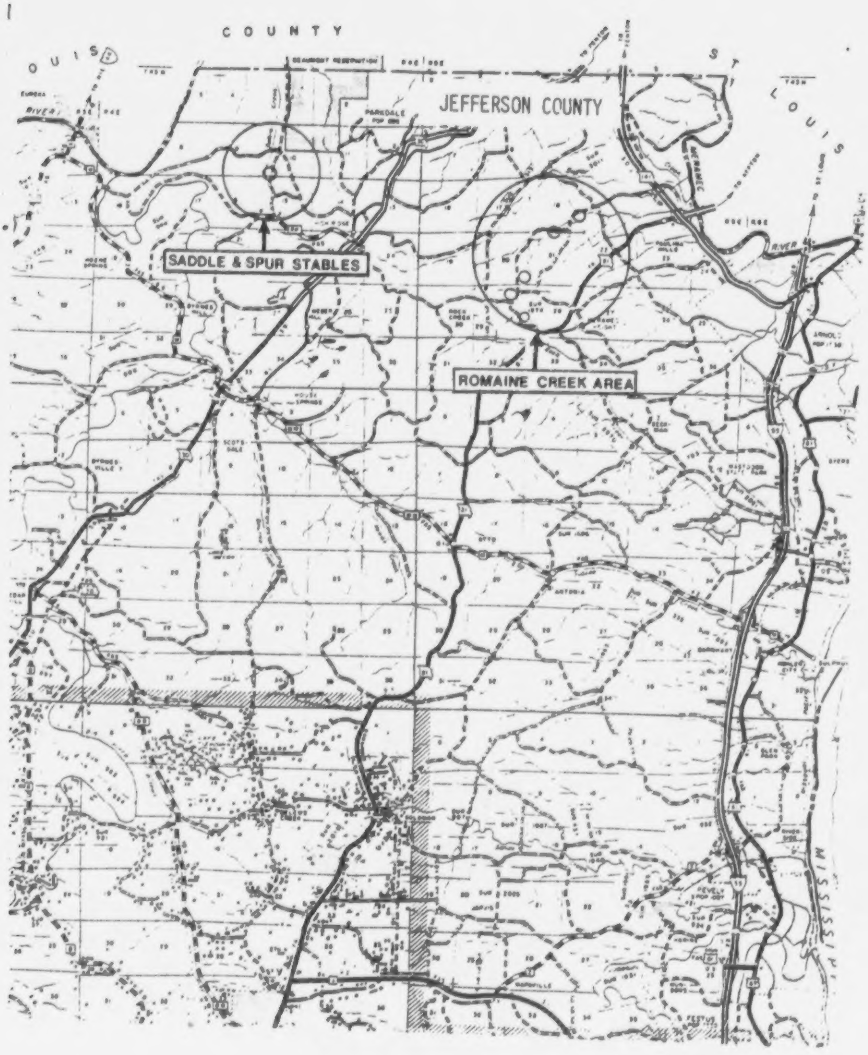
This site is located off Wild Horse Creek Road in Chesterfield. The known concentrations of dioxin range up to 82 ppb. This site was oiled by Bliss in 1971. Even though this site is a commercial nursery, there is little, if any, public access to the contaminated area.

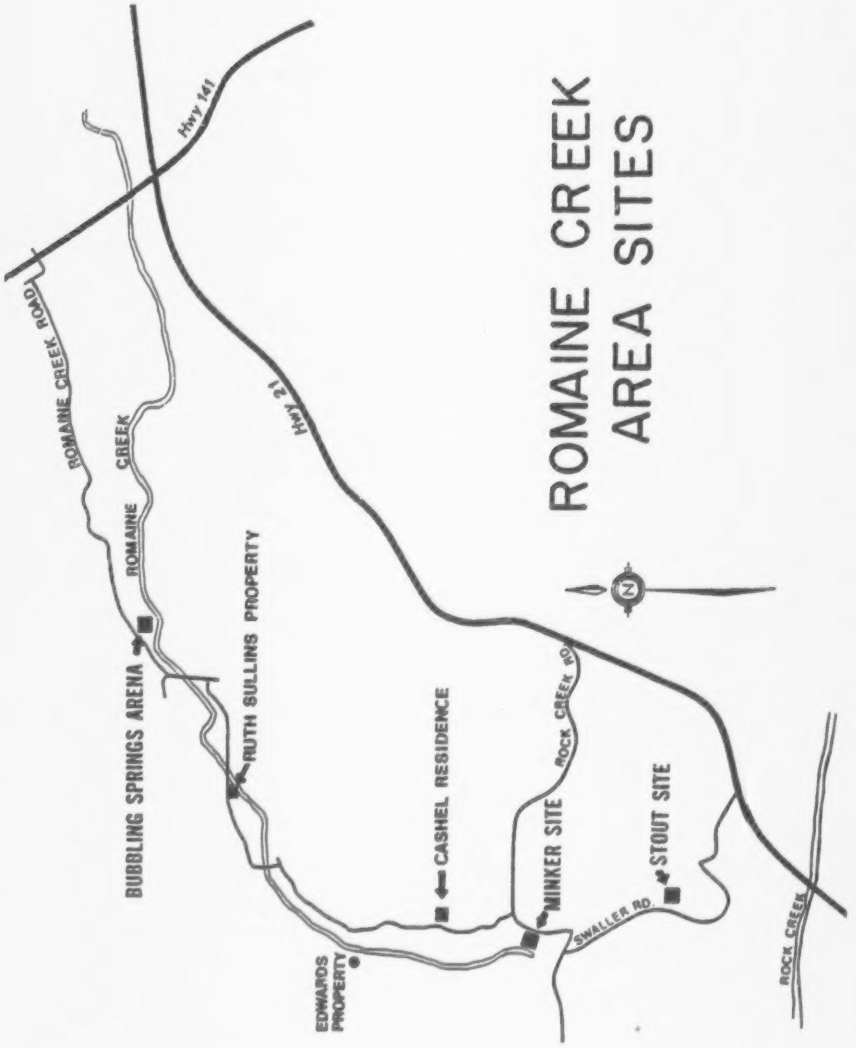

MONETT
COUNT
BARRY COUNTY
DENNEY FARM
CASSVILLE
BUTTERFIELD
M A R K
T W A

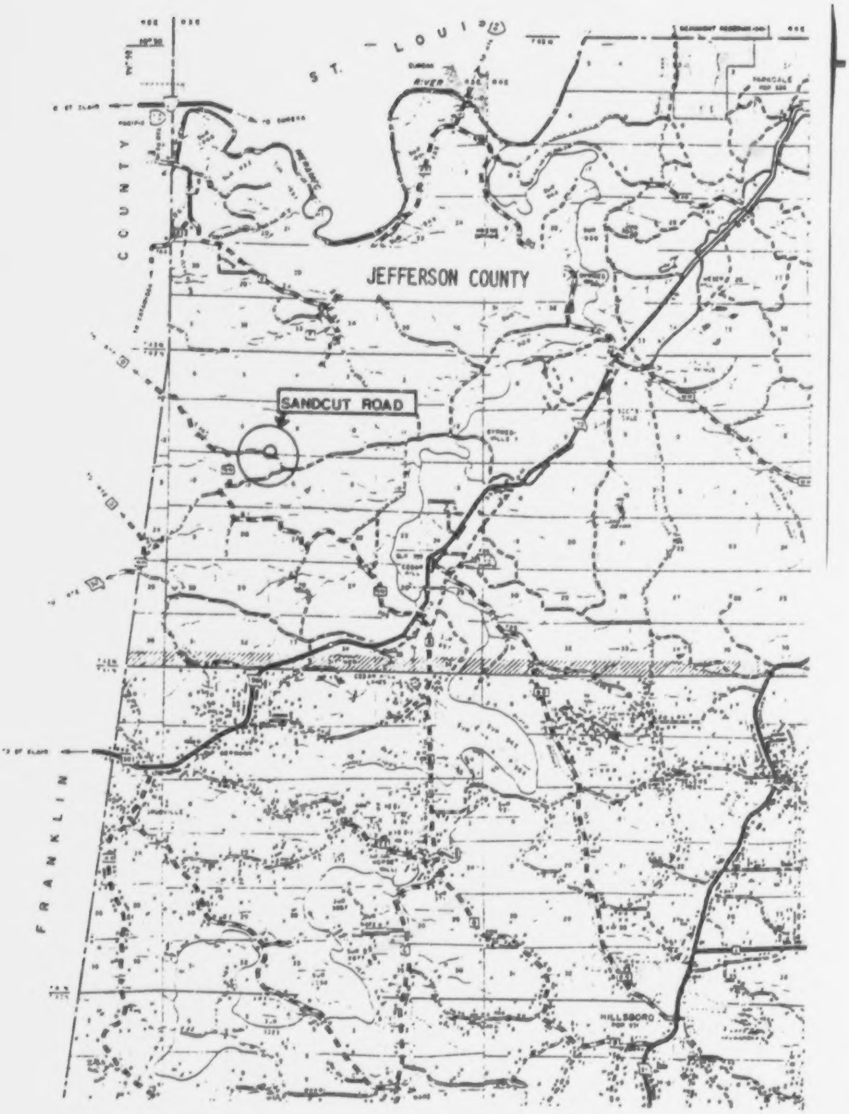

CALLAWAY COUNTY
TIMBERLINE STABLES
BOONE COUNTY
TO ASHLAND
HOLTS SUMMIT
CEDAR CITY
JEFFERSON CITY
LIBERTY
COTE SANS DESSEIN
COLE COUNTY
MISSOURI
OSAGE RIVER

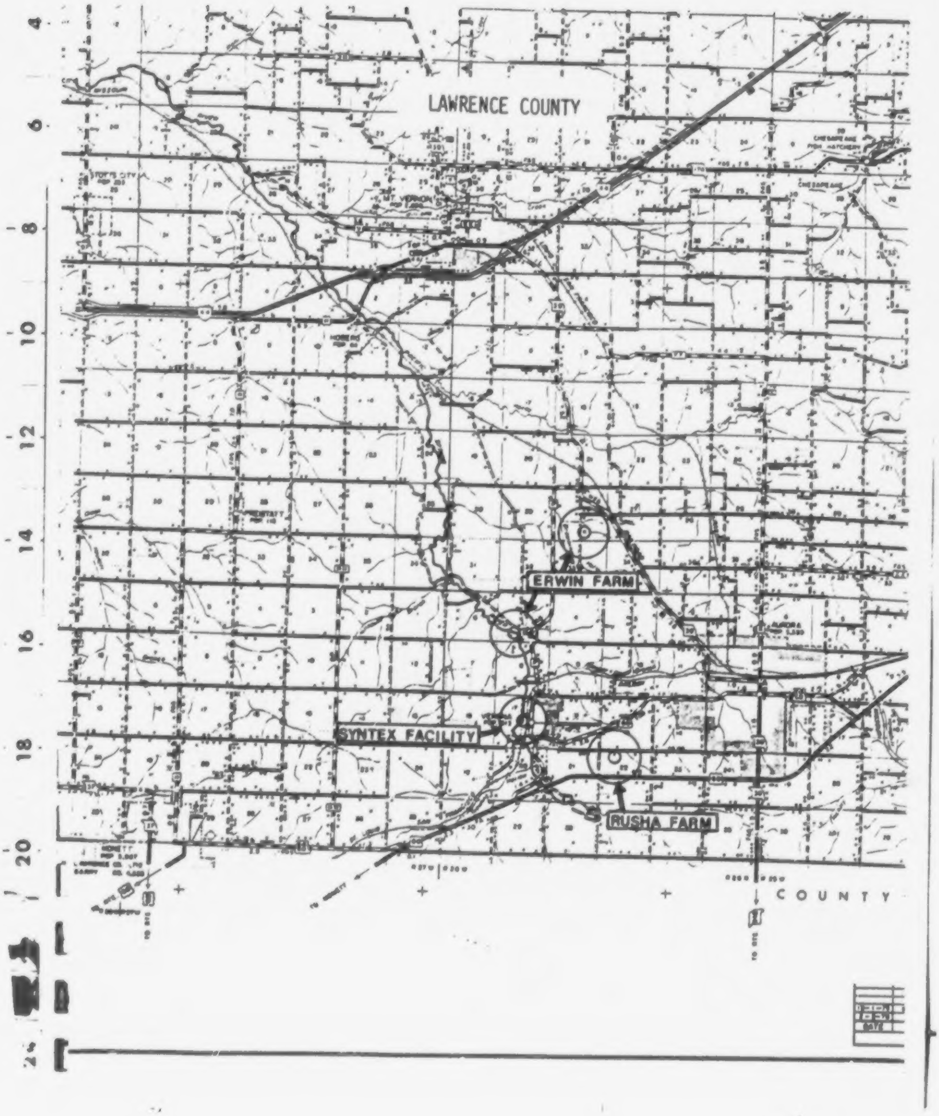

KEY MAP
SHOWING LOCATION OF COUNTY
ST. CHARLES COUNTY
RIVER
NAV
ST. LOUIS COUNTY
FRANKLIN COUNTY
QUAIL RUN MOBILE HOME PARK
PACIFIC
WASHINGTON

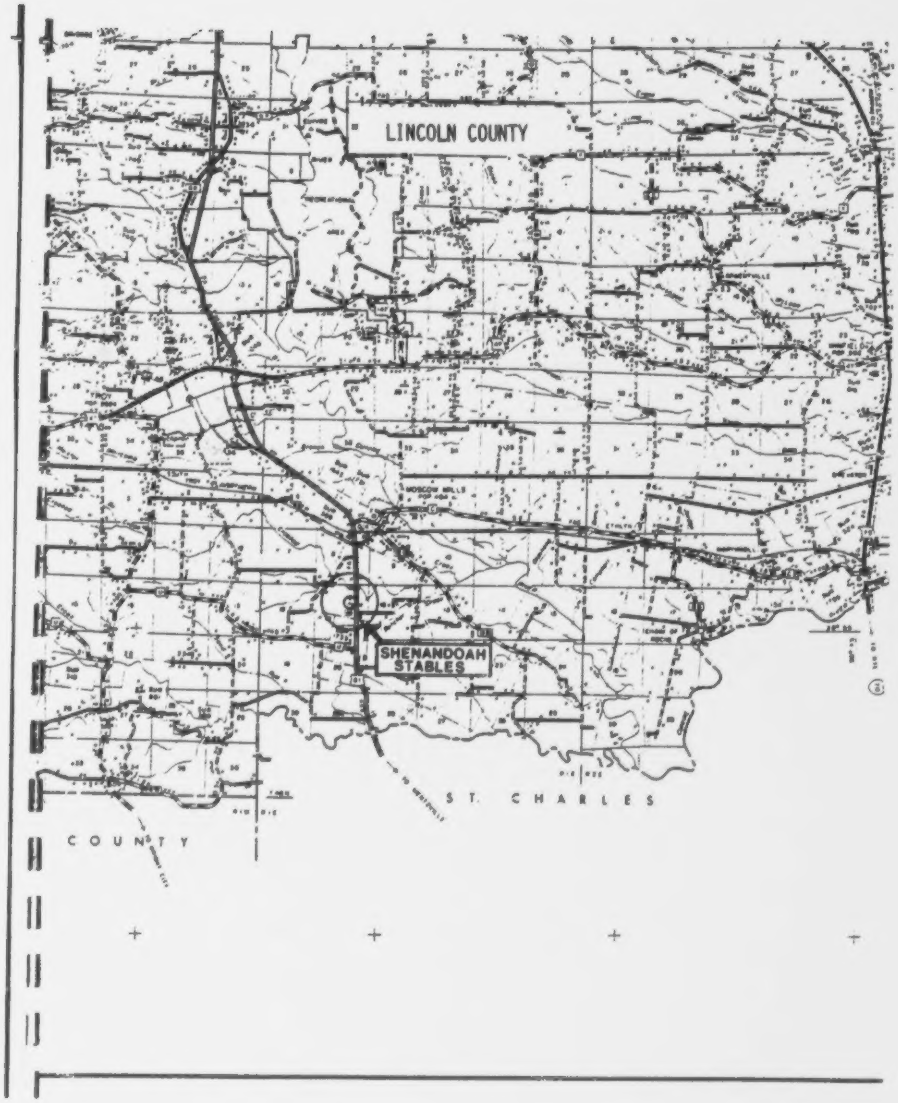

GREENE COUNTY
SPRINGFIELD
SYNTEX FACILITY

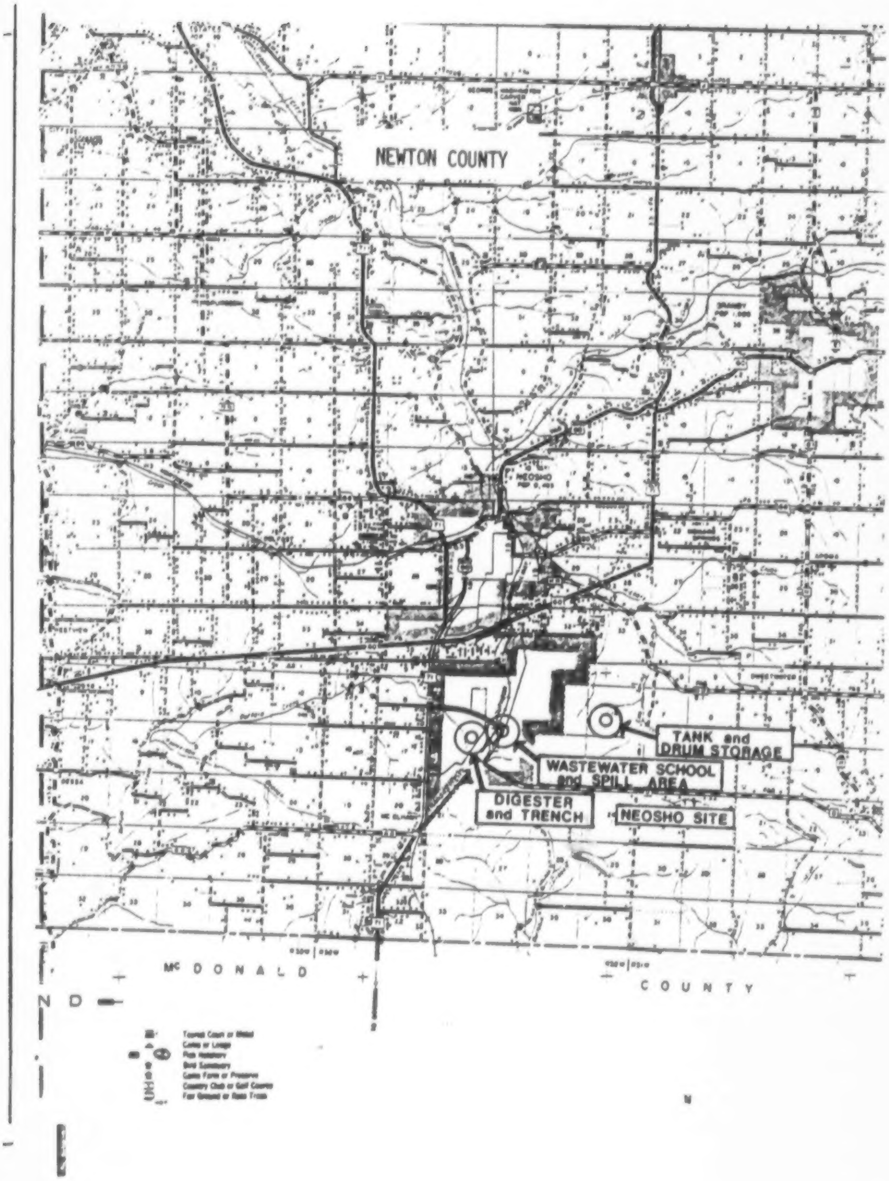

COUNTY
ST LOUIS
JEFFERSON COUNTY
SADDLE & SPUR STABLES
ROMAINE CREEK AREA
MISSISSIPPI

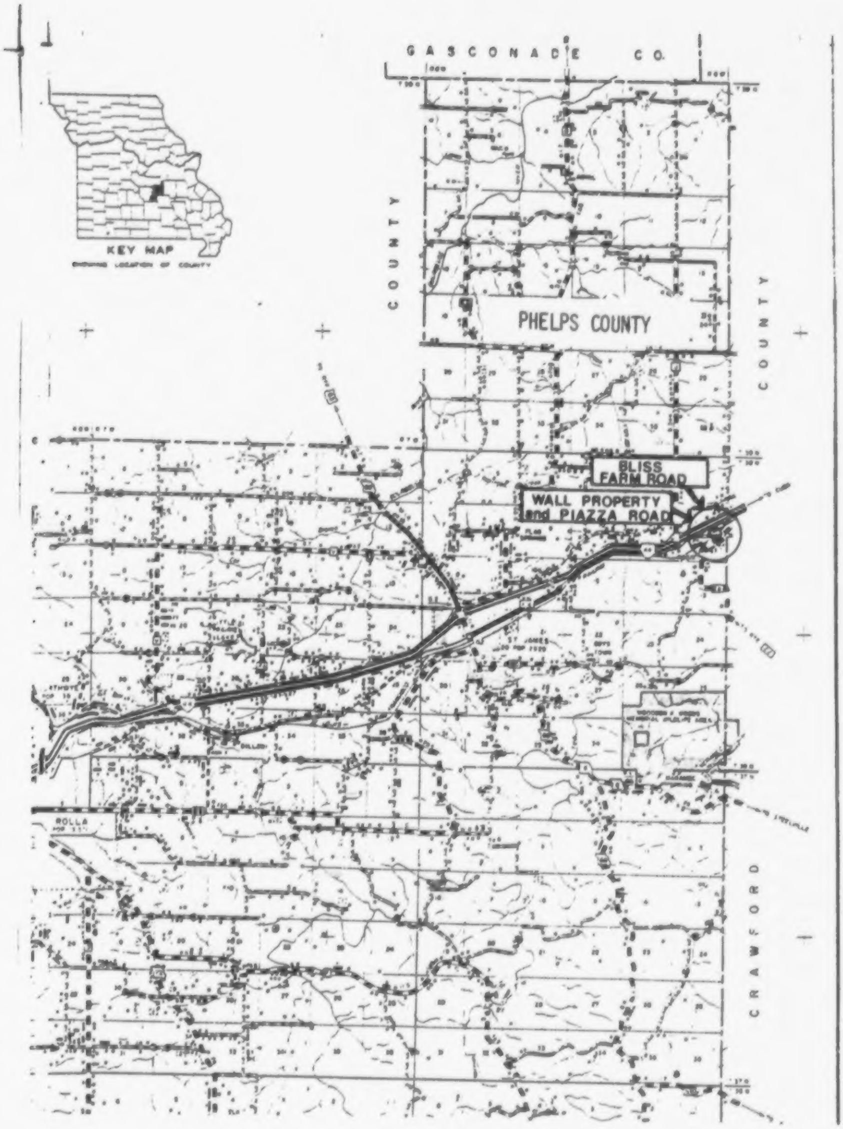

ROMAINE CREEK AREA SITES
Hwy 141
Hwy 21
ROMAINE CREEK ROAD
ROMAINE CREEK
BUBBLING SPRINGS ARENA
RUTH SULLINS PROPERTY
CASHEL RESIDENCE
EDWARDS PROPERTY
MINKER SITE
ROCK CREEK RD.
SWALLER RD.
STOUT SITE
ROCK CREEK
N

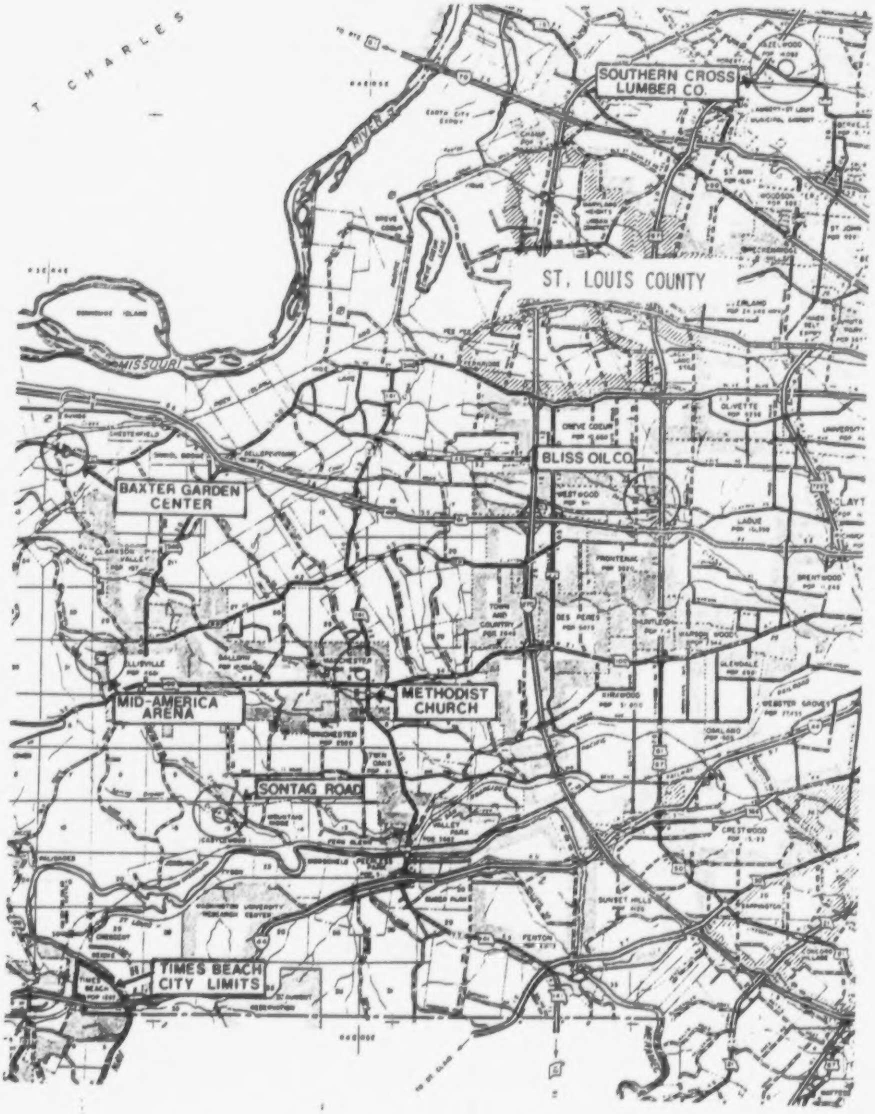

ST. LOUIS
RIVER
JEFFERSON COUNTY
SANDCUT ROAD
COUNTY
FRANKLIN
HILLSBORO

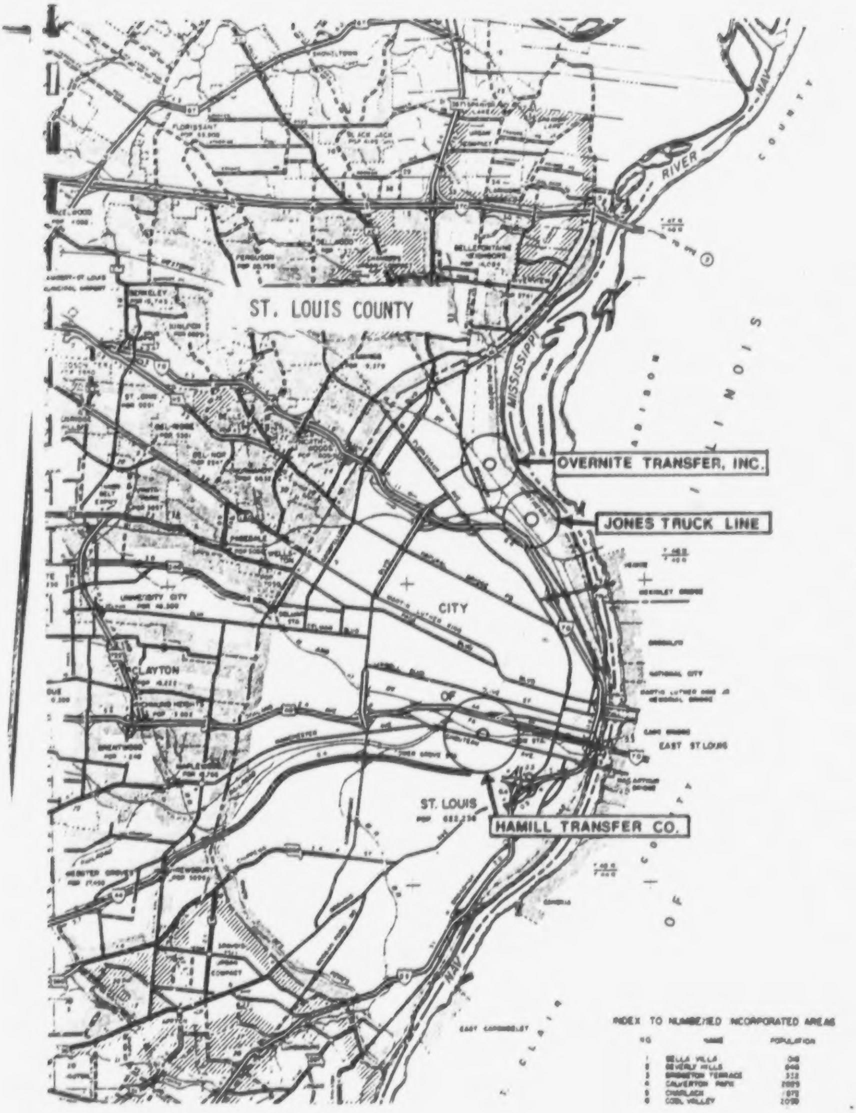

LAWRENCE COUNTY
ERWIN FARM
SYNTEX FACILITY
RUSHA FARM
COUNTY
4
6
8
10
12
14
16
18
20


LINCOLN COUNTY
MOSCOW MILLS
SHENANDOAH STABLES
ST. CHARLES
COUNTY


NEWTON COUNTY
NEOSHO
TANK and DRUM STORAGE
WASTEWATER SCHOOL and SPILL AREA
DIGESTER and TRENCH
NEOSHO SITE
McDONALD
COUNTY
N D
Tourist Court or Motel
Cabin or Lodge
Fish Hatchery
Bird Sanctuary
Game Farm or Preserve
Country Club or Golf Course
Fair Ground or Race Track


KEY MAP
SHOWING LOCATION OF COUNTY
GASCONADE CO.
COUNTY
PHELPS COUNTY
COUNTY
BLISS FARM ROAD
WALL PROPERTY and PIAZZA ROAD
ROLLA
CRAWFORD


T. CHARLES
SOUTHERN CROSS LUMBER CO.
ST. LOUIS COUNTY
MISSOURI
BLISS OIL CO.
BAXTER GARDEN CENTER
MID-AMERICA ARENA
METHODIST CHURCH
SONTAG ROAD
TIMES BEACH CITY LIMITS


ST. LOUIS COUNTY
OVERNITE TRANSFER, INC.
JONES TRUCK LINE
HAMILL TRANSFER CO.
CITY
OF
ST. LOUIS
MISSISSIPPI
RIVER
MADISON
ILLINOIS
COUNTY
OF
ST. CLAIR
FLORISSANT
FERGUSON
BERKELEY
UNIVERSITY CITY
CLAYTON
BRENTWOOD
WEBSTER GROVES
EAST ST. LOUIS
INDEX TO NUMBERED INCORPORATED AREAS
NO.
NAME
POPULATION
BELLA VILLA
BEVERLY HILLS
BRIDGETON TERRACE
CALVERTON PARK
CHARLACK
COOL VALLEY

APPENDIX VII

SUMMARY OF INFORMATION PRESENTED TO THE TASK FORCE BY CH_2M-HILL*

Remedial Action Alternatives Being Considered

Over 50 possible treatment processes and over 500 sources of information were considered in the initial screening of remedial alternatives. The treatment processes shown on Figure 5-2 were screened on the basis of 1) state of development, 2) health and safety risks, 3) process complexity and constructability, 4) reliability, and 5) cost to arrive at six alternatives for further detailed analysis.

The remedial alternatives shown on Figure 5-3 are currently under evaluation for the Minker/Stout site. Five of these alternatives are technically feasible, reliable, and can effectively mitigate the risk to public health, welfare, and the environment with the use of today's technology, while the sixth depends on emerging technology. Alternatives A through C rely on the principal of containment to reduce dioxin exposure which, due to the demonstrated insoluble nature of dioxin in these soils, center around immobilizing the contaminated soil particles. Alternatives D through F rely on the principal of treatment to reduce dioxin level, in addition to containment of the treatment residue.

At this time, no sites have been identified for siting a hazardous waste management facility; therefore, EPA has directed that, for purposes of this study, the Minker site be the assumed location. The socioeconomic, political, and demographic impacts on the community resulting from the siting of such a facility are well beyond the scope of the feasibility study and will not be considered at this time.

Other remedial alternatives are possible and may be considered for other sites, or as a part of an overall Missouri Dioxin Feasibility Study.

Alternative A

This alternative would secure the soil in-place, prevent public site access, and establish a hazardous waste landfill at each fill area. This would likely involve insitu soil fixation and securing of the site with a subsurface perimeter grout curtain, an impermeable cap over the contaminated area, and diversion of surface runoff. Permanent resident relocation and house demolition would be necessary at the sites, and for additional households inside a buffer zone. A long-term site monitoring and maintenance program would be implemented to monitor the groundwater and surrounding environment.

Alternative B

This alternative includes removing the contaminated soil and consolidating into one area on the Minker site. Due to the very shallow soil depth, unsuitable site hydrogeology, and the need for a positve liner and leachate collection system, an above-grade concrete vault would contain the contaminated soil. Permanent resident relocation and house deomolition would be necessary at the sites, and for additional households inside a buffer zone. A long-term site monitoring and maintenance program is also required.

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*CH_2M-Hill is a consulting engineering firm contracted by EPA to do the feasibility study for remedial action at the Minker/Stout site.

Alternative C

This alternative involves removing and transporting the contaminated soil to a secure hazardous waste landfill for disposal. Two potentially acceptable landfill sites have been located within a 600-mile radius of Minker/Stout. Per the April 3, 1983, Federal Register, EPA is currently considering the addition of dioxin wastes to RCRA regulations. If this proposed rule is promulgated, the design and operation of each landfill disposal facility will need to be thoroughly evaluated before dioxin can be added to their permit.

The removed soil volume and up to 2 feet of additional fill would be added to restore the site drainage and to cover any fugitive trace of contaminated soil. The site would be relandscaped and the houses rehabilitated, thus enabling the complete restoration of the area.

Alternative D

This alternative involves the direct thermal destruction of dioxin-bound soil. The contaminated soil would be removed to a concrete vault, where it would remain while the incineration process is pilot tested and developed for this particular contaminated soil. A significant permitting effort may be necessary for the siting of a full-scale facility. Following the permitting process and the construction of the facility, the soil would be transported to a size reduction and handling process, and then to an incinerator. The incinerator particulate and soil residue would be transported to a secure landfill site, unless extensive testing is able to prove them free of TCDD contamination.

The removed soil volume and up to 2 feet of additional fill would be added to restore the site drainage and to cover any trace of contaminated soil. Locating the storage vault and incinerator offsite would enable the area to be relandscaped and the houses rehabilitated, thus enabling the restoration of the area.

Alternative E

This alternative involves extracting the dioxin from the soil with a solvent, concentrating the solvent, and then destroying the dioxin in the solvent. The contamiated soil would be removed to a concrete vault, where it would remain while the solvent extracted process is pilot tested and developed for this particular contaminated soil. A significant permitting effort may also be necessary for the siting of a full-scale facility. Following the permitting process and the construction of this process, the soil would be transported to a size reduction and handling process, and then to the solvent extraction process. The resulting contaminated solvent would be concentrated, with the concentrate undergoing degradation and incineration. The incinerator particulate and the soil residue would be sent to a secure landfill site, unless extensive testing is able to prove them TCDD-free.

The removed soil volume and up to 2 feet of additional fill would be added to restore the site drainage and to cover any trace of contaminated soil. Locating the storage vault, solvent extraction process, and the incinerator offsite would enable the area to be relandscaped and the houses rehabilitated, thus enabling the restoration of the area.

Alternative F

This alternative allows for the development of emerging technologies. The contaminated soil would be removed and stored until such time that emerging technologies such as supercritical water reactors, wet air oxidation, and biological degradation are pilot tested and evaluated for their ability to be competitive with existing technology. Today, these emerging technologies are not considered to be commercially developed for this type of hazardous waste and each has several major technical hurdles to overcome. There is a risk that the emerging technologies may never become cost-effective for this waste and, therefore, a contingency plan will be necessary, should the research and development program not proceed as quickly as planned or is unable to demonstrate on acceptable alternate technology.

The prediction of the timetable for Minker/Stout remedial action is difficult due to unpredictable variables such as site hydrogeologic problems, technical difficulties which may arise during process development, permitting delays, design problems, bidding periods, construction delays, extended public comment periods, and possible legal intricacies. Based on the key assumption that none of these variables will pose an abnormal project delay, the following minimum schedules have been discussed for the completion of the remedial alternatives and the restoration of the site. Onsite remedial activities are only a small portion of the total required schedule.

Alternative	Minimum Total Schedule	Remedial Action
A	3 years	1 year
B	3 years	1 year
C	2-1/2 years	1/2 year
D	8 years	3 years
E	11 years	3 years

Remedial Action Costs

The following comparative cost estimate (+50, -30 percent accuracy) was established for 5,000-8,000 cubic yards of contaminated soil from the Minker/Stout site. This volume estimate is in the process of being revised to reflect the latest increases in the Stout area of contamination.

The estimated costs are intended to be inclusive of all of the direct and indirect costs associated with a particular remedial action for Minker/Stout. The enclosed figure, "Comparison of Costs Included in Remedial Alternatives," indicates a typical scope of what is included in the cost estimate. Many of the costs are highly site-specific for the difficult conditions at the Minker/Stout site and may not apply to any other site.

	Estimated Cost ($ Millions)	
Alternative	5,000 yd^3	8,000 yd^3
A	29	30
B	9	11
C	4 1/2	8
D	29	38
E	57	77

We are in the process of refining the basis of these estimated costs and to incorporate the data received since our March 30 presentation. At this time, it appears that the requirements for size reduction and solvent incineration may be significantly reduced from these initial estimates. Because of this, the capital cost of Alternatives D and E may be reduced by as much as 20 percent over the initial estimates.

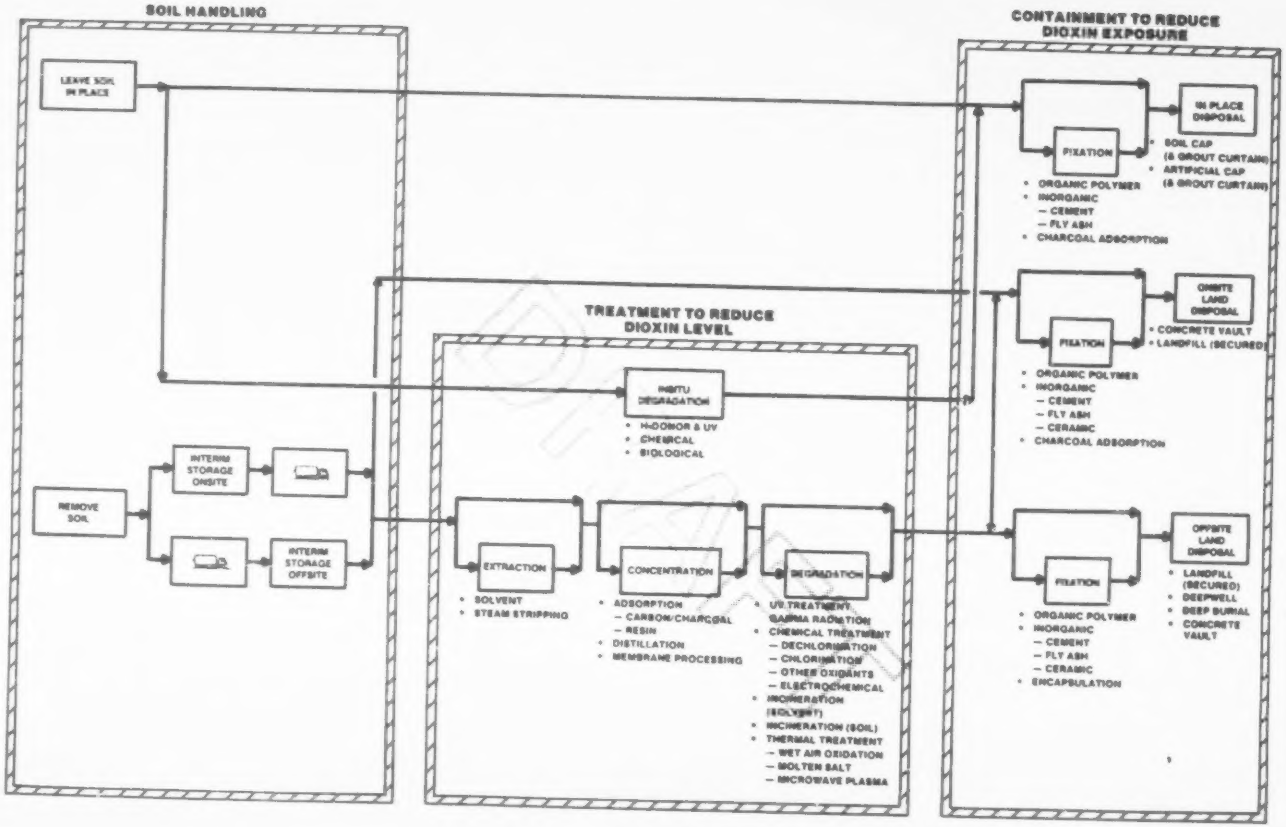
The estimated costs are prepared for guidance in project evaluation and implementation from the information available at the time. The final costs of the remedial action will depend on actual labor and material costs, competitive market conditions, final scope, levels of personnel protection and decontamination, implementation schedule, and other variable factors. As a result, the final project estimate and actual implementation cost will vary from the estimates presented.

Process/Technology	Description	Fate of Dioxin	Byproducts	Commercial Status	References
FIXATION					
1. Organic polymer	Soil containing TCDD is mixed with an organic polymer which hardens.	TCDD and soil are bound together	None	Used routinely on commercial scale for soil; no TCDD experience	
2. Inorganic with cement, fly ash, or ceramic	Soil containing TCDD is combined with portland cement or fly ash. Moisture is added as required to cause the mixture to set.	TCDD and soil are bound together	None	Used routinely on commercial scale for soil; no TCDD experience	
3. Carbon	Soil containing TCDD is mixed with activated carbon. Free or uncomplexed TCDD is adsorbed on the carbon	TCDD in soil and TCDD on carbon are mixed together	None	Conceptual	
4. Encapsulation	Soil containing TCDD is enclosed in a barrier of low permeability such as high density polyethylene	TCDD and soil are enclosed	None	Commonly used for encapsulation of hazardous waste	
SECURE IN PLACE	Securing the site and capping with bentonite clay	TCDD remains on-site in secured location	None	Demonstrated on industrial scale	
IN-SITU DEGRADATION					
1. Ultraviolet degradation	A suitable hydrogen donor (preferably non-toxic and non-volatile) is applied to the soil followed by irradiation with sunlight or high-pressure mercury lamps.	Soil remains in place; TCDD is degraded; TCDD may be spread by the solvent.	Soil contains TCDD decomposition products	Tried at Seveso with limited success	
2. Chemical degradation	A chloroiodide micellar solution is applied to the soil and the ether bonds of the TCDD are cleaved.	Soil remains in place; TCDD is degraded	Soil contains TCDD decomposition products	TCDD in a 100g sample of Seveso soil has been 52% degraded in a laboratory test	
EXTRACTION					
1. Solvent	Soil is processed to enable extraction with solvent and then TCDD is extracted from soil with solvent in either a batch or continuous operation.	TCDD in solvent	Soil containing solvent	Bench-scale experience only with soil but commercial experience with sludge from distillation bottoms at Verona	
2. Steam stripping	TCDD is volatilized from soil by contacting with steam.	TCDD with water as insoluble phase	Possible TCDD thermal decomposition products	Conceptual	
CONCENTRATION					
1. Adsorption, carbon	Solvent containing TCDD is passed through activated carbon	TCDD is adsorbed on carbon	TCDD free solvent	Pilot testing on herbicide orange	
2. Adsorption, resin	Solvent containing TCDD is passed through resin.	TCDD is absorbed on resin	TCDD free solvent	Laboratory tested to remove pesticides from wastewater; conceptual only for TCDD	
3. Distillation	Solvent is distilled and TCDD is concentrated in the distillation bottoms.	TCDD in distillation bottoms	TCDD free solvent	Commercially used in pesticide manufacturing process; also done at Verona	
4. Membrane processing	Selective passage of solvent through a membrane, as in reverse osmosis or ultrafiltration.	TCDD concentrated in solvent	TCDD free solvent	Conceptual	

TABLE 5-1
DIOXIN
PROCESS/TECHNOLOGY
SUMMARY

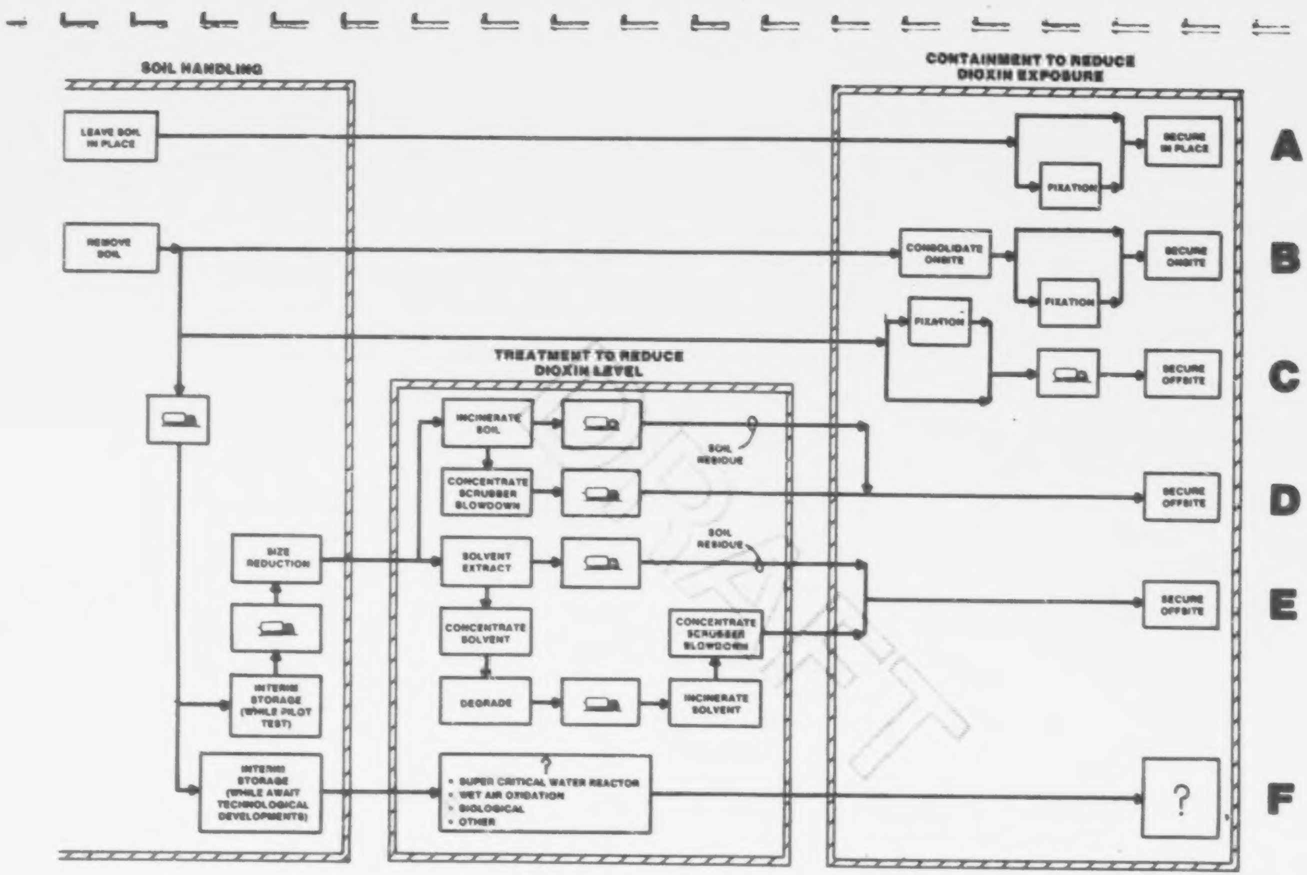
Process/Technology	Description	Fate of Dioxin	Byproducts	Commercial Status	References
DEGRADATION					
1. Radiation treatment					
a. UV degradation	TCDD is irradiated with sunlight or high pressure mercury lamps in the presence of a suitable hydrogen donor after solvent extraction from soil. TCDD in solvent is irradiated with gamma radiation.	TCDD degradation	Solvent with TCDD decomposition products	Commercially done at Verona	
b. Gamma	TCDD in solvent is irradiated with gamma radiation.	TCDD degradation	Solvent with TCDD decomposition products	Laboratory tests on TCDD in solvent	
2. Chemical Treatment					
a. Catalytic dechlorination	TCDD in solvent is dechlorinated with Nickel Borohydride catalyst.	TCDD degradation	Solvent with TCDD decomposition products	Conceptual	
b. Chlorination	TCDD in solvent is extensively chlorinated between 600 and 800° C and 150 psig.	TCDD degradation	Solvent with TCDD decomposition products of CCl_4, HCL, and $COCl_2$	Piloted on herbicide orange	
c. Ruthenium tetroxide	TCDD in solvent is oxidized with ruthenium tetroxide	TCDD degradation	Solvent with TCDD decomposition products	Laboratory tests only	
d. Chloroiodides	TCDD is reacted with chloroiodides in a micellar solution	TCDD degradation	Solvent with TCDD decomposition products	Laboratory tests only	
e. Ozone/UV	TCDD in solvent is reacted with ozone and irradiated with ultraviolet light.	TCDD degradation	Solvent with TCDD decomposition products	Laboratory tests only	
f. Chemical dechlorination	TCDD is reacted with an aromatic liquid and alkaline reactants.	TCDD degradation	Solvent with TCDD decomposition products	Laboratory tests only	
3. Thermal Treatment					
a. Wet air oxidation	TCDD is catalytically oxidized at 200° C in a wet air oxidation process.	TCDD degradation	TCDD decomposition products	Laboratory tests with TCDD	
b. Conventional hazardous waste incineration	TCDD in soil or solvent is combusted in a hazardous waste incinerator.	TCDD degradation	TCDD decomposition products	Commerical incineration available for PCB's, other hazardous wastes; TCDD incinerated with herbicide orange on the M/T Vulcanus	
c. Molten salt combustion	TCDD in solvent is injected below the surface of a molten salt bath in a reactor at 1500° F to 1800° F	TCDD degradation	TCDD decomposition products; spent molten salt melt	Laboratory tests with chlorinated hydrocarbons	
d. Microwave-plasma destruction	TCDD in soil or solvent is mixed with partially ionized gas produced by microwave-induced electron reactions.	TCDD degradation	TCDD decomposition products	Pilot-scale tested on PCB's and other toxic products materials	
e. Pyrolysis	TCDD in soil or solvent is decomposed by heat alone.	TCDD degradation	TCDD decomposition products	Conceptual	

TABLE 5-1 (continued)
DIOXIN
PROCESS/TECHNOLOGY
SUMMARY


SOIL HANDLING
LEAVE SOIL IN PLACE
REMOVE SOIL
INTERIM STORAGE ONSITE
INTERIM STORAGE OFFSITE
TREATMENT TO REDUCE DIOXIN LEVEL
INSITU DEGRADATION
• H-DONOR & UV
• CHEMICAL
• BIOLOGICAL
EXTRACTION
• SOLVENT
• STEAM STRIPPING
CONCENTRATION
• ADSORPTION
— CARBON/CHARCOAL
— RESIN
• DISTILLATION
• MEMBRANE PROCESSING
DEGRADATION
• UV TREATMENT
GAMMA RADIATION
• CHEMICAL TREATMENT
— DECHLORINATION
— CHLORINATION
— OTHER OXIDANTS
— ELECTROCHEMICAL
• INCINERATION (SOLVENT)
• INCINERATION (SOIL)
• THERMAL TREATMENT
— WET AIR OXIDATION
— MOLTEN SALT
— MICROWAVE PLASMA
CONTAINMENT TO REDUCE DIOXIN EXPOSURE
FIXATION
• ORGANIC POLYMER
• INORGANIC
— CEMENT
— FLY ASH
• CHARCOAL ADSORPTION
IN PLACE DISPOSAL
• SOIL CAP (& GROUT CURTAIN)
• ARTIFICIAL CAP (& GROUT CURTAIN)
FIXATION
• ORGANIC POLYMER
• INORGANIC
— CEMENT
— FLY ASH
— CERAMIC
• CHARCOAL ADSORPTION
ONSITE LAND DISPOSAL
• CONCRETE VAULT
• LANDFILL (SECURED)
FIXATION
• ORGANIC POLYMER
• INORGANIC
— CEMENT
— FLY ASH
— CERAMIC
• ENCAPSULATION
OFFSITE LAND DISPOSAL
• LANDFILL (SECURED)
• DEEPWELL
• DEEP BURIAL
• CONCRETE VAULT

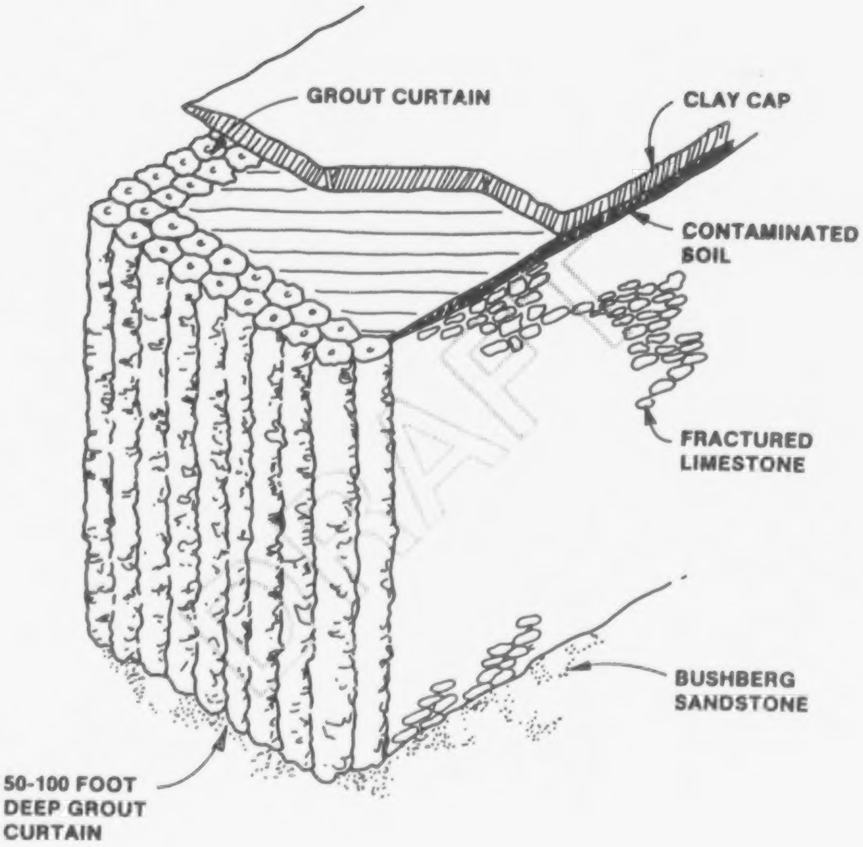
DRAFT

FIGURE 5-2
REMEDIAL ACTION APPROACHES
MINKER/STOUT


SOIL HANDLING
TREATMENT TO REDUCE DIOXIN LEVEL
CONTAINMENT TO REDUCE DIOXIN EXPOSURE
LEAVE SOIL IN PLACE
REMOVE SOIL
SIZE REDUCTION
INTERIM STORAGE (WHILE PILOT TEST)
INTERIM STORAGE (WHILE AWAIT TECHNOLOGICAL DEVELOPMENTS)
INCINERATE SOIL
CONCENTRATE SCRUBBER BLOWDOWN
SOLVENT EXTRACT
CONCENTRATE SOLVENT
DEGRADE
INCINERATE SOLVENT
CONCENTRATE SCRUBBER BLOWDOWN
SOIL RESIDUE
SOIL RESIDUE
?
• SUPER CRITICAL WATER REACTOR
• WET AIR OXIDATION
• BIOLOGICAL
• OTHER
FIXATION
FIXATION
FIXATION
CONSOLIDATE ONSITE
SECURE IN PLACE
SECURE ONSITE
SECURE OFFSITE
SECURE OFFSITE
SECURE OFFSITE
?
A
B
C
D
E
F

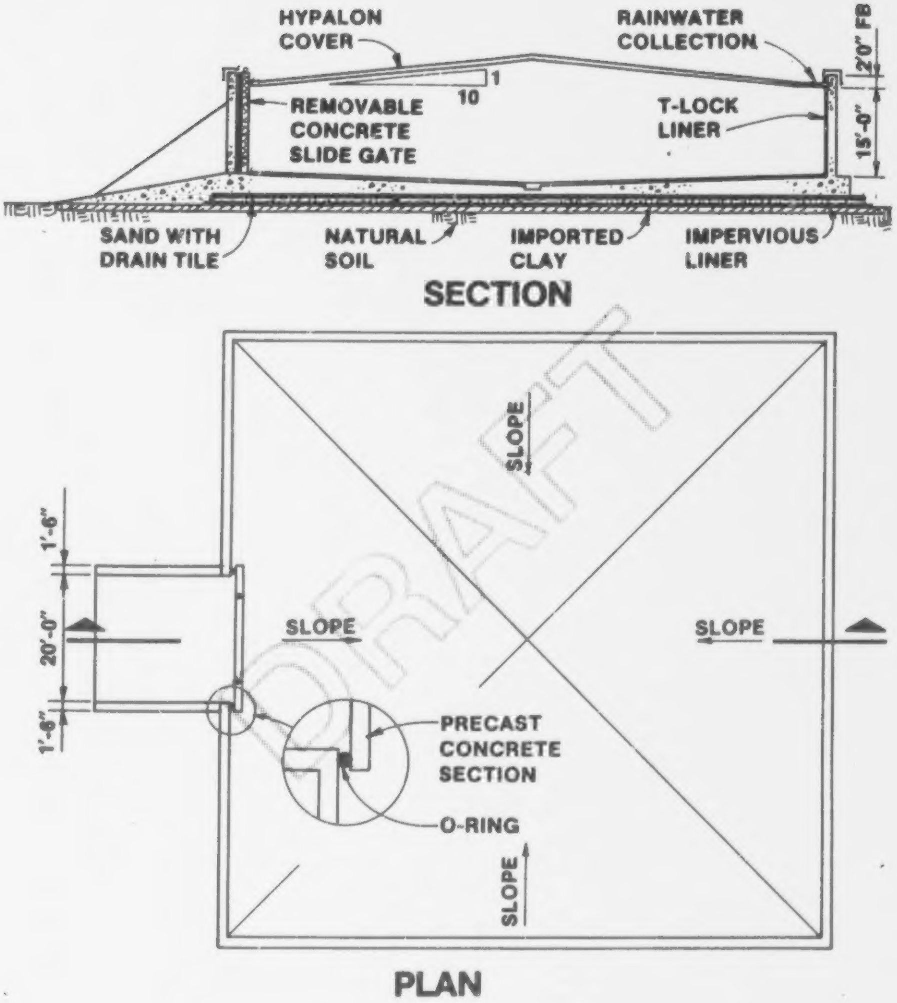
DRAFT

FIGURE 5-3
REMEDIAL ACTION ALTERNATIVES
MINKER/STOUT


GROUT CURTAIN
CLAY CAP
CONTAMINATED SOIL
FRACTURED LIMESTONE
BUSHBERG SANDSTONE
50-100 FOOT DEEP GROUT CURTAIN

DRAFT

FIGURE 5-4
SECURE IN PLACE (ALTERNATIVE A)
CONCEPTUAL DIAGRAM


HYPALON COVER
RAINWATER COLLECTION
2'0" FB
1
10
REMOVABLE CONCRETE SLIDE GATE
T-LOCK LINER
15'-0"
SAND WITH DRAIN TILE
NATURAL SOIL
IMPORTED CLAY
IMPERVIOUS LINER
SECTION
SLOPE
1'-6"
20'-0"
1'-6"
SLOPE
SLOPE
PRECAST CONCRETE SECTION
O-RING
SLOPE
PLAN

DRAFT

FIGURE 5-5
ABOVEGROUND STORAGE VAULT

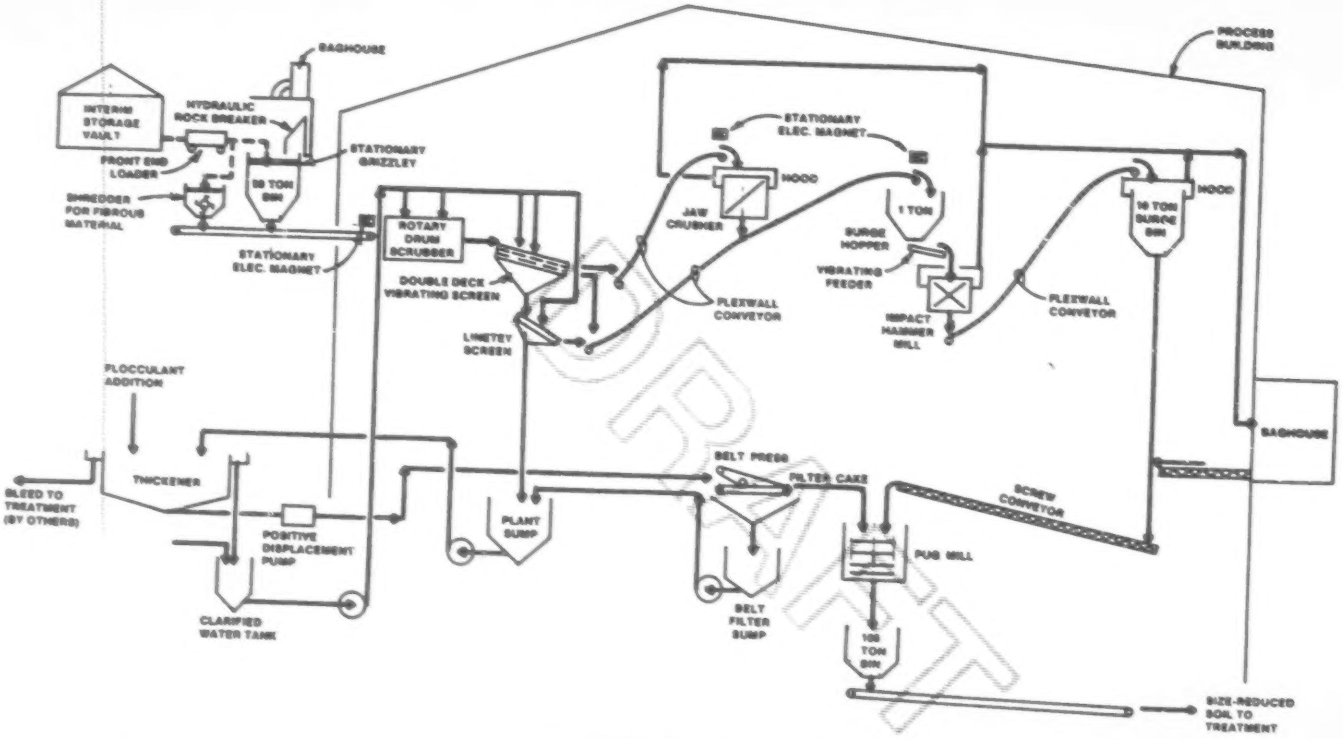

PROCESS BUILDING
BAGHOUSE
INTERIM STORAGE VAULT
HYDRAULIC ROCK BREAKER
FRONT END LOADER
SHREDDER FOR FIBROUS MATERIAL
30 TON BIN
STATIONARY GRIZZLEY
STATIONARY ELEC. MAGNET
ROTARY DRUM SCRUBBER
DOUBLE DECK VIBRATING SCREEN
LINETEY SCREEN
JAW CRUSHER
HOOD
STATIONARY ELEC. MAGNET
FLEXWALL CONVEYOR
1 TON
SURGE HOPPER
VIBRATING FEEDER
IMPACT HAMMER MILL
FLEXWALL CONVEYOR
10 TON SURGE BIN
HOOD
BAGHOUSE
FLOCCULANT ADDITION
BLEED TO TREATMENT (BY OTHERS)
THICKENER
POSITIVE DISPLACEMENT PUMP
CLARIFIED WATER TANK
PLANT SUMP
BELT PRESS
FILTER CAKE
BELT FILTER SUMP
SCREW CONVEYOR
PUG MILL
100 TON BIN
SIZE-REDUCED SOIL TO TREATMENT

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FIGURE 5-6
SIZE REDUCTION
CONCEPTUAL FLOW DIAGRAM

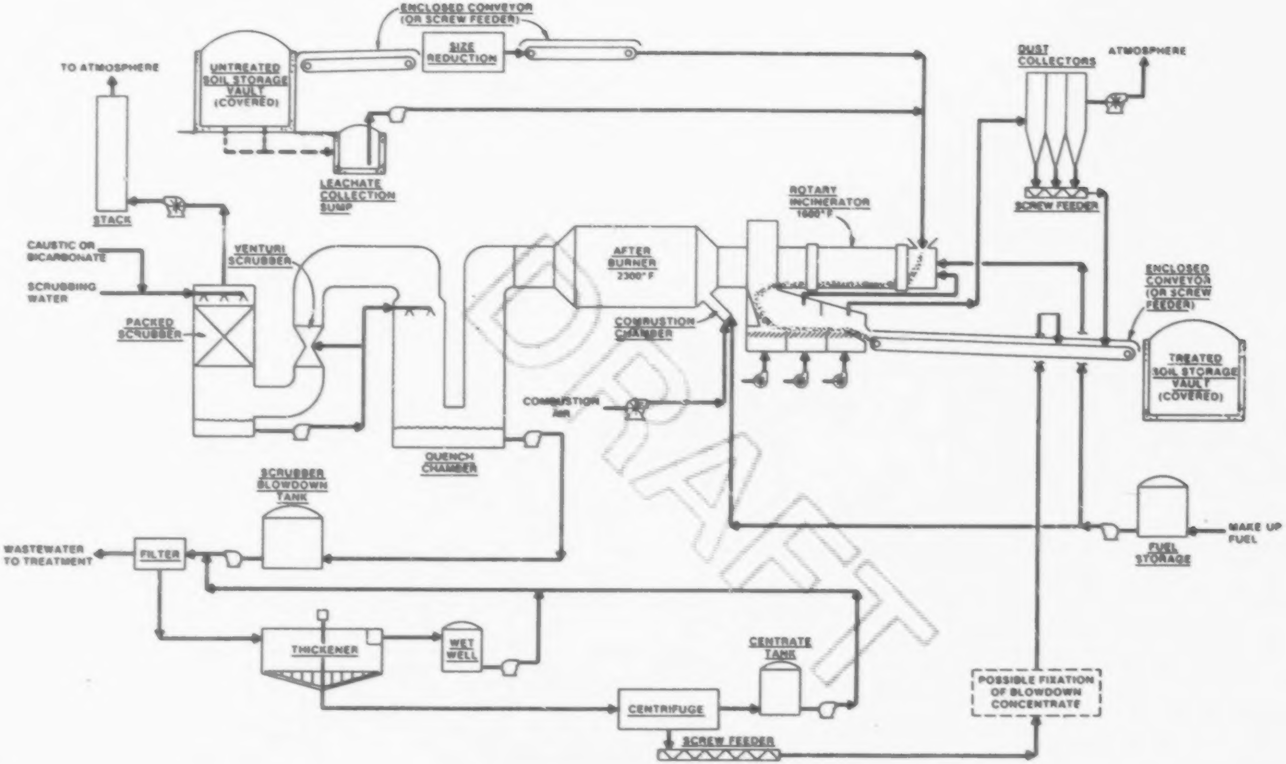
TO ATMOSPHERE
UNTREATED SOIL STORAGE VAULT (COVERED)
ENCLOSED CONVEYOR (OR SCREW FEEDER)
SIZE REDUCTION
DUST COLLECTORS
ATMOSPHERE
LEACHATE COLLECTION SUMP
STACK
SCREW FEEDER
ROTARY INCINERATOR 1600° F
CAUSTIC OR BICARBONATE
VENTURI SCRUBBER
AFTER BURNER 2300° F
ENCLOSED CONVEYOR (OR SCREW FEEDER)
SCRUBBING WATER
PACKED SCRUBBER
COMBUSTION CHAMBER
TREATED SOIL STORAGE VAULT (COVERED)
COMBUSTION AIR
QUENCH CHAMBER
SCRUBBER BLOWDOWN TANK
WASTEWATER TO TREATMENT
FILTER
MAKE UP FUEL
FUEL STORAGE
THICKENER
WET WELL
CENTRATE TANK
CENTRIFUGE
POSSIBLE FIXATION OF BLOWDOWN CONCENTRATE
SCREW FEEDER

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FIGURE 5-7
INCINERATION
CONCEPTUAL FLOW DIAGRAM

PRELIMINARY

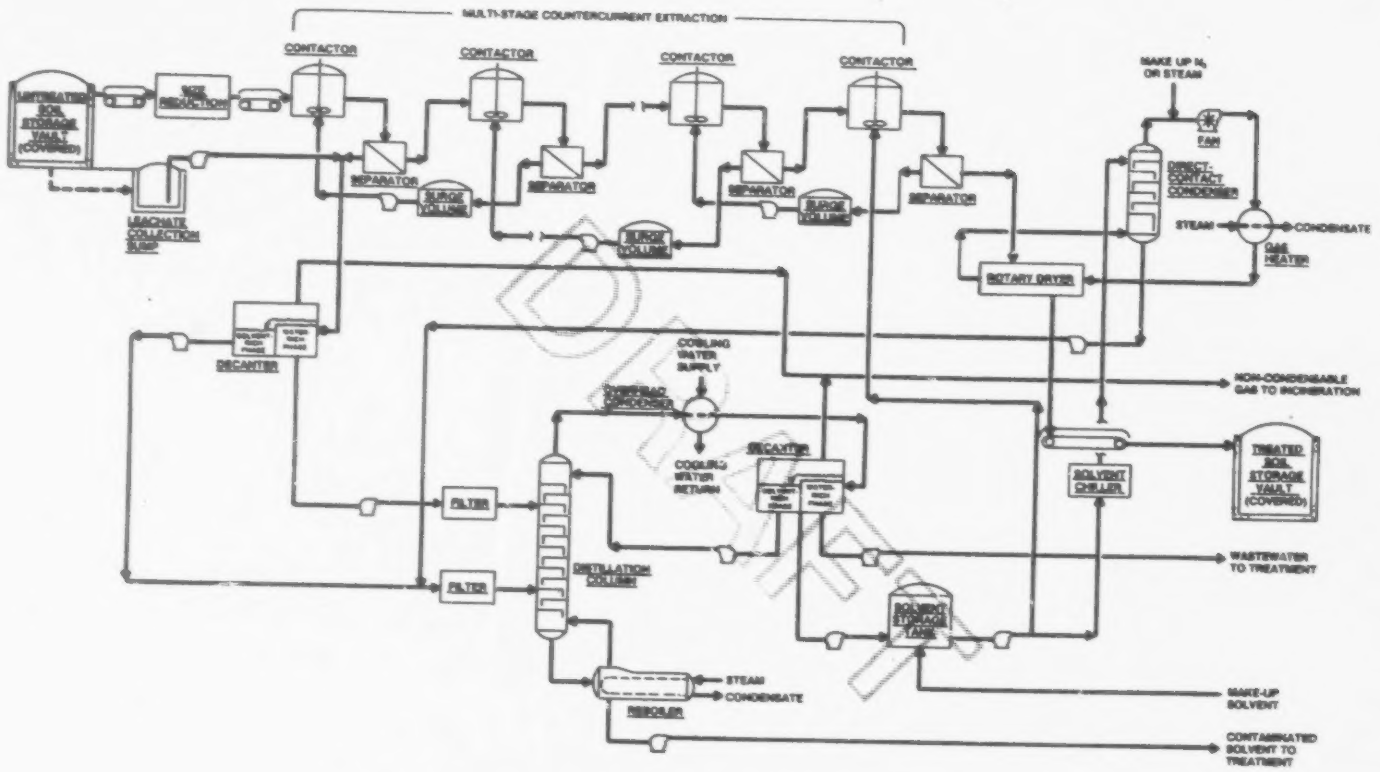
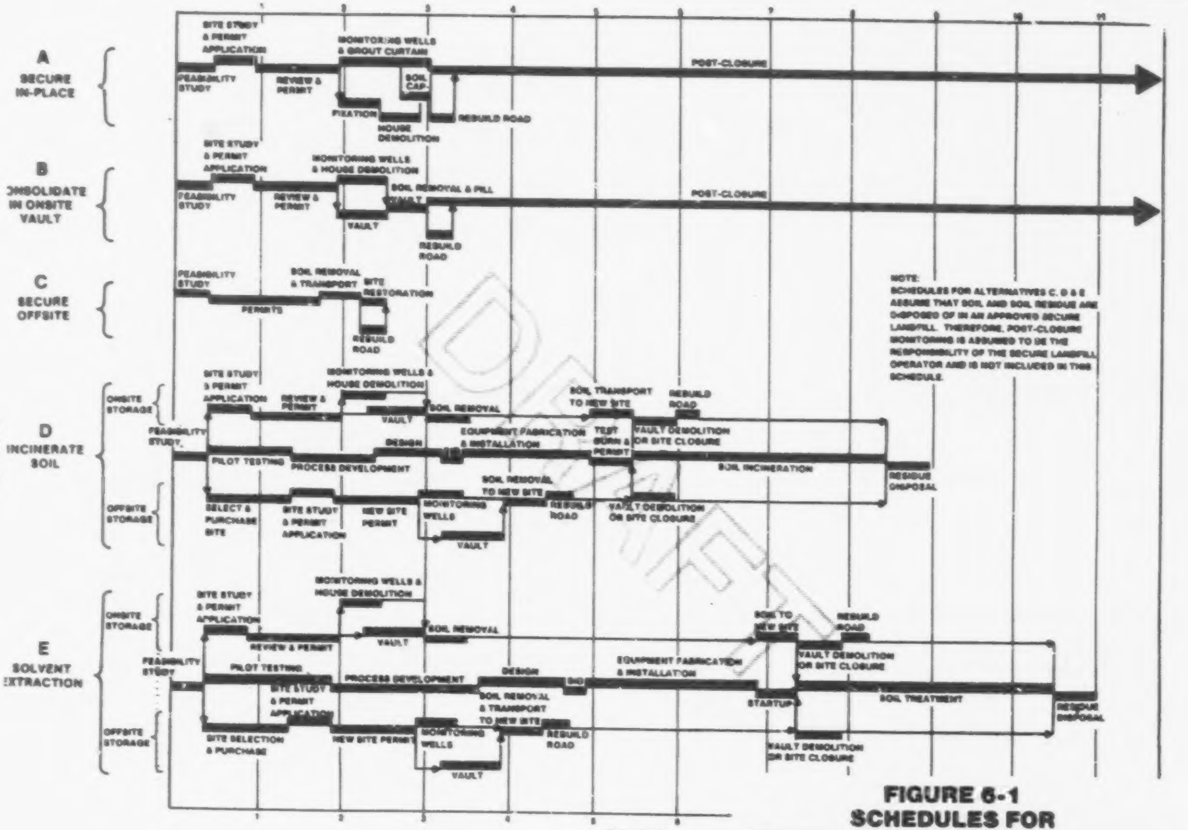

MULTI-STAGE COUNTERCURRENT EXTRACTION
CONTACTOR
CONTACTOR
CONTACTOR
CONTACTOR
UNTREATED SOIL STORAGE VAULT (COVERED)
SIZE REDUCTION
LEACHATE COLLECTION SUMP
SEPARATOR
SEPARATOR
SEPARATOR
SEPARATOR
SURGE VOLUME
SURGE VOLUME
SURGE VOLUME
MAKE UP N2 OR STEAM
FAN
DIRECT-CONTACT CONDENSER
STEAM
CONDENSATE
GAS HEATER
ROTARY DRYER
SOLVENT RICH PHASE
WATER RICH PHASE
DECANTER
COOLING WATER SUPPLY
OVERHEAD CONDENSER
COOLING WATER RETURN
DECANTER
NON-CONDENSABLE GAS TO INCINERATION
TREATED SOIL STORAGE VAULT (COVERED)
SOLVENT CHILLER
FILTER
FILTER
DISTILLATION COLUMN
WASTEWATER TO TREATMENT
SOLVENT STORAGE TANK
STEAM
CONDENSATE
REBOILER
MAKE-UP SOLVENT
CONTAMINATED SOLVENT TO TREATMENT

FIGURE 5-8
SOLVENT EXTRACTION
CONCEPTUAL FLOW DIAGRAM

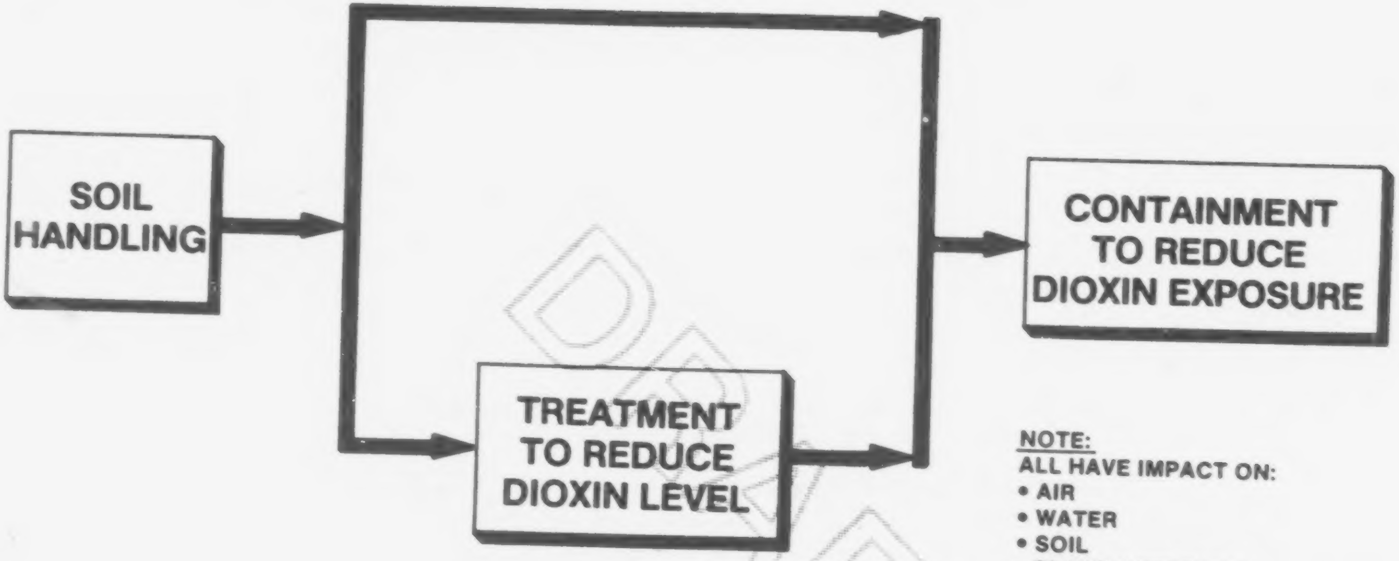
PRELIMINARY

DRAFT



NOTE:
SCHEDULES FOR ALTERNATIVES C, D & E ASSUME THAT SOIL AND SOIL RESIDUE ARE DISPOSED OF IN AN APPROVED SECURE LANDFILL. THEREFORE, POST-CLOSURE MONITORING IS ASSUMED TO BE THE RESPONSIBILITY OF THE SECURE LANDFILL OPERATOR AND IS NOT INCLUDED IN THIS SCHEDULE.

FIGURE 6-1
SCHEDULES FOR
REMEDIAL ACTION ALTERNATIVES
MINKER/STOUT


SOIL HANDLING
TREATMENT TO REDUCE DIOXIN LEVEL
CONTAINMENT TO REDUCE DIOXIN EXPOSURE

NOTE:
ALL HAVE IMPACT ON:

- AIR
- WATER
- SOIL
- PLANT & WILDLIFE
- WORKERS
- PUBLIC
- COMMUNITY

FIGURE 8-1
REMEDIAL ACTION
CONCEPTUAL APPROACHES
MINKER/STOUT

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TABLE 8-1
TCDD EXPOSURE HAZARD SOURCES PRESENT IN REMEDIAL ACTION ALTERNATIVES

TCDD EXPOSURE HAZARD SOURCES	REMEDIAL ACTION ALTERNATIVES					
	A	B	C	D	E	F
AIR ROUTE:						
• DUST GENERATION:						
- Onsite Soil Handling	?	•	•	•	•	•
- House Demolition	•	•		?	?	?
- Rock Drilling	•	•	•	•	•	•
- Highway Transport			•	?	?	?
- Soil Size Reduction Processing				•	•	?
- Residue Handling				•	•	•
• TREATMENT EMISSIONS:						
- Incinerator Emissions				•	•	?
- Solvent Processing Emissions					•	?
- Venting During Upset				•	•	?
SURFACE WATER/GROUNDWATER ROUTE						
• WATER CONTACTING CONTAMINATED SOIL:						
- Runoff From Soil Handling Operations	•	•	•	•	•	•
- Leachate From Containment	•	•	•	•	•	•
- Soil Size Reduction Processing Wastewaters				•	•	?
• WATER CONTACTING PROCESSED SOIL OR RESIDUE FROM TREATMENT						
- Solvent Processing Wastewaters					•	?
- Wastewaters From Incineration				•	•	?
- Runoff From Residue Handling Operations				•	•	?
- Leachate From Containment of Processed Soil Residue				•	•	?
- Emergency Spill				•	•	?
SOIL ROUTE						
• RELEASE OF CONTAMINATED SOIL						
- Onsite Soil Handling	?	•	•	•	•	?
- Soil Size Reduction Processing				•	•	?
- Highway Transport			•	?	?	?
• RELEASE OF PROCESSED SOIL OR RESIDUE FROM TREATMENT						
- Contaminated Soil From Solvent Treatment					•	?
- Contaminated Soil or Residue From Incineration Treatment				•	•	?

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TABLE 8-2
RELATIVE RANKING OF ADVERSE ENVIRONMENTAL IMPACTS OF REMEDIAL ACTION ALTERNATIVES

ADVERSE ENVIRONMENTAL IMPACTS	REMEDIAL ACTION ALTERNATIVES					
	A	B	C	D	E	F
PLANT AND WILDLIFE						
1. TCDD Exposure						
- Air Route	low	low	low	high	med	
- Water Route	low	low	low	med	high	
- Soil Route	low	low	low	med	high	
2. Habitat Destruction	med	med	low	high	high	
WORKERS						
1. TCDD Exposure						
- Air Route	low	low	low	med	med	
- Water Route	low	low	low	med	high	
- Soil Route	med	med	med	med	high	
PUBLIC						
1. TCDD Exposure						
- Air Route	low	low	low	high	med	
- Water Route	low	low	low	med	high	
- Soil Route	low	low	low	med	high	
2. Noise	high	med	med	high	high	
COMMUNITY RESOURCES						
1. Land Area Committed	med	med	low	high	high	
2. Land Use Change	med	med	low	high	high	
3. Energy Consumption	low	low	low	high	high	
4. Topsoil	med	med	med	med	med	
5. Superfund Funding	med	low	low	med	high	
6. Road Deterioration	high	med	med	high	high	

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COST ITEM	REMEDIAL ALTERNATIVES					
SOIL HANDLING/ SOIL REMOVAL MOBILIZATION/DEMOBILIZATION SOIL REMOVAL DECONTAMINATION PERIMETER FENCE SITE TESTING REROUTE RUNOFF REBUILD ROAD MISC SUBTOTAL CONTINGENCY, ENGINEERING, BONDING, INSURANCE, INFLATION ALLOWANCE, ADMINISTRATION TOTAL						
TRANSPORTATION PERMITTING LOADING AND SITE CREW HAULING DECONTAMINATION SUBTOTAL CONTINGENCY, ETC. TOTAL						
STORAGE VAULT SITEWORK OFFSITE: PURCHASE LAND GEOHYDROLOGICAL STUDY SITE PERMITTING CONSTRUCT VAULT MONITORING WELLS LOAD/UNLOAD VAULT DECONTAMINATION AND REMOVE VAULT SUBTOTAL CONTINGENCY, ETC. ONSITE DECONTAMINATION ONSITE HOUSE DEMOLITION CONTINGENCY, ETC. TOTAL * * DOES NOT INCLUDE PURCHASE OF LAND FOR BUFFER ZONE						
SIZE REDUCTION SITEWORK FEED CONVEYORS/HOPPERS DRUM SCRUBBER SCREENS/CRUSHERS/BALL MILL DEWATERING EQUIPMENT PRODUCT CONVEYORS/HOPPERS BUILDING AND ENCLOSURES SUBTOTAL CONTINGENCY, ETC TOTAL						
INCINERATION SITEWORK INCINERATOR/SCRUBBERS SCRUBBER WATER TREATMENT BUILDING/TANKAGE FIREWATER SYSTEM ONSITE CONSTRUCTION INTERCONNECTING PIPING INTERCONNECTING ELECTRICAL PROCESS INSTRUMENTATION SUBTOTAL CONTINGENCY, ETC. TOTAL OPERATING LABOR FUEL ELECTRICITY MAINTENANCE PILOT TESTING AND TEST BURN TOTAL SOIL RESIDUE VAULT TRANSPORT RESIDUE DISPOSAL OF RESIDUE TOTAL						
SOLVENT EXTRACTION SITEWORK CONTACTORS/MIX TANKS DECANT TANKS SEPARATORS DISTILLATION/CONCENTRATION SOIL DRYERS BUILDING/TANKAGE DEGRADATION TRANSPORT SOLVENT INCINERATE SOLVENT SCRUBBER WATER TREATMENT SUBTOTAL CONTINGENCY, ETC. TOTAL OPERATING LABOR SOLVENT ELECTRICITY MAINTENANCE PILOT TEST AND STARTUP TEST TOTAL SOIL RESIDUE VAULT TRANSPORT RESIDUE DISPOSAL OF RESIDUE TOTAL						

COMPARISON OF COSTS INCLUDED IN REMEDIAL ALTERNATIVES

PRELIMINARY
4-12-85

Mr. **Levitas**. Congressman Gephardt, would you like to make your statement at this time or would you like to wait?

Mr. **Gephardt**. I would like to wait and introduce the next group, which is from my district.

Mr. **Levitas**. Thank you.

Mrs. Leistner, you were sort of in the middle of this as an individual and also as a public official. From this point of view, I got the impression that after the Federal response was made and decisions were made, it appeared to me to be a lot of confusion and hassling and haggling within the Missouri State Legislature about whether they were going to put up the matching funds necessary, or how they were going to proceed, and all sorts of technical problems. Were you involved in this at all? Is my perception correct? Or did everything sort of run smoothly?

Mrs. **Leistner**. We have not been in on any of the meetings in Jefferson City, Mr. Chairman. We were made aware that there was a problem with who was going to hold title. There were many people who submitted requests to the State of Missouri to hold title to the property. However, the money had been appropriated, but the State did not want to take title to the property until the property had been acquired, so we had to find a titleholder.

The city of Times Beach submitted an ordinance to the State to hold title to the property until such a time as the buyout was complete. However, there were some problems with the ordinance. There was some concern that there would not be a city, that as each one of us were bought out we would move on. There would be no city officials. Therefore, without city officials how could there be a city to hold title.

However, the Governor finally did accede and the city was allowed to take title. But it was another 2 or 3 months haggling over who was going to take title. People at Times Beach have been on a roller coaster ride since day one of this. The title issue was a big issue in the State of Missouri.

Mr. **Levitas**. You describe the health impact that many people in Times Beach have experience, and you also made some reference to a CDC screening program, I believe, at St. Joseph's Hospital. Was this a treatment facility, or a diagnostic facility? Was this service provided by the Federal, or State medical authorities?

Mrs. **Leistner**. That was provided by both the Federal and State agencies, the Center for Disease Control, Missouri Division of Health. They held a screening at the St. Joseph's Hospital in Kirkwood, MO. Many people felt that the screen was a joke. They did not test but 60 people as far as blood work, skin tests, hair samples. There were 1,190 people who went through the screening. We do not yet have the results of those screenings.

Mr. **Levitas**. Do most of the residents of Times Beach so far as you know have adequate medical insurance to help them with their medical expenses?

Mrs. **Leistner**. Some do, Mr. Chairman, and some don't. In the case of my daughter, when she turned 19 years old, she was removed from our insurance policy because of her age. We appealed, as the union requested that we should. We got a letter from our doctor as to the condition of her health. We submitted all of this, and the fact that she was unable to work because of the seizure disorder, to the insurance company.

Unfortunately, she attempted to take her life in March. It wasn't until we submitted all the bills for the doctors and hospital that we learned that they did not put her back on our policy. So we have some 5,000 dollars' worth of medical bills that we are having to pay out of our own pocket. I am sure that there are many people there who are on fixed income, senior citizens who have. I know of one man who 2 years ago developed a seizure disorder at the age of 80. His wife also has many problems. And as you are well aware, their Medicare does not pay all of these bills. There is some $343 they have to meet themselves.

Mr. LEVITAS. History is full of examples of natural disasters, floods such as you had in Times Beach, hurricanes, tornadoes, earthquakes—acts of God. But there in Times Beach you got really, as you say, a double whammy. You not only had the problem of the flood, a natural disaster, but you also had a disaster that was a manmade disaster. This contamination by toxic chemical.

I wonder if you could give me some response to the extent to which the natural disaster contributed to your problems as compared to the manmade disaster. Which was the greatest, which is the most long range? And give me some sense of how man seems to have improved, if you will, on the ability to cause hardship and human suffering.

Mrs. LEISTNER. Sir, were it just for the December 5 flood, 95 percent of the community would have been rebuilt today. The businesses would be operating. It was the dioxin contamination that chased the people from their homes and closed the doors of the businesses.

Laine Jumper is here. He has talked with many, many people. He is very capable of giving you a very, very well-prepared answer to your question, if that is all right with you.

Mr. LEVITAS. Absolutely.

Mr. Jumper.

Mr. JUMPER. Mr. Chairman, I was building commissioner of the city of Times Beach for about a year and a half. We started, when I took over as building commissioner, cleaning the town up. It started out as a small river community. There were a lot of weekend homes and cottages and that sort of thing. Like the testimony said, we were transforming over to a lower middle-class community; a lot of new homes being built, a lot of things like that.

When the floods came, the flood water was in the town about 5 days. I think it would have left—we were back in about the 10th or 11th of December. Already at that time we had organized some 25 or 30 individual work crews to go in there strictly on a volunteer basis. There was no flood insurance available because Times Beach did not belong to the Federal program. But even without the Federal aid we had enough people, materials, donations, and that sort of thing, lined up to rebuild that town in less than 60 days.

We would have rehabbed most of the homes. Many of the homes were rehabbed by the owners, even during all of this going on, by themselves. There was all kinds of volunteer aid, like I said. Most of it organized and put together by the building contractors and concerned citizens of Times Beach. We would have done, taken care of our own needs without Federal aid, without State aid that we aren't eligible for, I believe, within 2 or 3 months. But when the dioxin announcement came, everything stopped.

The mayor used the word "incredible." That is exactly what it is. I myself had, I think, 11 or 12 crews of 6 or 8 craftsmen, each lined up ready to go on the 20th of December. The 23d that announcement came up and my phone rang off the hook. People said, "We just cannot go back in there. We can't go in there and do this for you. We are sorry." Church groups, unions, people of that nature were more than happy to give out of their heart to help us put the town back together after the flood, but when the dioxin hit that all stopped within 5 days. The town just sat there, and that was the end of it.

Mr. **LEVITAS**. Thank you, Mr. Jumper.

I just have one last question, Mayor Leistner. How would you describe your dealings with the Federal officials and State officials who were working with you to respond to the disaster? Was it efficient? Was it good? Was there smoothness? Could you just give us some idea of how this working relationship between yourself as mayor and also the citizens of Times Beach and the Federal officials, State officials, who were working with you?

Mrs. **LEISTNER**. At first it was absolutely incredible. We would make one phone call. At the onset of this there was nothing exchanged as far as paperwork was concerned. A call to the Governor's office, or call to Pat Brehaney at FEMA. We had anything that we needed. I think that we have found out since then that some of the things that we were told were untrue—well, I shouldn't say untrue, but I think answers were given when answers shouldn't have been given, because some of the officials were not aware of the Superfund laws.

But in February the businessmen were reassured that they would be reimbursed for their loss of income. However, we find out now that that is not true. They are not going to be reimbursed for their loss of income. As far as the emergency help, it was amazing, the help that we had. Everyone responded immediately to the phone calls from city hall. But it seems like in the last 2 or 3 weeks that response is getting slower and slower. I think everybody is very, very tired of hearing about the whole situation in the State of Missouri.

Mr. **LEVITAS**. Well, thank you very much, Mayor Leistner. I believe your statement, and I am sure the statements we will be hearing later today should provide not just the Members of Congress but everybody in America with a real understanding of why we have got to expand and extend the Superfund law. We are all Americans, and we share the loss and the suffering and the anguish that you have communicated. And we need to respond.

Mr. Molinari.

Mr. **MOLINARI**. Thank you, Mr. Chairman. Mayor Leistner, could you tell us how much the cost of the buyout was for Times Beach?

Mrs. **LEISTNER**. The Federal Government appropriated $33 million. The State matched that with 10 percent. I believe it is $36.5 million. That also involves the Minker-Stout site.

Mr. **MOLINARI**. Minker-Stout sites?

Mrs. **LEISTNER**. Well, yes.

Mr. MOLINARI. Does this cover all the area of contamination, or just part of it?

Mrs. LEISTNER. In my estimation there is no way that that amount of money can be—can reimburse the people for the loss of their health. Thirty-six million dollars is not near enough.

Mr. MOLINARI. Well, I understand that, but that is not what I am getting at. What I am suggesting is, that $36-plus million that is being spent, is that for one geographical portion of Times Beach, or is it all of Times Beach, or is it all of the affected area?

Mrs. LEISTNER. It was all of Times Beach. Everything within the corporate limits of the city of Times Beach.

Mr. MOLINARI. Now, is there any indication that the contamination has spread beyond the Times Beach area so that there may be a need for an additional buyout of other areas?

Mrs. LEISTNER. I am not aware of any other areas that are to be bought out at this time other than the Minker site.

Mr. MOLINARI. I have read, and wonder if you can confirm this, that the company responsible for the production of what was the dioxin eventually discovered at Times Beach could have legally disposed of that material for less than $100,000 annually. Is there anybody on the panel that can tell us whether that is accurate or not?

Mr. JUMPER. I am not sure about the figures, Mr. Molinari. But Mr. Bliss, Bliss Oil Co., that hauled this waste oil out of a Napaco plant that manufactured Agent Orange was paid to haul that away. Originally the Napaco people were contracting with, I believe, Rollins Co. in Louisiana, to have it incinerated at I am not sure how much per barrel, but they were paying a fairly high cost for it.

Your figure of $100,000, there was about 18,000 gallons of this waste oil sludge, $5 or $6 a gallon, that would be about $250 or $300 for a 50-gallon drum. I have seen somewhere in the paperwork we have come up with that Rollins at that time was charging about $400 a drum. So that is probably a very accurate figure. That it would have been disposed of for that much money.

But you have got to remember, Mr. Bliss was paid to haul it away, and he was paid by the city of Times Beach also to put it on the roads.

Mr. MOLINARI. I understand that. That is not unusual in this business of hauling toxic waste. They get paid to take it away, and then they get paid to dump it. But the fact is that we are learning that here is a concrete example of where for less than $100,000 the site could have been legally cleaned up. Through neglect, over $36 million has been earmarked, maybe not spent yet but earmarked, and that is not going to be enough to compensate, from what the Mayor said, the people for their true financial loss, leaving aside for the moment the health impacts and health injuries and medical bills that have been or will be incurred.

Mr. JUMPER. That is true. The State of Missouri has said that there are approximately 150 sites. Times Beach is one. Minker-Stout is another. That leaves the possibility of another 140 some potential sites, of which there are over 30 or more known. The mayor's brother lives on a site down in Castlewood. It is incredible.

Mr. **Molinari**. It has been estimated that the total cleanup will exceed $100 million. Now, I don't know if that is accurate or not. But when we compare what it would have cost to legally dispose of that material to what we have before us today, I think we have a concrete example of what the chairman was talking about.

I would like to change the focus for a bit. Mayor, attached to your testimony there is a letter, the Department of Health, Education and Welfare, Centers for Disease Control, dated October 2, 1974.

Mrs. **Leistner**. We attached that because of the date. The State of Missouri knew in 1974 that this problem existed and we wanted the committee to make note of the fact.

Mr. **Molinari**. We are making note of it. That is why I want to ask the questions which will follow. This letter is dated October 2, 1974, sent by the Center for Disease Control, Atlanta, GA, to the Missouri Department of Health. It talks about the contamination of the horse arena soil by TCDD, dioxin as we generally might refer to it. It talks about heavy concentrations, as high as 1,000 parts per million, which is very high. Then it goes on to say that there remains untouched in a storage tank in Verona, MO, an additional quantity of this substance, and recommends disposal through a reputable service.

The letter continues, and advises that prior to disposal, there be consultation with Dow Chemical in Midland, MI. I don't know if that would have been such great advice in light of other disclosures. Then it goes on to say that TCDD is a highly toxic material and its toxicity cannot be overemphasized.

The question I have, of course, is, do I understand correctly that the material that was still remaining in that tank was subsequently, together with the contaminated soil, brought to the Minker-Stout sites?

Mr. **Jumper**. No, sir; the spraying of the oil at the stables occurred in 1971. The soil from those stables was excavated after the horses died—I think twice—and wound up as residential fill at the Minker-Stout site. This took place in 1971 and 1972. It wasn't until 1974 that the TCDD was identified by the CDC as the toxin that killed the horses and made the children ill.

Mr. **Molinari**. What happened to the material, then, that was remaining in the tank?

Mr. **Jumper**. I believe the Missouri Department of Natural Resources, after 4 or 5 years of negotiations and testing, had it incinerated in Louisiana. The tank was taken to Louisiana and buried—and sunk in a bayou, I believe, way out somewhere in Louisiana. We were under the impression that that tank can be incinerated, but it wasn't. That tank still exists.

Mr. **Molinari**. Do we know where it is buried?

Mr. **Jumper**. I am sure that the Missouri Department of Natural Resources, and I don't know which State or Federal agency was involved in halting that thing, knows where it is. But it still exists and it is my understanding sunk in a bayou somewhere.

Mr. **Molinari**. Mr. Jumper, with respect to both the remaining residue in that tank referred to in this letter and the contaminated soil that they speak about, all materials were legally disposed of, is that correct?

Mr. **Jumper**. No; the soil I was talking about from the stables that were sprayed in 1971 was hauled and used as fill at the Minker-Stout site. That is how the Minker-Stout site came into being. Russell Bliss sprayed the horse arenas with this dioxin-contaminated sludge. There was almost a hundred horses that had died from three or four different stables. Soil from the Shenandoah stables, which is very close to where they lived, was hauled to that area and used as landfill.

Mr. **Molinari**. What I am trying to determine—maybe you can help me—the soil, the contaminated soil they are referring to here, was that properly disposed of?

Mr. **Jumper**. No, sir; it was not.

Mr. **Molinari**. Now, if the Missouri Department of Health had knowledge, as obviously they did by this letter, as to the seriousness of the material they were dealing with—and there are criteria here, recommended criteria for the disposal of it—why did the Missouri Department of Health not respond properly?

Mr. **Jumper**. I think the misunderstanding we have is that this letter is in 1974 and death of the horses and removing of the soil occurred in 1972, I believe. 1972.

Mr. **Molinari**. But this letter of 1974 referred to contaminated soil. The question was, after that, was any of that contaminated soil disposed of illegally?

Mr. **Jumper**. No, no; I don't know if it was disposed—nothing was ever done since. There is another letter from the CDC—I'm not sure if it is in here or not—that recommends that that soil be excavated and removed to a remote deep landfill somewhere away from these people. That was never done. They just left it sit. There was some controversy at that time about the half-life of TCDD. CDC recommended taking a test of dioxin levels and coming back in 6 months and 1 year, and testing again to see if it actually did degenerate in the soil. They took the first set of tests and that was the end of it. They never took the second set of tests, and the soil remains there to this day.

Mr. **Molinari**. Did anybody ever come back with a determination on what the half-life of it is?

Mr. **Jumper**. Originally, they tried to say it was from 6 months to 1 year. There is still some debate on that, but they know it is in excess of 20 years.

Mr. **Molinari**. Mayor, how many people are we talking about who are involved in Times Beach, roughly?

Mrs. **Leistner**. Approximately 2,200 people. Our 1980 census showed—was showing that we have 2,041 people living in the community, and we are estimating it at 2,200 people. But there are also a lot of people, telephone company, union electric workers that came into the community to work.

Mr. **Molinari**. This condition was discovered when, approximately?

Mrs. **Leistner**. We first learned about it in November 1982.

Mr. **Molinari**. When did that health screening that you talked about before take place?

Mrs. **Leistner**. That took place in January, February, and March.

Mr. **Molinari**. Of this year?

Mrs. Leistner. Yes, sir.

Mr. Molinari. I assume that a number of people have moved to other parts of the country?

Mrs. Leistner. There are only 14 families still living in the city of Times Beach.

Mr. Molinari. Would you know whether there is any system of monitoring those who lived there who were exposed to this TCDD and are now living in other parts? Are they being monitored? Is their health being checked periodically?

Mrs. Leistner. We are not aware of any monitoring. However, approximately 3 weeks ago at my home in Pacific, at my temporary home, a young man knocked on the door and he held some paperwork in his hand. I invited him in. It was a screening. Well, it was a questionnaire that is being provided by the Missouri Division of Health and CDC, some kind of comparison study. But as far as monitoring the people that have been exposed, I know of no system.

Mr. Molinari. Do you have a record of where the residents of Times Beach have moved to, their present whereabouts?

Mrs. Leistner. Yes, sir, we have a record. But it is incomplete.

Mr. Molinari. Has CDC or any other health authority asked you for a copy of these records so that they could communicate with these people from time to time?

Mrs. Leistner. No, sir, they have not. FEMA has a copy, or I have requested it from FEMA but they won't provide it because it would be a violation of the Privacy Act. But they have a lot of our people in temporary housing, and they have their new addresses. However, not all the people from Times Beach took temporary housing.

Mr. Molinari. So what we are talking about is a substantial number of former residents of Times Beach now living in other parts who may be exhibiting some signs of physical disability that could be attributed to the exposure.

Mrs. Leistner. Yes, sir. I have had people call me from California that moved away from Times Beach approximately 2 years ago telling me of health problems, deformities in children, and heart defects and diabetes. I have had people call me from all over the United States that formerly lived in Times Beach, and what I am telling them to do is to contact the CDC in Atlanta. And then I turn their name and new address over to the Missouri Division of Health.

Mr. Molinari. Has the regional office of the Environmental Protection Agency indicated any desire at any time to do a followup study on these people?

Mrs. Leistner. If they have I am not aware of it.

Mr. Jumper. Mr. Molinari——

Mr. Molinari. Yes.

Mr. Jumper. Could I make one comment about this health survey?

Mr. Molinari. Surely.

Mr. Jumper. When the health survey was first announced everyone in the city thought it was a good idea because of the confusion and fear and that sort of thing. When the first people went down to the health survey, to fill out this long questionnaire and sit down with a caseworker, the only things that they would include on that questionnaire are things that you could verify that you had been treated for by a doctor.

Originally most of the publicity was about this chloracne and skin rashes that were the only outward visible signs of dioxin contamination. Many of the people of Times Beach were poor and got by without going to dermatologists. When they got to the survey, these workers refused to put it down if they couldn't document it. The word got around within a few days that they were not interested in our medical problems, and that kind of messed up the viability of that health screen. Because the word got around that they weren't interested and many, many people refused to go to it.

One elderly gentleman that had a lot of skin problems for the last 5 or 6 years told me, he says, "I'm—I went down there and they didn't even write down, they didn't want to talk about what was wrong with me, so I'm not going." That got around and that caused a lot of the problems.

Another case, the Bothimer family discussed in the mayor's statement, they took their sons to a dermatologist on the recommendation of this health screening, medical people at the health screening. He diagnosed both their twins as having this chloracne, yet they still insist there is no chloracne in Times Beach.

Mr. MOLINARI. Who performed this health screening?

Mr. JUMPER. It was put together by the Center for Disease Control and Missouri Division of Health. There was another problem that came up in the health screening. The doctor put in charge of interpreting these results, Dr. Steven Ayres at St. Louis University Medical School in St. Louis, had testified earlier in 1982 at a trial that dealt with medical concerns from a dioxin spill at Sturgeon, MO. This doctor testified for the chemical companies and for the railroad company that dioxin was not harmful. He was their medical expert as to the lack of toxicity of dioxin. Six months later this doctor was appointed to the health screening to interpret the data for Times Beach at another dioxin site.

This word got around very quickly. People, there is very little credibility to the health screening from the people of Times Beach's viewpoint.

Mr. MOLINARI. In fairness to the other members of the subcommittee, Mr. Chairman, I am going to pass for the time being. But I would like to make the observation that it is rather shocking to hear that with all the notoriety we have heard about Times Beach and with all the various health related agencies that have visited the site, we are learning today that they really overlooked the health aspects. In some cases deliberately so, it seems to me. And to not follow these people and to create a medical history to determine what may occur, I think is inexcusable. We know in some cases these conditions may not show up for years.

I think that this subcommittee is going to try to get some answers from CDC and Missouri Health Department, and whoever else. I will defer now, Mr. Chairman, to the other members of the subcommittee.

Mr. LEVITAS. Thank you very much, Mr. Molinari. I think your observations are well taken.

Dr. Rowland.

Mr. ROWLAND. Thank you, Mr. Chairman.

Mrs. Leistner, I must say I am overwhelmed by the medical problems in Times Beach, MO. It is really almost inexpressible that so many illnesses occurred there. Over what period of time did these illnesses occur? Can you tell me how many years?

Mrs. LEISTNER. The dioxin-contaminated oil was sprayed on the streets in 1972, Mr. Rowland. And most of the illnesses that we have, most of the people that we have been in touch with who now have illnesses that may be dioxin-related started in 1974, 1975. And we even had a diagnosed case of chloracne in 1973.

Mr. ROWLAND. You mentioned problems in young people. One young person had a tumor on which there was still no diagnosis. There was no pathology report from the doctors indicating the type of tumor it was?

Mrs. LEISTNER. The doctor removed one very large fibroid tumor and his remark was there were seven other growths. He said I don't know what they were. I have never seen anything like them. However, I removed them.

Mr. ROWLAND. No pathology report on that, do you know?

Mrs. LEISTNER. No, sir, there were none.

Mr. ROWLAND. A group of 15 homes you said and 7 of the families had seizure disorders?

Mrs. LEISTNER. Yes, sir. In just one block of the community, there were seven seizure disorders.

Mr. ROWLAND. And breast cancer in young women some in their teens?

Mrs. LEISTNER. Yes, sir. A 23-year-old woman a month ago had a breast removed that lived on one of the hottest streets. In this report here, there is a report given by Mr. Jim Curtis, our water commissioner, who is also a minister in the community, of problems in his area of the community alone. He was telling me a month ago a young lady by the name of Rankins had a breast removed at age 23.

Mr. ROWLAND. This has been going on for 6, 7 years. Did insurance companies refuse to provide hospital insurance for the people that lived in Times Beach in view of all this, the problems they were having.

Mrs. LEISTNER. No, sir, not to my knowledge.

Mr. ROWLAND. They still provide insurance. What about life insurance? You would think life insurance with all the problems going on would rate people up that live in Times Beach.

Mrs. LEISTNER. That possibly does now exist. Many people in the community are concerned. And as well as the other dioxin sites, that the possibility exists in the future that because of the exposure to the dioxin they will not be able to acquire health or life insurance.

Mr. ROWLAND. But it has not occurred as of this point as far as you know?

Mrs. LEISTNER. No, sir, not to date.

Mr. ROWLAND. Mr. Chairman, it would certainly be interesting to get some medical reports. This is just overwhelming. I would really like to see some medical reports concerning these people in detail. I would hope that the subcommittee would be able to obtain some of those.

Mr. LEVITAS. Dr. Rowland, specifically I think in light of Mr. Molinari's questions and yours, we will be making specific inquiry of officials at CDC and Missouri health officials to get answers to these questions. In addition to that, tomorrow on the panel we will be hearing from some medical experts on both sides of the issue. And one of them at least was specifically involved in the Times Beach matter. So it may be helpful to ask questions tomorrow.

Mr. ROWLAND. Thank you very much. Thank you for being here all of you.

Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you.

Mr. Wise.

Mr. WISE. No questions, Mr. Chairman.

Mr. LEVITAS. Mr. Albosta?

Mr. ALBOSTA. Thank you, Mr. Chairman.

I want to compliment the mayor on her presentation. I think it was excellent. It reminds me of the time when I was chairman of a special committee in Michigan in the State house of representatives when I was investigating PBB. And it still remains somewhat of a mystery. Still we do not have in the State any agency that would respond quickly to the kinds of problems that you had at Times Beach or another PBB incident. So I just wanted to commend you.

I think anyone that wasn't moved by your testimony would be totally lacking in understanding of the kinds of problems that you have gone through.

Mrs. LEISTNER. I thank you. I might add here to the committee that, since the December 23 announcement of the dioxin contamination in the community and the fact that we have been in this tug of war—some doctors say it is harmful, some doctors say it isn't—the community has mail that has come from all over the United States. We have even been in touch with the Cremer Warner consulting engineering and scientists from the Italy incident. Also many Vietnam veterans have sent literature of all kinds. The city hall is stacked with literature on dioxin.

Of all the literature we have seen, all of them say the dioxin is harmful to your health with the exception of the American Medical Association's report which is attached to my statement.

Mr. ALBOSTA. If I might add to that, I don't think that the American Medical Association as such approved that. I think it was more the Missouri delegation that was in Illinois at that time. Certainly anyone that would look at the results of dioxin on animal tests would have to agree that it is a very toxic substance, particularly the one that is 2378-TCDD. We have some of that up in Michigan. I have worked and now have a study underway that is going to produce some results, I believe, in telling us the true facts about dioxin, how we should control it.

We have proper protocol, I believe, established for that. We are asking an arm of Congress to oversee that particular study. Then we are going to have the National Academy of Science give peer review on the study after it is completed. Recommendations will come out of that. That is not going to help you now in your present condition.

I think it is the responsibility of Congress to move as swiftly as we possibly can to avert and to help in problems such as you people have in Times Beach because you are not the only ones that are involved in this either, by the way. New Jersey has these problems, other places in the country, and I just read in this morning's New York Times that Austria has now shut down its last plant in Western Europe that produced dioxin as a byproduct from chemical manufacture. They have a process of burning that waste out in the North Sea on a ship.

Other areas of the country and world feel that it is a very toxic substance and that something ought to be done about it. Hopefully, your statement will get heard by more than just this committee here today. And that more people will be moved to do something to help people under the kinds of circumstances that you are in.

Mrs. **Leistner**. We in Times Beach feel that whether or not dioxin is harmful to your health because of all the conflicting stories, whether or not it is, the traumatic effect that it has on your life once you have been made aware that you have been exposed to it is overwhelming.

Mr. **Albosta**. I can understand that.

Mr. **Levitas**. Thank you, Mr. Albosta.

Congressman Young.

Mr. **Young**. Mr. Chairman, thank you very much. I just have a brief comment. I appreciate the opportunity to be able to sit with your committee today. I think as your committee, a suggestion I would have as your committee looks at the whole situation, you might want to consider field hearings in the Metropolitan St. Louis area so that you could bring all of the response teams that were suppose to be working. And I would have hoped would have worked a little closer together in the situation as it developed at Times Beach. Maybe your Oversight Committee, investigation and oversight, would like to try to get all those people together in the St. Louis area.

I think you will get a better picture. As I mentioned, this Times Beach and Minker-Stout seems to be a testing ground for EPA response, and FEMA and things like that. I want to compliment you again Mr. Chairman and ranking minority member for your interest in this particular problem.

Thank you.

Mr. **Levitas**. Thank you, Mr. Young.

Mr. Breaux, do you have any questions for this panel?

Mr. **Breaux**. No, Mr. Chairman.

Mr. **Levitas**. Thank you. Again on behalf of all of us and on behalf of the Congress, and I believe on behalf of the American public, you have our appreciation for coming and sharing with us the sad story of Times Beach. There is no doubt in my mind that your testimony here today will make a great contribution to an improved response by the Congress of the United States to deal with this problem, and not just for the future but, if possible, for those of you who are already victimized by this situation.

So I want to thank you and appreciate your being with us today.

Mr. **Albosta**. Mr. Chairman, I think the suggestion Mr. Young had was an excellent one, and would want to support his idea of moving the committee to the area. Let me tell you that what the mayor said about the psychological aspects is a reality. I think this committee could do a lot to help people understand what the problem is simply by having a hearing there and at least alleviate some of their concerns that no one cares. Hopefully we will be able to follow up eventually with proper legislation.

But I am sure with my experiences with the PBB incident in Michigan, the same thing was true. I held public hearings across the State to give people the opportunity to talk to their representatives. I am just sorry to say that nothing legislatively took place from our problems in Michigan to alleviate it. I hope that doesn't happen here. Those hearings would be very useful to help the people in the Missouri area.

Mr. LEVITAS. Thank you very much, Mr. Albosta. We appreciate your participation.

Mrs. LEISTNER. We appreciate the opportunity to be here today and all thank you very much.

Mr. LEVITAS. Thank you. I might say along the lines of Mr. Albosta's statement, I hope that you understand and will on our behalf communicate to the people of Times Beach that you are not forgotten people. We do care. And we will do what we can to deal with this problem, both for the future and the present.

Mrs. LEISTNER. I might say the people of Times Beach have become ardent fans of this committee. They watch you very closely in the newspapers and on television.

Mr. LEVITAS. Thank you.

Mr. MOLINARI. I just have a couple of observations I would like to make, Mayor. First, I want to thank Congressman Young and Congressman Albosta for their concern. This is not the first time that we have had the request to conduct field hearings at Times Beach. It is something we have been trying to do. But I do want to emphasize the fact that we are going to be following up with the Centers for Disease Control about health effects monitoring.

We do know one thing about dioxin. We know it is cumulative in the fatty tissues of the body; it builds up and remains for long periods of time. So it is kind of shocking that there isn't any monitoring. We are going to go into that.

Just one or two quick questions. You mentioned there was no flood insurance available.

Mr. JUMPER. No, sir; the city of Times Beach, through a nonbinding referendum elected to opt out of the Federal Flood Insurance Program because it would basically have shut the town down if we could not grow in any way at all. The people chose not to have flood insurance and most of them were willing to take that chance, live on a river and take the chance of the flood. Many long-time residents had seen one major flood in 1957. You need to understand that the people of Times Beach just weren't worried about the floods. We would have recovered. We would have made it.

Mr. MOLINARI. I just wanted that for the record. How about private insurance?

Mr. JUMPER. There is no private flood insurance available. The Federal Government subsidizes all flood insurance and regulates it.

Mr. MOLINARI. I am not talking about flood now. I am talking about beyond flood damage. Was there any private insurance available?

Mr. JUMPER. No, it is kind of interesting. Shortly after the flood I became a rather public figure in all of this, somewhat regrettably to my wife. But I became very unpopular with a very small group of people that just didn't want to hear anything about anything in the town. And about a week after the flood my home was vandalized. Not only with the flood damage but they broke out all the windows and a lot of other vandalism. My homeowner's insurance refused to pay for it simply because it had been flooded, and they said it was from the flood. Homeowner's insurance was being canceled by the insurance companies until the State division on insurance put a stop to it.

Could I share one final point with the committee here? The international aspect of this publicity I hope won't reflect poorly upon the Federal response. But we received a letter from the Cremer Warner Co. that did the engineering studies about what to do in Italy. They wrote us a letter and said, this man said, "Although I write from several thousand miles away from Times Beach I have been appalled at the press reports on the dioxin disaster that have struck your town. I also read with interest news of the personnel of the Federal Environmental Protection Agency and felt that you and your colleagues could well need alternative expert advice concerning this dioxin problem."

The Federal response has been good. We appreciate the fact that they are going to pay us for our real estate. But what we would really like to see, most of the people would have liked to have seen the town relocated like it says in the Superfund law. But I think it needs to be looked at, too.

Mr. MOLINARI. One last question. Business insurance. Were there any insurance proceeds for business to pay for interruptions?

Mr. JUMPER. I was overinsured as a contractor. I had every kind of insurance there is, but I think they used the flood as an excuse and refused. In one instance I had a big pile of material, homebuilding materials that floated away and knocked another man's home down completely. They refused to pay for it complete.

Mr. MOLINARI. I wasn't asking about you only. Was there any kind of insurance anywhere?

Mr. JUMPER. No.

Mr. STONE. There was 347 mobile homes in the park. Mobile homes in the State of Missouri are treated as personal property. About 85 percent of those people did have flood insurance.

Mrs. LEISTNER. It was covered under the comprehensible part of your policy.

Mr. MOLINARI. So they did effectuate some kind of recovery?

Mr. STONE. True.

Mr. MOLINARI. Thank you very much.

No further questions, Mr. Chairman.

Mr. LEVITAS. Something you said just now triggered a question in my mind. The $33 million figure which was contributed by Superfund, do you know how that $33 million figure was arrived at?

Mrs. LEISTNER. I might just probably guess on that. It has never been confirmed. But at the onset of this we submitted this folder to the President of the United States. In the folder you will see the businesses of Times Beach, their property value and their gross yearly income. Then we were told that they estimated each home at $40,000 and somehow arrived at this figure.

Mr. JUMPER. I might add when I came to Washington for the one meeting we had here on President Reagan's Times Beach Task Force, they asked me what I thought it would cost to purchase the real estate in Times Beach. I told them somewhere around $35 million. That is very close to what they have come up with.

Mr. LEVITAS. In other words, the $33 million that has been committed from the Superfund is for the items you have just described. It doesn't have anything to do with a cleanup of the problem that is there.

Mrs. LEISTNER. No, sir, it does not.

Mr. LEVITAS. So it doesn't even address that, nor does it provide any compensation for those other health matters, personal property, loss of income.

Mrs. LEISTNER. No, sir.

Mr. LEVITAS. It is just as we both said, the tip of the iceberg.

Mrs. LEISTNER. Correct.

Mr. LEVITAS. Congressman Young, you wanted to add something.

Mrs. LEISTNER. Congressman Young was just telling me that he was responsible for that figure. They took 800 homes at $40,000 each, and that is how they arrived at the $33 million. $32 million.

Mr. YOUNG. That is not an expertise as you well know, Mr. Chairman. But it was confirmed by some other agency after my office, I asked them early in December to try to come up with a figure of what a buyout of the whole town would cost as far as the real estate. They took the 800 homes and worked out about a $40,000 figure. Somebody else reiterated that figure later in January.

Mr. LEVITAS. I see.

Mrs. LEISTNER. We were advised it was because of this report. Then the balance of the homes, because 337 of those homes were mobile homes and 85 percent of those people were covered by some insurance.

Mr. LEVITAS. Still, it just doesn't deal with the problem that still is there on the ground, nor does it fully compensate or make whole, as you pointed out.

Mrs. LEISTNER. No, sir, it does not.

Mr. LEVITAS. Thank you very much. We appreciate your being here.

Mrs. LEISTNER. Thank you.

Mr. LEVITAS. The next panel of witnesses, if they would come to the witness table, consists of Shirley and Ronald Payne of Pacific, MO, Rita Jones of Staten Island, NY, John Hollingsworth and Drew Rainer of Lake Charles, LA. I want to express my heartfelt appreciation to each of the witnesses who are appearing before us today.

Also, I want to welcome a Member of Congress, a colleague who is here in the room today who has asked to say a few words of introduction about the people who are testifying this morning, and with respect to the issues that this subcommittee is attempting to address.

We will begin with Congressman Gephardt, who has been here and listened to the earlier testimony. We appreciate your participation.

TESTIMONY OF HON. RICHARD A. GEPHARDT, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF MISSOURI

Mr. GEPHARDT. Thank you, Mr. Chairman, members of the committee. I really appreciate your continuing attention to this problem as it presents itself in Missouri and across the country, and believe that the faith of our citizens in the Government is immensely helped by the work of this subcommittee, in trying to understand the problem and trying to fashion solutions to the problem.

I think the people that are here today from Missouri who are constituents of mine, Shirley and Ronald Payne, bring to us a very important message regarding what is obviously a pressing and difficult problem, and one that will be with us for decades to come. I was interested in your trying to figure out what the costs are going to be. When I just look at the costs down the line of the total Missouri problem with just dioxin, I see figures that would exhaust the Superfund with Missouri alone, if present technology is as expensive as I think it is in dealing with doing something with the dioxin.

The message this panel brings to us is that the past practices of dumping of our hazardous wastes in a haphazard way has some very real side effects. It impacts the health and welfare of tens of thousands of our fellow citizens, if not more.

Mr. Chairman, I have only taken a deep interest in this issue during the last year. While Love Canal was a tragedy, I guess in Missouri we never thought that it would happen to us. However, it has and in recent months we have found numerous hazardous waste sites throughout the country with many more still to be discovered.

In my district alone there are several sites that are contaminated with the chemical dioxin. Throughout the State of Missouri more than 30 sites have been uncovered in the past year with another 100 or more sites still under investigation. All of these sites are the result of haphazard dumping by only one waste disposer; who knows what else lies within the confines of my State and others.

Although we have been able to get some action on a few of the sites within our State, that has only been through constant efforts by the entire Missouri delegation. At some point the present system which we have set up to address these problems will not be able to cope. Already strains are being felt.

One of the major problems that we have is that the science on the chronic health effects of many of our wastes is not clear. On what is known as the Minker-Stout site in my district, the EPA finally offered permanent relocation assistance to 11 families. Yet, families living just a few feet away from levels well in excess of the acceptable level which the Centers for Disease Control has adopted will receive no assistance at all. These families are forced to live just a few feet away from a very grave risk and, in my mind, there is no doubt that they are being subjected to undue health risks.

At a meeting in my district earlier this year several residents asked the officials involved in making the decisions on this issue whether they would live there. To a man, each official said that if it was them, they wouldn't live across the road from the Minker-Stout site. I might say parenthetically that the Payne family, until about a year ago, lived across the street from the Minker-Stout site. What you are going to hear today are the physical problems they experienced living 20 feet from the high levels of dioxin. That is why I have been asking EPA to do something about the families that have lived in the very near proximity of this site.

We have asked that a "zone of safety" be created around the site to guard against the risk several years from now, upon finding out that dioxin is much more toxic than we had once thought, that we had made the wrong decision. The cost of relocating just a few more families is minimal in relation to the eventual health problems they might incur.

I know that you and the members of your subcommittee have been addressing this problem for some time and are much more experienced in this area than I. However, I would like to offer an idea which I think will address some of the problems arising out of our numerous waste sites. A system such as the national flood insurance system in scope could be set up to address the long-term health problems coincident with our waste sites. Premiums could be paid through a variety of sources: the Superfund, HHS, and the States in which a site is located.

The residents at and near these sites, within a "zone of safety" would then be afforded long-term health care for their problems arising out of the site. To address the problem of how to measure a "zone of safety," a concept similar to that used by French electric companies in charging for electrical service might be applied. The closer an individual lives to a nuclear powerplant, the cheaper the cost of their electricity. Thus, as an individual lives farther away from the site, they may be asked to pay a portion of the premium out of their pockets.

I don't know if our Nation can financially cope with compensating individuals with money damages for the damage they have incurred. We can, however, ensure that their long-term health problems are addressed.

I apologize for taking up so much of the subcommittee's time. I am going to have to run out the door for a hearing with my own committee, but I appreciate having my constituents and other constituents from around the country here to better understand their problems.

Mr. Levitas. Thank you very much, Dick, for your statement and for introducing the Paynes to the subcommittee today.

Your suggestions are very provocative and I think should be considered as one of a number of possible ways of responding to this cause, you are quite correct. Just based on what we know about the cleanup, forgetting everything else, just the cleanup, the Superfund will not clean up half the priority sites already identified, 419, and that represents a fraction of the sites we already know about each day.

And each day there is a new one.

As I pointed out this morning, the headline in the Atlanta Constitution was the discovery of an abandoned hazardous waste site that apparently is leaching into the soil.

Your suggestion is one we certainly will consider. I appreciate your being here.

Mr. **Gephardt**. Thank you.

Mr. **Levitas**. I think we will introduce all the witnesses and then let them speak as a panel. So at this point I would like to now call upon our colleague and member of the subcommittee, Congressman Breaux, who has constituents here today, but also because Congressman Breaux has truly been one of the leaders in the Congress in dealing with this matter of Superfund and hazardous waste sites.

As I recall, Congressman Breaux managed Superfund legislation when it came through this committee and has recently introduced other legislation that is extremely important. I will let him talk about it if he so chooses. But he has truly been one of the people in Congress who early on saw the problem and for a long time was a voice in the wilderness.

I am grateful for his continuing interest and participation today. I recognize the gentleman from Louisiana.

TESTIMONY OF HON. JOHN BREAUX, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF LOUISIANA

Mr. **Breaux**. Thank you very much, Mr. Chairman. Also thank you for your very generous words. I know some people think I am still in the wilderness, trying to come out, but you are correct in the sense that when we did first draft Superfund legislation, the real goal at that time was to try and find a quick way of addressing the cleanup problems we had in this country.

Quite frankly one of the missions at that time was the failure of the Superfund to include any kind of compensation system within the structure of the fund for victims of these toxic waste sites throughout the United States.

I would commend the chairman and members of this subcommittee for addressing the serious problem of—after we clean up the sites—what do we do for the people who have suffered injuries who have no other recourse to turn to as far as getting their injuries taken care of.

I think one thing we have been learning, Mr. Chairman, is, while we have been focusing the last several years on toxic waste and EPA problems, one thing we have, I think, found very clearly is that most of the problems that we are finding in the United States are coming not from illegal dumpers who go around at midnight and dump toxic wastes in ditches and canals, but most of our problems are coming from legally permitted waste sites.

Companies have gotten a permit from the Federal Government. We in the Federal Government have given them a permit and we have said it is all right to go dump in this particular place, and those are the type of dumps causing the injuries we are hearing about in this country.

It means we need to change the way we have been doing things. I have introduced legislation and will be offering that bill as an amendment next week on the House floor, which would eliminate all land disposal of liquid hazardous waste.

I feel very certain that unless we set some standards and deadlines, companies are going to continue to use the Earth's surface as a means of dumping liquid toxic waste.

We have found no matter how safe people tell us it is, it has not been safe. We have people like the witnesses today who are up here telling us some real horror stories about problems that have occurred many times from a waste site that we in the Government have said it is all right to put there. That is certainly not acceptable.

The interesting argument that some people make is that, well, it is going to cost too much if we can't dump it in the ground.

I would only submit that if we look at the costs these people have incurred, if we look at the fact we have had to buy up an entire city like Times Beach, if we look at the destruction of natural resources and look at the loss, look at the emotional and mental problems people are having as a result, if you put a bottom line on a cost figure for what that would cost, I would dare say alternatives that are available today are a lot cheaper in the long run than the method we are using right now.

So, Mr. Chairman, I think that you are moving in the right direction towards compensation as part of a Superfund because, without it, these people are going to have no place to turn.

I would just in the way of introduction, we are very pleased to have two people from my district, Lake Charles, LA. Mr. Drew Ranier is a prominent attorney, a good friend of mine from Lake Charles, who not only is involved with representing Mr. Hollingsworth this morning in the claims for personal injuries, but has also been a leader in the movement.

That is not something he has taken up just because of this incident, but is a real leader in the movement in southwest Louisiana to try and bring about rational policy toward regulating hazardous waste. It is those types of citizen movements that are really going to, I think, ultimately get us to do what is right. We are pleased to have both Mr. Hollingsworth and Drew here this morning. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you, John.

The next person I want to recognize is the ranking member of this subcommittee, Congressman Guy Molinari of New York.

Let me say publicly that Guy Molinari is personally responsible for the entire investigation into the EPA Superfund program and hazardous waste. It is almost 2 years ago that he first called the matter to my attention.

At his persistent urging, I went to Staten Island to see for myself the problems of hazardous waste disposal as it exists in the Metropolitan New York City area. I was so shocked by what I saw on that occasion that I promptly ordered the commencement of hearings which began really at the end of 1981. What has happened since then is commonly known history.

So all of us owe Congressman Molinari a great vote of thanks, not only for his continued interest, but I think very importantly and significantly the fact that he was the one who was the cause of getting this whole situation under the spotlight of public scrutiny.

It proves to me that one person can make a difference, and a big difference.

Congressman Molinari, you are recognized for purposes of introducing your constituents, and if we have some hard questions we will ask you.

Mr. Molinari. Well, I am just about speechless after those kind remarks, Mr. Chairman. I appreciate it. While we are throwing bouquets around, it should be recognized that without a chairman who was responsive we wouldn't have been able to accomplish what we have accomplished.

I would also like to pay tribute to Mr. Breaux, because the subject we are discussing today, victims compensation, was included in his original legislation. If he had his way, and supporters of his legislation, it would be law today. Unfortunately my understanding is that it was stopped in the Senate.

Mr. Chairman, I would like to introduce the next witness, the lady on my left, Mrs. Rita Jones. Mrs. Jones is one of the founding members of a Staten Island community group called IRATE, a group formed after it was revealed that toxic waste dumping had taken place on a large scale in the Brookfield Landfill. This is a New York City garbage dump about a block from her home.

Her testimony, Mr. Chairman, will be a classic illustration of what happens to a family living near a toxic dump. It will be a presentation filled with the frustration and despair that perfectly innocent people face when they are confronted with a maze of agencies and bureaucracies that seem indifferent at best, callous at worst. There is a Rita Jones in every congressional district in this country, Mr. Chairman, because the problem of toxic waste dumping affects every district in the Nation. Her story is one of individual tragedy but is also one being played out in thousands of homes across the Nation. In small towns, rural hamlets and sprawling cities, people like Mrs. Jones are experiencing the same fears and frustrations you are about to hear.

She is representing not only herself but thousands of families in every State who are trapped and asking for help. Mr. Chairman, there are many important issues in this country that affect people in any number of ways. But few issues are as tragic as that of the families that are caught up in the web of toxic waste dumps.

It strikes on things they care about most, their children, their health and their homes. And Mr. Chairman, if I may, just so you and the other members of the subcommittee can have a perspective as to what she is going to talk about, we do have two blown up pictures here showing the residential community where Mrs. Jones lives. In the foreground is the Brookfield dump. The pictures show a ditch that is 100 feet wide, 30-some-odd feet down, that was dug through a municipal landfill.

Testimony in a Federal court and in the New York State Supreme Court indicated that there was dumped illegally between 3 and 10 million gallons of toxic chemicals. Interestingly enough, Mr. Chairman, they were dumped by tank trailers which are not legally allowed in a New York City garbage dump. So we had five tank trailers a night for a year and a half during the week, and usually nine on a Saturday and Sunday night, going in and out of that dump.

There have been people put in jail as a result. There were payoffs, corruption—government at its worst. But here you have the residential community a stone's throw away. From the dump to the street is about 200 feet. And this young woman is going to speak for herself and for the families and friends in her neighborhood, Mr. Chairman.

I know she is a little nervous about being here today, but I know her message is an important one and I thank her very much.

Mr. LEVITAS. Thank you, Congressman Molinari.

Before we hear from the panel I observe there is a recorded quorum call taking place on the floor of the House. Rather than unduly interrupt the presentations of the panel of witnesses I would suggest that we briefly recess and then return without delay to commence their testimony. I will say that during the course of the proceedings on the floor today there may be other votes. It is just the way business works around here. From time to time we will have to take recesses of this sort. I hope they would not be unduly intrusive. Every time I say that I am proven wrong, but we will do the best we can, and appreciate your indulgence.

The subcommittee will stand in recess for 10 minutes.

[Recess.]

Mr. LEVITAS. The subcommittee will please come to order.

At this time I would like to welcome all of the witnesses at the witness table today. As I said, we are very appreciative for your making the effort, as well as the personal sacrifice to be with us today, and to share with us your experiences so that we might better understand the dimensions of this problem as it affects human beings and thereby be better able to respond.

So with those few words I would first like to ask Mr. and Mrs. Payne if they would proceed.

TESTIMONY OF SHIRLEY PAYNE AND RONALD PAYNE, PACIFIC, MO

Mrs. PAYNE. I would like to thank you all for inviting my husband and me here today. At first, when I was asked to appear, I was hesitant because I had never spoken before a group as large as this and because I had never even been on an airplane.

But, Mr. Chairman, I began to think—my family has experienced many health problems which, I believe, are associated with having lived across the street from the Minker dioxin site. If, by telling our story, it will help us, our friends and acquaintances, and those who are or will be in the same situation, then it is worth it all. And that is why we are here today.

In June 1975 we bought a house on 4038 West Rock Creek Road in Imperial, MO. We lived there 6½ years until we moved out in November 1981.

Our house was located across the street from property owned by Harold Minker. It was on the Minker site that dioxin-contaminated dirt from the Bubbling Springs horse stable was dumped.

Being across the street from our home, it was also where my four children waited for the school bus and played. For 6½ years we breathed the contaminated dirt and dust from the Minker property.

As I mentioned earlier, I believe our family health problems have been a direct result of our exposure to dioxin. I would like to tell you about these. In doing so, I have tried to recall the exact time to the best of my recollection. In any case, I have some of our hospital records with me today, and I would be happy to submit them for the record.

Beginning about 1978, I began experiencing respiratory trouble. On three occasions, I couldn't hardly breathe, and Ronny had to rush me to the emergency room at St. Joseph Hospital in Kirkwood. I visited the doctor's office several times for this, and was given shots and medication for 7 or 8 weeks.

Since 1978, I have also had constant drainage of the nasal and throat area, so much so that the only way I can sleep many a night is sitting straight up in a recliner, as my doctor requested that I do.

In 1978, I was hospitalized for poor circulation and tested at St. Joseph Hospital for the possibility of blood clots in my legs. I spent 10 days there at that time. The doctors could not determine the cause of the swelling or the lack of circulation.

In 1982, I went to St. Joseph Hospital emergency room for a bleeding kidney and bladder problems and was placed on medication. During that year I was also treated for sores that left scars. The doctor said he had no idea and no answer for them.

In 1983, I was tested by Dr. Fisher from CDC. He told me to have an echocardiogram, and I was detected to have high blood pressure and an extremely nervous condition, which, he said, I should have checked. Also, Dr. Sage of CDC took immunity tests in our home and immediately he was concerned about how my tests reacted and was at my house the next morning to look at my arm. He was concerned by the way it looked. Not all of our results of the other CDC tests taken in February 1983, liver and immunity tests, are back yet.

My husband, Ron, has also had a number of problems. On November 1, 1981, he experienced swelling of the face, lips and eyes which lasted for 2 or 3 months. His eyes were at times swollen shut. He went to the hospital and doctor's office a number of times for this. At first we thought it was allergies. He got shots and we checked everything in the house—furniture, everything. Eventually the swelling went away, but one day he went back to the Minker site and before we got home, he noticed a rash coming on his arms, face, and neck.

In 1982, he completely lost his voice for over 5 weeks and was still hoarse for about 3 months after. He has nasal and throat drainage that keeps his throat sore, and bronchial troubles which medication does not seem to help.

In May of this year, he was admitted to Normandy South Hospital with severe headaches. No medication would relieve the pain. They ran all kinds of tests and diagnosed his condition as cluster headaches, for which he is now on medication and oxygen. In fact, we have an oxygen tank in our home, and whenever he gets these headaches, whether at work or someplace else, he has to come home and take oxygen for 15 to 20 minutes. Nothing else will relieve the pain.

In addition, my four children have experienced numerous problems. Ron, Jr., who was 17 years old when we moved across from the Minker site, has had respiratory trouble, severe stomach pains and numbness of the entire body. He visited St. Joseph Hospital twice in 1979, and once in 1980. Like the rest of us, he has repeated throat infections and colds. Medicine does not seem to help. I also want to add that his child, a baby of 8 months, already is experiencing severe immunity problems. He can't seem to fight colds, and all of us are very concerned about his fragile state. He has been to the doctor's office eight times already and taken to the hospital twice. Before he finishes his medication, he has got another virus, or bronchial problem. That is just the way it is.

I have one daughter, Kathy, who was 11 years old at the time we moved in. Kathy has had her nose cauterized for nose bleeds, but the past several years her nose has bled more often and lasted much longer than before. She will just be sitting and her nose will start to gush.

In 1978, she was diagnosed as having an enlarged pulmonary artery. She still has severe chest pains and shortness of breath.

In December 1982, she was rushed to the hospital because of severe stomach cramps, admitted and placed immediately on IV's for a 4-day period, and diagnosed as having hemorrhagic cystitis and influenza. Five months later, she again suffered severe stomach pains and this time her M.D. sent her to a specialist, and she was diagnosed to have hemorrhagic cystitis again. She is still on medication and being treated. A week and a half after she came out of the hospital for hemorrhagic cystitis, she came down with strep throat, even though she was still on antibiotics.

I am concerned about her weight loss. Since her hospital stay in December, she has gone from 130 pounds to 116.

This past February, she was tested by CDC and hasn't got all the results back yet. She was seen by Dr. De Val from CDC for growths on her face and neck. The medication did remove most of them, but now they are coming back. She is allergic to penicillin, Keslin, Keflex and IVP dye. She also shows reaction to insect bites. She has had her share of sore throats, too.

My son, Ricky, who was 4 years old at the time we moved in, has had throat infections, and it got to the point that medication would not clear up these infections. So, on April 14, 1980, Normany Hospital removed his tonsils. Then, 8 days later, at 3:30 a.m., he started vomiting blood and we rushed him to the emergency room. They removed four blood clots the size of grapes from his throat. In August of that same year, 11 teeth had to be removed for unknown reasons, although he had had regular checkups at the dentist.

In 1980, he was diagnosed by Dr. Spain as being anemic, for which he is still on medication and has blood tests run every 6 months. In 1980, Ricky's teacher at school complained about his nervous condition. She suggested he be seen by a doctor, which I did. He put him on nerve medication. He has also had stomach problems and headaches.

My last son, Chad, who was 10 months old——

Mr. LEVITAS. Just take your time.

Mrs. PAYNE. My last son, Chad, who was 10 months old at the time we moved in, has had health problems, also. He had severe stomach pains, diarrhea and vomiting. His doctor was concerned and sent him to Cardinal Glennon Children's Hospital for further testing. They did surgery on him in October 1980 and removed three growths from his small intestine. He still suffers severe stomach pains, diarrhea, and is often sick to his stomach.

In September 1982, he was taken to the hospital for respiratory problems, where x rays revealed signs of enlargement of the heart, expanded lungs, and acute bronchitis. He has experienced sores frequently on his upper eyelids. I have talked to the doctor about this condition and he has prescribed medication.

Mr. Chairman and members of the committee, our family has gone through a lot. It seems that at one time or another, one of us is sick. I am always running off to the doctor, and it has been very frustrating. We have seen many doctors and specialists, but each time we ask what is causing this, they don't know. As I noted to you earlier, the final results of all the CDC tests are not known as yet, but I would point out that we received a letter from the State health department which indicates that there is a strong possibility that dioxin is the cause of our problems.

I am concerned about my family and about my friends and neighbors, who have all experienced similar problems.

Again, as I said in the beginning of my statement, we moved from our home near the Minker site in November 1981. Since this was before the dioxin issue became public, we were fortunate enough to be able to sell our house without a loss. However, it has not been fortunate for the people who bought our house. First of all, they have a 6-year-old son who was born with spina bifida. He is now living on one kidney. They are very concerned, living 80 feet from the contaminated dirt, about his condition, which is getting worse. Second, even though they would like to sell their house because of the dioxin issue, it is almost impossible.

Murray Abrahamson, who lived next door to us, has been checked by CDC and his results came back as having extremely high cholesterol. His reading from CDC was 416, when a normal reading should have been around 250. Also, his triglycerides were extremely high at 2,140 when a normal reading should have been around 150. He is under constant doctor's care as is his son Duane, who has experienced severe headaches, nausea, and stomach pains, and his wife, Shirley, who, in the late 1970's, started having headaches and numbness of the hands, face, and feet.

Even though we haven't experienced property losses, to the best of our calculation the last 6½ years have cost us at least $25,000 in medical expenses not covered by health insurance.

Finally, I am concerned about the other people who still live near and around the Minker site. The contaminated dirt is still there; there are no fences and no warning signs. Some of the houses on the site are being bought out, but those across the street, like my former residence, won't be because EPA is using the street as the cutoff point.

Mr. Chairman, this is our story and I appreciate the opportunity to tell it.

Mr. LEVITAS. Thank you very much, Mrs. Payne, for sharing this with us. I know it is very painful for you to have to recount it. But as I said earlier, what you are saying today makes a great deal of difference in giving us a real understanding just what people have experienced in terms of health and economic loss and psychological pain.

We will have some questions in a few minutes. But at this time I would like to ask Mrs. Jones if she would share her experiences with us.

TESTIMONY OF RITA JONES

Mrs. JONES. Mr. Chairman, members of the committee and staff, my name is Rita Jones. I want to thank you for inviting me to testify before your committee. Frankly, I never dreamed that I would ever be in this position.

I was born and raised in the Flatbush section of Brooklyn, NY. It is a crowded urban area—an area where I did not wish to raise my children. I wanted my own house with a little lawn on which my children could play. I wanted to belong to a pleasant community of friends and neighbors—a community that was safe, clean, and enjoyable. So, in June 1977, when my daughter Dana was 6 and my daughter Stephanie was 3, we moved to 112 East Brandis Avenue on Staten Island. In short, I thought I bought the American dream.

We moved into this area because it was a new development consisting of young families such as mine; because the area was open and green; and, in contrast with my city experience, looked and felt like heaven on Earth. As I recall that time and the pleasant anticipation of a new life we all experienced, and I compare it to the problems and uncertainties over our health with which we have been forced to live, I cannot help but feel a sense of despair.

I wanted to bring my children out of the crowded, dirty urban sprawl so that they could grow up in a healthier environment. Instead I have two children who suffer recurrent upper respiratory infections, high fevers, nose bleeds and now asthma.

One of the more pleasant aspects of living in a small community is the daily meeting of mothers waiting with their children for the school bus. The conversations during these times naturally center upon the children and the family. It was during these chats in the month of January 1982 that I and other mothers in the community discovered that our children were getting ill at an alarming rate and that they were all experiencing much the same illnesses—skin rashes, nose bleedings, unexplainable bouts with high fevers, and increased asthmatic attacks.

It was also in January 1982 that the city had started excavating a trench through the Brookfield dump. It is quite a trench, measuring about 100 feet across and tapering down in a "V" shape. It extends roughly 30 feet below ground level. For your convenience, I have brought a number of photographs of the Brookfield dump area showing the trench and its proximity to my community.

At first no one thought that there might be a relationship between the digging in the dump and our illnesses. It wasn't until January 15 that we put the two events together. On that day our neighborhood was literally attacked by an odor so strong that no one could go outdoors without immediately being seized by a fit of coughing and gagging. Even indoors the stench was so overpowering that many of us gagged and retched.

I called my neighbors, and we compared notes on what was happening in each household. What we discovered was startling. Beyond the symptoms of gagging, beyond the physical vomiting that was affecting almost everyone, the other mystifying illnesses that had recently appeared were occurring in full force. Children and adults together were suffering from eye irritations and skin irritations. One child had developed a fever and a number of children were experiencing asthmatic symptoms.

Together, over our phone network, we pieced together these individual events and came to the realization that the Brookfield dump was probably causing our problems.

In 1980 during a trial in Pennsylvania, a man named Russell Mahler testified that truckloads of toxic wastes had been dumped into the Brookfield dump. The news of this illegal dumping was uncovered by the Staten Island Advance and a story about this toxic waste dumping was published just a few days before the odor attack of January 15.

On January 15, someone mentioned the illegal dumping and wondered out loud if our neighborhood was being exposed to toxic chemicals. I can assure you that nothing I have ever experienced in all my life has ever frightened me so much as the thought that what I and my children were experiencing was not being caused by nature, but was a result of something being carried on the air into my home to attack us.

Fear set in very quickly, but fortunately, in our case, it propelled us into action. I called every local, State, and Federal agency, every elected official I could think of, the Health Department, the environmental agencies, the Interstate Sanitation Commission, my councilmen, assemblymen, and my Congressman. Every one of these agencies responded in a disinterested fashion. They took the information and promised to investigate. Some of the agencies went so far as to tell me that they were aware of the odors that were coming from the excavation in the dump and suggested that we should be patient and wait for it to go away.

No one wanted to hear anything about toxic waste dumping or the possibility of our illnesses being a result of exposure to toxins. No one except Congressman Molinari. Within an hour after I spoke to the Congressman, there were three cars, one van, and seven men representing various city, State, and Federal agencies, all running around my neighborhood taking all kinds of measurements. But, after this brief flurry of activity, everyone disappeared, and no one was willing to talk to us and help answer our questions concerning the possibility of our whole community being exposed to toxic chemicals.

In fact, the sanitation commissioner publicly called Congressman Molinari an alarmist for expressing concern over the possible level of contamination at the Brookfield dump. In an effort to establish the cause of our illnesses, Congressman Molinari arranged to have nine women tested at our local hospital.

The results of our blood tests showed that three of us, myself included, had high white blood cell counts. One woman had problems with her liver. The doctors were puzzled as to the cause of these results, and while they could not say with certainty that we had been exposed to something in the air, they agreed that toxic exposure could produce our results.

We continued making phone calls seeking help in determining whether the illnesses we suffered from were a result of the trench construction. It is only one block away, and each day hundreds of cubic yards of possibly toxic dirt were dug up and tossed onto a mound.

Finally, Dr. Stephen Schultz, an epidemiologist from the city health department, visited the neighborhood to survey the area.

After visiting the area and interviewing me and others, Dr. Schultz said that he was concerned about our health. He said that, he was less than confident about the air samples taken by the EPA and not sure he agreed with their findings. He said that at that point no one should be saying that there were no other dangerous toxic substances being emitted.

We didn't know whether to laugh or cry when Dr. Schultz made his statements. Here was a member of the New York City Health Department saying to us that there was a chance that we were indeed being exposed to toxic material. I, at least, believed that now our officials who had held back waiting for some evidence would do something to help us. Nothing happened.

In March 1982, 2 months after these illnesses started, a group of my neighbors and I met with City Sanitation Commissioner Norman Steisel. We pleaded for his help. Mr. Steisel assured us that the department would look into the matter, but cautioned us to remember that the toxic dumping was only alleged. We all pointed out that the neighborhood was experiencing unexplainable episodes of dizziness, nausea, high fevers, and chest irritation. Our pleas fell upon deaf ears. Commissioner Steisel, after listening for roughly 45 minutes, asked to be excused so that he could attend another meeting.

On April 1, 1982, desperate to make our plight known, some 300 people from the Brookfield dump area staged a demonstration to force a halt to the sewer construction.

We marched at the entrance to the dump. We were all determined to get some help. We are not professional pickets. This was the first time any of us had done something like this. The police, though sympathetic, were instructed to be firm. They warned us that they would jail any and all of us if we overstepped our given boundaries. We felt alone and rejected. We were being treated as if we were the wrongdoers, we were the criminals by daring to demand action. I remember thinking that none of this made any sense. We had done nothing more than to ask those whose duty it was to protect our health to perform their duties.

The demonstration had a side effect. It caused further illnesses. Children who were perfectly normal prior to entering the dump started to develop rashes on their faces. Many people, myself included, suffered from severe eye irritation. People were coughing and gagging. Some developed severe headaches and had to leave the dump. The appearance of these illnesses while we were at the dumpsite only served to strengthen our belief that toxic chemicals were buried within the dump.

In an act of final desperation, I and three other women hired an attorney who issued a restraining order against any further work in the dump.

Four days after that order was issued, while we were in court, the city agreed to work with the residents to help solve the problems of Brookfield. They have never lived up to their agreement. In fact, before the day ended, truck traffic and construction was back to normal, and we were faced with the difficult task of trying to raise the money necessary to post a bond in order to enforce our injunction. The city knew that we would not be able to raise the required sum and it just violated our agreement.

By August 1982, almost 8 months after the onslaught of our illnesses, we were still at square 1. Dr. Saul Blumenthal, head of New York City's Bureau of Biostatistics, had examined the mortality rates for our area for the previous 6-year period and had determined that there was no indication of any additional mortality in that time period. Dr. Blumenthal said the odors may be aesthetically unpleasing but they weren't killers. I can only add an amen to Dr. Blumenthal's high-handed remark. It was bad enough that the dump was causing illness. Who needed to have people die.

Dr. Blumenthal's statement, however, represents the attitude typical of our supposedly responsible officials. It seemed that everyone we spoke to wanted a body count before they would do anything.

In his continuing effort to help, Congressman Molinari assigned to his staff the task of performing a health survey among the area residents to try and determine the extent and frequency of our illnesses. The health department, shamed by this action, announced that they would also perform a survey.

Then, during the summer of 1982, we learned that the alleged dumping had, indeed, taken place. That, in fact, anywhere from 3 to 8 million gallons of liquid wastes were sent by a company called Hudson Oil, or Quanta, and were dumped at Brookfield over the 2-year period from 1977 to 1979. I understand that this company is notorious for illegally disposing of toxic wastes.

Also, during the summer, the State of New York rated the level of toxicity of Brookfield and assigned to it a toxicity level of 80 points on a scale of 0 to 100. We again accepted these revelations with mixed feelings. On the one hand the true nature of the Brookfield threat was finally being exposed—on the other hand it was like the voice of doom. I could see my dreams crumbling. My oldest daughter was developing a chronic wheeze, my youngest kept getting sore throats and nose bleeds, and I constantly suffered from dizziness, nausea, and a slight but noticeable loss of equilibrium.

In October 1982, Congressman Molinari presented the preliminary findings of his health survey. The city health department issued their findings that November. Both surveys found that the Brookfield area residents experienced an illness rate twice that of any other area on Staten Island.

Finally, I thought, we have everything necessary to get direct swift action. Brookfield dump has now been shown to contain hundreds of thousands of gallons of toxic waste, the land has been disturbed through construction, and now the health department agrees that people have been getting sick as a result of being exposed to this construction. I truly thought our problems were at last over.

On November 5, 1982, almost a year after our troubles began, we called upon the mayor of New York City to come to Staten Island, examine the evidence his agencies had accumulated, listen to our problems and help us solve them. The mayor refused. He responded to our pleas by saying that he was not going to come to Staten Island to be badgered by Staten Island housewives. He had commissioners to deal with these problems.

Now, almost 18 months have passed. I am exhausted. We have held demonstrations. We have gone to court. We have met with every agency and every official that would meet with us. We have been labeled as hysterical, frustrated housewives by the department of sanitation. The health department wants dead bodies before it will count our complaints as valid. Our illnesses have been branded as hysterical responses based on an overactive imagination.

We need your help. I believe that the illnesses that I and my family suffer from are directly related to the digging of that huge trench in the Brookfield dump. I believe that the opening of this trench released those toxic materials into the air causing the whole community to suffer. And I am convinced that the continued ill health of the entire community is a direct result of the callous disinterest of my local and State officials and the agencies they manage, coupled with their own inability to handle the problem of toxic waste dumping. I think the problem is too big to be handled by the city, or even the State.

Eighteen months is too long to wait for the pittance of help we have received. I don't know how you can get what needs to be done accomplished, but if I could make a request of you, I would ask that you assure me and others who may be in my situation that these never-ending investigations will be shortened. That whenever, as in our case, there exists reasonable evidence that the public's health is being affected, action be taken first and proof positive determined afterwards.

I thank you for your time and patience. I once bought the American dream. It has been snuffed out. I come here today in the hope that you may be able to help me once again to rekindle that dream.

Thank you.

Mr. Levitas. Thank you very much, Mrs. Jones, for that eloquent statement. Given the trail of tears and frustration that you describe, I am surprised that your anger is not even more manifest. Just hearing what you said has gotten me even more angry just to hear the story. And you have lived through it.

Next, I would like to ask Mr. Ranier and Mr. Hollingsworth if they would offer us their contribution this morning. We are very appreciative of your presence here today and look forward to hearing from you.

Mr. Hollingsworth.

TESTIMONY OF JOHN HOLLINGSWORTH, LAKE CHARLES, LA

Mr. HOLLINGSWORTH. Yes; my name is John Hollingsworth. I am from West Lake, LA. I am 23. I have two small children I am trying to raise with no income because of an accident that happened at this BFI landfill site.

I had been working there for 3 years. I started having back trouble, and I was put in a hospital for a few days. They told me I had two broken vertebras. They turned me loose. I went back to work. I worked for a few weeks, and it started—the pains got bad enough where I couldn't work, and I went back to the hospital.

And they put me in—run tests, and I must have went out for awhile. I come to in a few weeks, and they had took part of my back, cut them out because of infection which they don't know what it was. And I stayed in intensive care for 2 months. I was in the hospital for 3 months altogether. And I am trying to raise my family off of nothing. And I received in the mail a bill from the hospital, saying please pay in full, $96,000.

At the time when I became ill they had had a train derailment in Livingston, and this site was receiving all of that chemical there. I never even knew what it was. It was all, the chemicals were all mixed together. I hadn't been ill. I hadn't had a cold of any sort or anything for at least 6 years. And then all of a sudden this came up. And they don't know; the doctors are still calling me.

We have people around in New Orleans that have run biopsies, different tests, and nobody—they just all tell me they don't really know.

I have tried for different incomes, Social Security or so, but I haven't got anything, no information back on that. They paid me 6 months while I was out of—from the time I went and saw my first doctor, they paid me 6 months after that. And I haven't been receiving anything since.

I don't—the chemicals that were taken in there, we don't even—we have never—some of them they came from—they didn't even know, you know. It was just take it. That is the way it is. And so I am hoping that somehow, get something started, because I know it is bound to happen to others, because there is at least 10 of us that did work there that—I imagine 30 all together, but 10 that handled chemicals some times 36 hours at a time, 100 hours a week. That was very easily done. And it would almost cost you your job if you didn't do it.

[The statement of Mr. Hollingsworth follows:]

TESTIMONY OF JOHN KENT HOLLINGSWORTH

My name is John Kent Hollingsworth. I am 23 years old. I am married and have 2 children, one 5 years old and one 19 months old. I graduated from Sulphur High School, Calcasieu Parish, Louisiana in 1977.

I had worked for Browning Ferris Industries for almost 3 years, when in December of 1982, I developed a serious infection around my spine, which was followed by other complications. I was admitted to Lake Charles Memorial Hospital for 3 days in November of 1982. I was readmitted on January 18, 1983 and remained in the hospital for 72 days. I presently am aware of almost $110,000.00 in hospital and medical bills. To my knowledge, none of these bills have been paid.

When I became ill I was working as a tractor operator for the BFI landfill operations at Willow Springs in Calcasieu Parish, Louisiana, and my duties consisted of mixing hazardous wastes with kiln dust and moving the sludge into waste pits. At the time I became ill, to the best of my knowledge, I had been working for several weeks with waste received from the Livingston Parish Illinois Central Gulf Railroad derailment, which involved a large chemical spill and fire.

I weighed 160 pounds at the time I became ill, lost 40 pounds during the illness, and am now back up to 150 pounds. Prior to going to work for Browning Ferris, I had worked for several months for Lone Star Feed Store in Westlake, Louisiana; for about a year at Brimstone Fabricators in Sulphur; and for several months at various plants in the Baton Rouge area. At the time of the illness, I had been working for BFI for almost 3 years.

I was making about $250.00 net per week at the time I became ill.

When I became disabled, I continued to receive reduced salary based on 40 hours a week. I received this from the time I was hospitalized on January 18, 1983, until approximately April 15, 1983. Since that time I have had no income and have relied on savings, which are now exhausted, and the help of friends and family.

I have filed for social security and my claim is pending.

I have had no significant illnesses, diseases or injuries prior to this one, nor does my family have a history of illness or disease in any way similar to this one.

Although I hope to return to work, I have been advised by the doctors to remain off work indefinitely.

I have received no workmen's compensation benefits and none of my hospital, medical or drug bills have been paid.

My local doctor has called in a specialist from New Orleans. We have not yet received her report regarding the connection between my illness and my exposure to hazardous wastes at work.

Mr. LEVITAS. Mr. Drew Ranier.

TESTIMONY OF DREW RANIER

Mr. RAINER. Mr. Chairman, if I could, just briefly, on Mr. Hollingsworth's situation, he was employed as a backhoe operator in Parrishville at BFI landfill. He got very ill, required operation on his spine and 3 months hospitalization. He had numerous complications following initial draining of the abscess. He failed to respond to antibiotic treatment for most of that time until the very end of this stay. The doctors are convinced that there is some immune problem involved here.

The waste that he was dealing with for several weeks prior to becoming ill was, as he said, from the Livingston Parish train derailment. That is an Illinois Central-Gulf derailment involving many different types of chemicals. It may be that in the fire that ensued that the dioxins were created in the burning. We are not sure of that. We are in very early stages of discovery in the case, finding out exactly what we are dealing with.

So we are in a position, or Mr. Hollingsworth is in a position of being very uncertain in terms of medical proof about what has happened to him. But we do know that there was a massive immune breakdown in his case. The situation in terms of compensation support is dire. First of all, the workmen's compensation fund, or funds which would normally be available to someone who becomes ill because of working conditions, has denied coverage on the grounds that there is no certain medical proof at this point that this is even a work-related condition, although we know from the process of elimination that they have no other theories as to why that is so, why he got sick.

So the problem is dire in that normally workmen's compensation carrier or insurance would provide at least for his hospital and living expenses. They have not done that in this case, and they have denied that to date.

On the other hand, the hospitalization insurer has taken the position, we believe, they haven't answered the suit against them yet.

But they will do so shortly; that, in fact, this is work related and they have refused to pay. So there are approximately $110,000 in outstanding medical bills now with the workmen's comp insurer saying on the one hand this is not work related and the hospitalization insurer, on the other hand, saying it is. He is in a limbo, then, of no compensation at all.

Now, there may be other remedies. There may be remedies against the railroad. There may be remedies against his employer in tort other than workman's compensation. That remains to be seen. We are naturally going to explore those. But to the extent my testimony could help the committee, it would be about what the remedies are and what could be done.

As I understand the committee's inquiry about victim compensation, it regards basically remedying bad situations. I would make the following suggestions. First of all, what we need and what will not cost the Government a dime or the Superfund a dime are increased remedies. For instance, we protect people in society. An example I can think of, because I tried a case like this last week in New Orleans, we protect seamen. Under the Jones Act of 1920 we provide them with very special remedies. There are no such remedies for people who are victims of hazardous wastes, whether they work there or they live next to them, or otherwise.

The standards of proof in almost every State are the same as they are for every other litigant. It is easier for a seaman to recover from injuries than it is for a John Hollingsworth, or someone who lives next door to a site. So without even spending a dime from the Superfund, the problem can be addressed.

The way it can be addressed, very simply, is by reducing the burdens of proof that are put on these people to show that there is a relationship between their injury and their working conditions or their living conditions. There is a very simple way to do that. Require only that they show that they were exposed to significant levels of toxic chemicals, and then let the burden shift to the disposer or the producer of the chemical to then prove that in fact the illness is not related to their exposure rather than doing the way it is done now, and the disposers and producers say simply, proof that it is related.

And the problem is that that is very, very difficult proof and the companies, disposers or producers would be in a much, much better position to know exactly what was there, exactly what the people were exposed to. And in the case of Mr. Hollingsworth they certainly should be medically concerned about one of their own employees. So why not put the burden on the producers and disposers when it comes to medical proof?

The other problem brought up by Mrs. Jones, of course, is that the cost of these things is phenomenal. For an injunction you have got to put up a bond, so an injunction is almost meaningless because to shut down a profitable hazardous waste disposal site would be interrupting profits that may be in the region of hundreds of thousands of dollars per week and the bond would have to cover that potential loss. So it is literally impossible to obtain injunctions.

With regard to keeping medical burden of proof on the people who are injured, they have to spend conservatively, to prove their case in court by a preponderance of the evidence that their chemical exposures caused their injuries, at least $10,000, and as high as $50,000. We are talking about medical testimony that costs $350 an hour to obtain. So as long as the burden remains on the injured individual to establish the connection, the cost of litigation is exorbitant. And only the most, most serious of cases can afford to be litigated.

I think that the Superfund may be of some help in this regard in the temporary or short term. That it could provide compensation with subrogation rights. And by that I mean that the Superfund could provide temporary living expenses in the place of workmen's compensation and that that would be recoverable if and when recovery was made against some third party, the person actually responsible and at fault for the exposure.

I think it would be a lot better for this country if the people who are actually at fault for creating the problems have to pay, rather than a Government mandated insurance program. And one of the main reasons for my belief of that is that you can't actuarially make these places safe. The only way to make them safe is by making the people who are responsible for the unsafety pay for them. To the extent that those people are gone or no longer exist, the Superfund is there. It should be there as a backup. It ought to be there for temporary help. But the fact is that the people at fault for causing the problems are the ones who should pay, not the taxpayers and not some federally mandated scheme.

That is the best, the most efficient, the most direct way of solving this problem. And the best way to do that is to make a very simple change in the law. And that is treat people who are injured, who are exposed, differently than you treat other people in court. And you do that by reducing their burdens of proof. It would be a very simple way of doing it.

Mr. Hollingsworth's remedies in this case, he faces almost insurmountable medical proof problems when you have to go into court and prove by a preponderance of the evidence that what has happened to you is related to this specific exposure. It is a mountain to climb. It is a mountain to climb, evidence-wise. You have got to bring in experts that are not available in Lake Charles, LA.

He has good treating physicians but they are not trained toxicologists, they are not trained in hazardous waste exposures. They have got to go outside the community. They have got to go to the major medical areas, the few areas where you can find that kind of expert testimony. That testimony does not come cheap at all. So the evidentiary problems as they presently exist for Mr. Hollingsworth or anyone else in this room who has hazardous illnesses are almost insurmountable without change in the present law. And that change can be made without great expense to the Superfund.

I would suggest the Superfund remain there for what it is, and that is a backup when we can't find financially responsible parties and when we need it for temporary purposes. And then allow the fund itself to recover if and when we can establish fault.

Thank you, Mr. Chairman.

[The statement of Mr. Ranier follows:]

TESTIMONY OF DREW RANIER

My name is Drew Ranier. I am a lawyer practicing in Calcasieu Parish, Louisiana. I represent John Kent Hollingsworth in connection with a severe illness he incurred while employed by Browning Ferris Industries at the Willow Springs hazardous waste landfill disposal site.

A claim for workmen's compensation benefits was filed on April 8, 1983. The statutory weekly benefit and hospital and medical expenses have not been paid to date. The weekly benefit would be approximately $200.00.

Consequently, demand was made on the group health insurer, Provident Life & Accident Insurance Company, to pay hospital and medical expenses which are approximately $110,000.00. These hospital and medical charges were not paid as of July 5, 1983, and suit was filed accordingly. Presumably, the hospitalization insurer takes the position that the illness is work-related.

Also, on June 30, 1983, suit was filed in tort against the employer, Browning Ferris Industries, alleging intentional acts by the employer which caused Mr. Hollingworth's illnesses. These allegations primarily concern failure to provide adequate safety at the waste disposal site. If Mr. Hollingsworth can prove that these acts were intentional, rather than negligent, then he may be able to recover his full loss of wages, hospital and medical expenses, and items of general damages under Louisiana law such as physical and emotional pain and suffering. I do not want to comment on specific work practices by BFI because those practices are being litigated in the tort action. However, we can say that it is our belief that they were hopelessly inadequate.

In general, there are adequate remedies under Louisiana law for compensation of Mr. Hollingsworth. There are, however, two problems worth discussing:

(1) The cost of obtaining competent medical evidence on the relationship between toxic wastes and illness; and

(2) The living expenses of the injured party and his dependents during the lengthy period of time when evidence is being gathered and prepared for trial.

I would not propose a modification of the remedies available to Mr. Hollingsworth. However, I would be in favor of providing some type of temporary living allowance from available funds with subrogation rights against prospectively liable defendants. I would also be in favor of increased education and training of the medical profession, so that the treating physicians in communities with significant hazardous waste activities can develop expertise in treatment and analysis of toxic waste related illnesses, making it unnecessary for litigants to obtain distant and expensive medical testimony regarding the relationship between toxic waste and illness. In Mr. Hollingsworth's case, we are confident that it will require tens of thousands of dollars in medical and related expenses to prepare and try the matter.

Mr. LEVITAS. Thank you very much, Mr. Hollingsworth and Mr. Ranier, for your contribution. I have some questions, and I would imagine that the other members of the subcommittee do as well, that we will now present.

Before I do so I am informed there are representatives of Browning Ferris Industries present, and note that at this time in the interest of objectivity that we will afford them an opportunity to comment for the record on your statements if they so desire, because we are really trying to develop as objective and balanced an approach to this matter as we can.

Let me just take this opportunity to make this observation, not in any way in responding to the types of situations which the witnesses today have presented but just because it is a fact. For almost 100 years now—certainly for 50 to 75 years—we have lived in a society that has improved its quality of life and benefited from the scientific and industrial base of this country, and from the chemical products that have been developed to bring about lower costs, convenience, and industrial progress in our society.

At the time when these developments, in some instances—I guess even in most instances—were coming along and these chemicals were being used and developed and disposed of, they were for the most part being handled in accordance with the state of the art at that time, and what was then known. And now, in the last number of years, we are beginning to pay a price for that. We are finding out new information about these products and their consequences, and we are finding hazardous waste dumps that, in some instances, are 20, 30, 40, even 50 years old, and causing problems.

So what the Superfund Program was designed to do and what our concerns are is how do we deal with this? In some instances--and, again, I would suggest in most instances—the people who created these sites at the time and handled the chemicals at the time were doing what either they thought was correct or, in some instances, were actually doing what the Government told them to do.

On the other hand, we do know in recent years there have been extremely serious examples of the illegal dumping of the sort that Mrs. Jones described, that we have heard of from other sources. But while we do need to focus our attention on holding those people at fault who are at fault, we have a question which transcends that, and that is: What do we do about the problem? What do we do as a nation, as a society?

There is a shared social responsibility here that we simply cannot ignore, that is not going to go away, and I suggest that as our science improves we are going to find out that there are additional problems we don't even know about yet. By the same token, there are going to be additional chemicals, products that will be developed for the purpose of making a better life, whether it is in plastics or pesticides or pharmaceuticals or insulating materials or the like.

So while there is no doubt in many areas blame to be assessed, and individuals specifically to be held responsible, both civilly and criminally, in some instances, there is even a broader question, and that is: How do we deal with this enormous problem that Congressman Gephardt identified earlier, both for the resources of our Nation, and, to me, more importantly, for the human beings who have suffered who are clearly innocent parties?

I just make that observation because I think we need to put it in context.

Now, Mr. Payne, Mrs. Payne, you mentioned in the course of your testimony that on several occasions you have been seen or treated or examined by physicians from the National Center for Disease Control. I was wondering how you first got involved with CDC.

How did you happen to come to them, or they to you?

Mrs. PAYNE. When they found out that the dirt was contaminated, the St. Joseph Hospital set up a clinic for us to go over there and have tests made; anyone that lived around or close to the contaminated dirt, that we definitely should have tests run.

Mr. LEVITAS. And so it was through the St. Joseph Hospital?

Mrs. PAYNE. Yes.

Mr. LEVITAS. And the notoriety that surrounded the identification of this dump that you got into the CDC?

Mrs. PAYNE. Yes.

Mr. LEVITAS. What is the status of the Minker-Stout site? It is a closed site, I take it, now. Has it been cleaned up?

Mr. PAYNE. No, sir.

Mrs. **Payne**. I haven't seen anything they have done. Where the dirt was dumped, there are no signs. There is no fence. There is nothing at all.

Mr. **Molinari**. Would the chairman yield for a moment?

Mr. **Levitas**. I would be happy to yield.

Mr. **Molinari**. Mrs. Payne, do I understand that you have a sketch or diagram there showing the layout of the houses and your prior house?

Mrs. **Payne**. Yes.

Mr. **Molinari**. Do you have copies of that?

Mr. **Payne**. We have a few, yes.

Mr. **Levitas**. While we are getting copies of that, let me state that, without objection, that the complete statements, written statements, of all of the witnesses will be made part of the record at the appropriate place.

Mr. **Molinari**. Mr. Chairman, if I could continue with two questions on that score?

Mr. **Levitas**. Go right ahead.

Mr. **Molinari**. The Minker site is almost diagonally across the street from your prior home; isn't that correct?

Mrs. **Payne**. Yes.

Mr. **Payne**. Yes.

Mr. **Molinari**. If I understand this diagram right, we find homes behind your home, or down the road, to where Abrahamson lived, Henderson and Webb. They would be compensated in the Federal buyout program, but—no, I am sorry; the homes included are on the other side of the street, away from Minker.

The point I am trying to establish is, it is my understanding that although your home is directly across the street from the Minker site, you were not eligible for a buyout. Yet, others on the other side much further removed from the actual site, the Minker site, were included in the buyout program. Is that correct?

Mrs. **Payne**. That is right.

Mr. **Payne**. That is correct.

Mr. **Molinari**. Did anybody ever give you a reason for that?

Mrs. **Payne**. The road was the cutoff.

Mr. **Molinari**. The road was determined arbitrarily.

Mr. Wessel, do you have anything you could add?

Mr. **Wessel**. The Center for Disease Control indicated that unless there is dioxin on your property at a level of 1 part per billion or more, you will get no relocation assistance. They recommended that to EPA and FEMA concurred with that. The families across the street did not have any measurable dioxin. They are still being tested at this time. But they have not had any kind of relocation assistance extended to them because of that reason.

Mr. **Molinari**. But everyone that was included in the buyout did have levels that exceeded that figure?

Mr. **Wessel**. One part per billion or more, yes.

Mr. **Molinari**. Thank you.

Mr. **Levitas**. Mrs. Payne, in visiting the physicians, as you have described, did you ever discuss with them the possibility of dioxin as being a cause of some of these problems? And, if so, what response did you get?

Mrs. **Payne**. Not until after I found out dioxin was there. I have talked to my doctors several times and they don't have an answer.

Mr. **Levitas**. They have never said one way or the other as far as the possibility of dioxin being the cause?

Mrs. **Payne**. No; they said they don't know.

Mr. **Levitas**. Just for the record, you have described the many serious problems which your family has suffered, health problems. Could you just tell us if either you or your husband's family have a history of that type of medical problem which you related today?

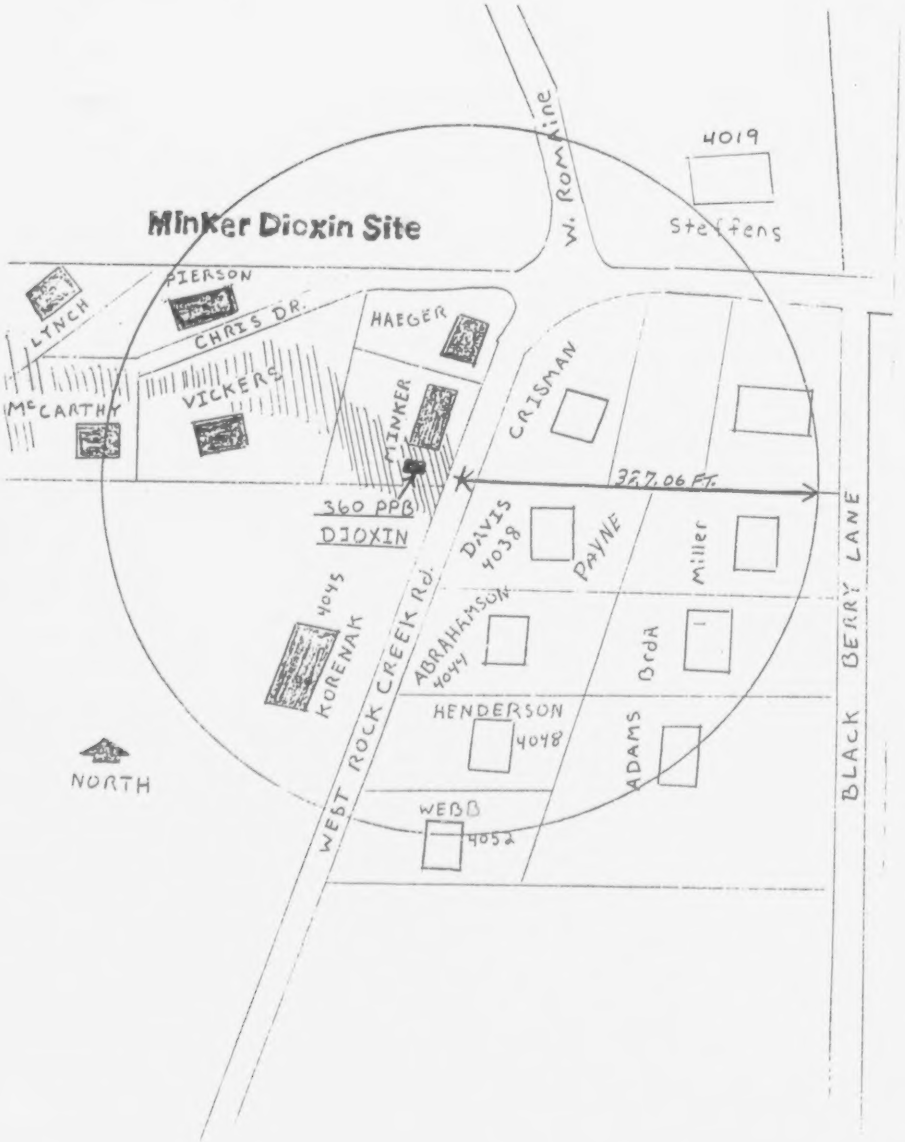
Mrs. **Payne**. No; on both sides, our families are fine.

Mr. **Levitas**. I am sure you are not fully capable, Mr. Wessel, of answering this question, but CDC, of course, doesn't make actual decisions as to how these sites would be handled. But it just seems to me totally arbitrary looking at this chart to have eliminated Payne and included Pierson or Vickers.

Mr. **Wessel**. Well, if you look at the Pierson property, there is dioxin on their property even though it is not directly adjacent to their house. On the Pierson and Lynch property—I believe there was the chance of migration at that time. I don't think that CDC believes there is the chance of migration across the street. If you look at the Korenak house, however, they have dioxin on their property in that small corner. If you look at the distance between the Korenak family and dioxin contamination in relation to that of the Paynes to the dioxin contamination, the Paynes would have lived closer so you would expect that they should have received some kind of assistance, also.

Mr. **Levitas**. Without objection, the diagram of the vicinity of the Minker dioxin site will be made part of the record.

[The diagram referred follows:]


Minker Dioxin Site
W. ROMAINE
4019
Steffens
LYNCH
PIERSON
CHRIS DR.
HAEGER
McCARTHY
VICKERS
MINKER
CRISMAN
327.06 FT.
360 PPB
DIOXIN
DAVIS
4038
PAYNE
Miller
4045
KORENAK
ABRAHAMSON
4044
Brda
HENDERSON
4048
ADAMS
WEBB
4052
WEST ROCK CREEK Rd.
BLACK BERRY LANE
NORTH

Mr. LEVITAS. Mrs. Jones, have you ever been able to ascertain what chemicals and substances were illegally dumped in that——

Mrs. JONES. It has been told there is a pool of cyanide and arsenic; we have been told there is toluene, benzene. We have seen no reports that show there is any high level of these toxics that are supposedly in there. We have been to meetings, Subcommittee on Crime in Manhattan, where two gentlemen testified that they dumped it.

Mr. LEVITAS. Did they say what the source of this material was, where they got it?

Mrs. JONES. One was the Quanta that I mentioned in my testimony, and there were other companies involved, also.

Mr. LEVITAS. We heard from the people who formerly lived in Times Beach that when news of the dioxin problem got around, there were not only fears and concerns that the people directly affected felt amongst themselves, which you described occurred in your neighborhood, but they also talked about the sort of reaction of people who didn't live in that area, treating them almost as pariahs, as lepers.

Mrs. JONES. That is true.

Mr. LEVITAS. Did you have any experience of that sort in your area?

Mrs. JONES. People that came to the neighborhood that were looking to buy homes stayed away from the neighborhood. People that put their houses up for sale, they sold, but they lost a lot of money. People that had binders on the house, the minute they mentioned stay away from Brookfield, they lost their binders. We were lepers. We are still considered that way.

Mr. LEVITAS. The next question I would like to put to Mr. Hollingsworth.

Before the time you dealt with this material before the train accident, were you in generally good health?

Mr. HOLLINGSWORTH. For 6 years, I know I hadn't missed a day of work because of illness. When it happened, I worked for at least 3 weeks, staying up all night with the pain and putting up with it during the day to hold my job. And they are the ones that told me to go get it checked, to go to the hospital. And they kind of left me alone.

Mr. LEVITAS. Have any of the other people who worked or lived near the BFI site, that you are aware of, experienced any illnesses of the sorts that you described?

Mr. HOLLINGSWORTH. Well, it has been a while since I have even been around there. But my dad lives down the road. They buy their water because you can't even take a shower. It really smells. They haven't had tests run that would tell them what really was in it. But the people that worked there I haven't seen since I have been in the hospital.

Mr. LEVITAS. Before you started doing work on cleaning up this material from the train wreck, did your employer give you any information about the type of materials you were going to be working with or any warning as to whether or not they were hazardous, and did they provide you with any special type of protective clothing and equipment? And, if so, did you use it?

Mr. **Hollingsworth**. We had masks, rubber boots, and things like that to handle it with. But late in the night when you were just about the only one there and these trucks constantly 24 hours a day hauling this stuff, if you got some inside your boot, or the masks would only last so long—well, you were more or less without. When I first went there, there were pits that had been there for years, and we had nothing whatsoever to wear, no masks. We mixed these pits until they were solid just to cap them over so they wouldn't be open to the air.

Mr. **Levitas**. Was a biopsy done on the growth after your surgery?

Mr. **Hollingsworth**. Right before I was let out of the intensive care, I had fat where they could find it removed and blood and different things. And I have been back once because they claimed they just let the blood spoil or something. They were not in that big a hurry. And of what they came out of my back, it was infections is all I know. That is all I have heard. While I was in there a few times, I swole up the size of the bed. And a couple times they punctured my chest, at least nine times, to put tubes in my lungs, and couldn't give me pain medicine, you know. They gave me very little so I wouldn't get where I needed it. They would just throw a towel over my face, poke a hole and do what they had to do. Wasn't very——

Mr. **Levitas**. You said that you are married and have two children?

Mr. **Hollingsworth**. Yes.

Mr. **Levitas**. Did they suffer any health problems during the time that you were working at the landfill?

Mr. **Hollingsworth**. My wife had a miscarriage. She lost lots and lots of blood. But, luckily, it hasn't shown up in my kids or anything.

Mr. **Levitas**. One final question about the equipment you said you were furnished. You said you had boots and masks. What type of masks were you provided?

Mr. **Hollingsworth**. The masks were organic masks.

Mr. **Levitas**. Did it cover your whole face?

Mr. **Hollingsworth**. You could get—they give you a choice. They didn't tell you what to wear, but they give you the mask. You had a full face, which didn't cover your hair, and that was just as easy to get it through your hair as it was by putting it on you. You worked in it whether you had it or not.

Mr. **Levitas**. One question to you, Mr. Ranier: I gather from your description of what you understand the law to be in Louisiana, the burden of proof is on the plaintiff and there is no doctrine of absolute liability in the case of toxic chemical——

Mr. **Ranier**. That is generally correct. Now there are strict liability doctrines for dangerous substances in Louisiana. But that doesn't apply. You still have the burden of proof of your case medically. In other words, you could prove you were exposed, you can prove you were entitled to recovery because of the exposure, but you still have to preponderate; that is, you have to have the medical doctors come in and say as a matter of reasonable medical probability this condition is caused by that.

Everywhere that I know of in the country that has strict liability doctrines, the strict liability does not extend to the medical proof. That is the real problem in these cases. It is not the problem of proving they were exposed; it is a problem of proving what caused it, how it caused it and getting the doctors to come in and say it is a matter of medical probability that it did.

Mr. LEVITAS. Yes. And, in fact, in some instances, in fact, many instances, in the case of exposure to toxic chemicals, the particular condition may not manifest itself for literally years after exposure to the condition. And you have got all sorts of questions—statute of limitations, finding the people—all manner of questions which we are going to be addressing in our further hearings, and particularly tomorrow where I expect to have some extremely useful testimony on both sides of this issue.

Mr. Molinari?

Mr. MOLINARI. Mrs. Payne, I think it was in your testimony you made reference to the fact that you had a letter from the State Department of Health indicating that dioxin may have been the cause of your medical problems.

Mrs. PAYNE. Yes.

Mr. MOLINARI. Do you have a copy of that with you?

Would somebody bring a copy of that up, please?

Mr. Chairman, I would like to ask that this letter of the Missouri Department of Social Services, Division of Health, dated May 11, 1983, addressed to Representative Richard A. Gephardt, be introduced as an exhibit and made part of the record of this hearing.

Mr. LEVITAS. Without objection, it is so ordered.

[The letter referred to follows:]

MISSOURI DEPARTMENT OF SOCIAL SERVICE,
DIVISION OF HEALTH,
Jefferson City, May 11, 1983.

Representative RICHARD A. GEPHARDT,
St. Louis District Office, St. Louis, MO.

DEAR REPRESENTATIVE GEPHARDT: The Ronald Payne family is included in both the health interview, and the physical examination and laboratory aspects, for the current Missouri dioxin investigation. Families in whom there were findings among the physical examinations or initial laboratory data were notified of these findings in April. We anticipate that it will be a matter of several weeks before all of the remaining test data are available for review by the medical panel. At that time, Mrs. Payne's letter will be reviewed by the panel along with other available data.

In the meantime, it appears to me that some of the symptoms reported by Mrs. Payne could be classed as compatible with reported health effects of dioxin. These include hemmorrhagic cystitis, "nerves", "repeated sore throats and colds", and various rashes.

It must be emphasized that while these symptoms may be so classified it remains to be determined whether they are or are not actually related to dioxin. They are all somewhat nonspecific—i.e. there are other factors which could be responsible, they are also somewhat vague and would have to be more completely documented before determination could be made with greater degree of assurance.

Since extensive EPA testing has failed to confirm the presence of dioxin in the former Payne premises now occupied by the Davis family, it remains unclear how, if at all, the various family members would have been actually exposed to dioxin at levels sufficiently high to cause the health problems reported in Mrs. Payne's letter of April 14.

Thank you for your concern.

Sincerely,

H. DENNY DONNELL, Jr., M.D., M.P.H.,
Director, Section of Disease Prevention.

Mr. **Molinari**. Thank you.

Mr. **Levitas**. Also, I believe, Mrs. Payne, you said you had your hospital records, medical records, available. I would request that they be made available to the subcommittee staff. I will not make them part of the record, but I would like to have them for our examination.

Mrs. **Payne**. I have some of them. I didn't have a chance to get all of our records.

Mr. **Levitas**. Thank you.

Mr. **Molinari**. Mrs. Payne, the letter was dated May 11. I notice in the first paragraph it says: "We anticipate that it will be a matter of several weeks before all of the remaining test data are available for review by the medical panel."

Mr. Payne, Mrs. Payne, Mr. Wessel, does anybody know whether those tests were concluded and what information is available?

Mr. **Wessel**. The raw data was received by the Center for Disease Control in Atlanta and has been shared with the State of Missouri. They have not interpreted the data yet. They expect to release that some time late August, early September.

They had two phases, I guess you could call it, of the health data. Early on, they had a group of 130 residents who were tested, 112 of whom were listed as having abnormal symptoms. Those included such things as anemia, et cetera. The most important tests for liver disfunction and immune system and to detect soft tissue sarcoma, those are in-depth tests and will be forthcoming at the end of August, we think.

Mr. **Molinari**. Now, let's then turn to the second paragraph, if we can. In the second paragraph, for the sake of those in the room, and for the sake of the record, Dr. Donnell, Director of the Section of Disease Prevention, mentions:

> "* * * it appears to me that some of the symptoms reported by Mrs. Payne could be classed as compatible with reported health effects of dioxin. These include hemmorrhagic cystitis, 'nerves,' repeated sore throats and colds, and various rashes."

Mrs. Payne, we have heard from your testimony that you and your family have suffered from some of these symptoms. Could you tell us whether these health effects described by the doctor were observed in the neighborhood in other residents, as well, or to what extent?

Mrs. **Payne**. Just about all of my neighborhood have had the same complaints, health problems, as we have.

Mr. **Molinari**. These symptoms that are being described here?

Mrs. **Payne**. Yes. Their children and——

Mr. **Molinari**. I do want to incorporate for the record a caveat: Dr. Donnell's letter, referring to the symptoms, states "while they may be so classified, it remains to be determined whether they are or are not actually related to dioxin." Nevertheless, we are seeing throughout these hearings, and any time we have been to a site, the same kinds of complaints.

I think our observations tell us something. You start to draw a picture when you see these symptoms and read of them.

I turn to Rita Jones. Rita, nerves is one symptom that medical experts say could be related to dioxin. I suggest that they may be related to other toxins, as well. Have there been suggestions that you and others, are suffering nothing more than nervous problems?

Ms. JONES. Yes.

Mr. MOLINARI. A nervous problem?

Ms. JONES. Yes, there have been. It seems there is a lot of people in my area, we are all suffering from the same thing, nervousness. We are all breaking out with two similar rashes, and when we do break out, it is a lot of people at once.

There are two different types of rashes, only two. One is an acne type of rash that is mostly in adults, and children come down with a rash that looks like ringworm, and the doctors usually in the area prescribe medication, hydrocortizone, I believe it is, in a cream base, and they use it for 4 to 6 weeks, and the rash will clear up and it comes back again.

Can we all be suffering from nerves?

Mr. MOLINARI. Rash is one of the more common symptoms of people exposed to toxic chemicals and hazardous substances. Do you know people in the neighborhood who go away on vacation for maybe an extended period of time? What happens to them while they are away?

Ms. JONES. I, myself, my family included, we have been away to Washington. We went from Washington. We weren't home more than hours and my husband started with severe nosebleeds again. My little one was complaining about a sore throat again. My big one was wheezing all over again.

We had to start medication again.

Mr. MOLINARI. I take it what you are saying is when you are away, these symptoms disappear?

Ms. JONES. There were no symptoms whatsoever. I didn't have to administer any medication for 4 days or 4 nights to either child.

Mr. MOLINARI. How long after you leave the area going on vacation do you notice that the symptoms disappear?

Ms. JONES. A few hours later.

Mr. MOLINARI. That quickly?

I would like to turn to Mr. Ranier.

Mr. Ranier, I have been interested—I am an attorney myself—in your testimony. I think you have touched on some of the serious problems that people afflicted with the conditions that we are discussing today have when they go to a lawyer. Have you tried cases of toxic exposure, or problems associated with it, before?

Mr. RANIER. Yes, sir, I have.

Mr. MOLINARI. How do you make a determination? I heard you say—in fact, I think you used the term "insurmountable." How do you make a determination? Do you take these on a contingent fee basis?

Mr. RANIER. That is correct.

Mr. MOLINARI. I assume that would be the only way.

Mr. RANIER. Yes, sir.

Mr. MOLINARI. How do you make a determination, then, to take a case? What factors go into determining whether you will handle a case or not, since it is going to take a tremendous amount of your time? I would assume you would have to have some reasonable expectation of success before you would take on a matter such as this.

Mr. RANIER. Probably the principal controlling factor is the magnitude of the injury. That is, you just can't afford to devote the kind of time and money and effort on a contingency basis that you have to with one of these cases unless you know that you have got a very serious injury and the prospect of recovery is going to be substantial enough to compensate you for your work and for the amount of expense.

I think it is true in almost every State that, technically speaking, the expenses of litigation are the client's responsibility, but in fact, someone in Mr. Hollingsworth's position, who already has $110,000 in unpaid medical bills and has been out of work since January, and has no immediate prospect of returning to work, they are not going to be able to afford those.

So the basic answer to your question is, one, that you have to believe that eventually the medical test is going to show that there is a connection and, second, that the risk you take is at least for a severe enough case that it is going to justify your involvement.

Mr. MOLINARI. In the State of Louisiana, would you have a class action possibility if you had an area such as some of the areas described where there are substantial numbers of people affected?

Mr. RANIER. Yes, you do.

Mr. MOLINARI. I have no further questions, Mr. Chairman. I would just like to wrap up by making the observation that I have been rather sickened by what I have heard today, not that it is anything new. But we have people from three different parts of the country testifying here today, and we can see not only the physical pain in their eyes, but the mental pain that they have exhibited. It places a very heavy burden upon us, because they are crying out for help, it is obvious. We would like to help, and I think that some of the suggestions that you have made, Mr. Ranier, and those of the other, witnesses are going to be seriously considered by the subcommittee.

And for the rest of you, I just want to express my own deepest appreciation.

Mrs. Payne, your first airplane trip—that has to be an experience to do that to come here. You deserve our attention—not only our attention, but our action, and I hope that we can play some role in addressing the problem properly so that we can offer some assistance to others like yourself. Rita, I appreciate your coming, as well as you, Mr. Hollingsworth, with a horror story that has us all shocked.

Thank you.

Mr. LEVITAS. Congressman Breaux.

Mr. BREAUX. My time is very brief.

Mr. Ranier, is the landfill still open at Willow Springs?

Mr. RANIER. It is, and the Environmental Control Commission of Louisiana, I believe, has issued a closure order for December of this year.

However, that has been appealed by BFI. I think that is the present status of the operation.

Mr. BREAUX. There are a lot of examples with regard to common law cases that have been developed over the past that provided compensation for plaintiffs who have been exposed to chemical toxic waste areas.

Mr. **Ranier**. There are traditional theories of recovery. They are still burdened with the problem of proving that the plaintiffs undertaking—and they are available to people. The problem with an employee, of course, his rights against his employer, Mr. Hollingsworth against the employer, are severely curtailed even to begin with, and directed into worker's compensation in general.

There are some exceptions to that. But the workmen's compensation remedy in this case has been meaningless to date. No workmen's compensation has been made. They voluntarily kept him on salary up to a period of several months of his going into the hospital, but the problem he was faced with then was he had to decide under the Louisiana law within 6 months whether to make a claim.

As soon as he made the claim, the volunteer payments stopped. He has been without compensation now since the middle of April, which is the time that the claim had to be made to ensure that he did not lose all of his rights in the future.

The 6 months did not run and voluntary payments could stop and there would be no legal remedy, so there are remedies, they are restricted even in worker's compensation, you still have the same burden of connecting the working environment with the injury.

Mr. **Breaux**. I think what this panel has shown to us, Mr. Chairman, is that we really are talking about a new area of the law, that the old rules of standards and burdens of proof are going to be impossible for plaintiffs to ever get any resources.

We are in the ironic situation, as staff pointed out to me, if you have a pine tree in your front yard, you could get compensated if the tree died, out of the Superfund. Because you are a human being with hundreds of thousands of dollars of suffering, the law doesn't cover you, and there has to be something wrong with that type of setup that needs congressional attention and congressional direction.

I would say to all of you, I think that by your testimony you perhaps made it a lot easier for those who are going to follow you and for future generations, and we thank you very much for your appearance.

Mr. **Levitas**. Thank you, Mr. Breaux, for those observations.

The subcommittee will stand in recess for 10 minutes, and then I think we will be able to conclude very shortly thereafter.

[Recess.]

Mr. **Levitas**. The subcommittee will please come to order.

I have been advised that one of the ironies of some of these things that keep cropping up is that to the extent that the problems in Ms. Jones' neighborhood, Staten Island, were caused or triggered by the digging of that large trench, which we saw on the photographs that were provided us, that the reason the trench was dug was to allow for the construction of a sewer line required by a sewage treatment project which was funded by the United States Environmental Protection Agency Construction Grant Program, in response to the Clean Water Act, and I just find that to be a very ironic development in this matter.

I want to express, again, my gratitude to all of you for being here today. I don't think you have any idea the importance of your testimony. It is testimony that certainly gives substance to the concerns that many people have, and the type of information you have provided is similar to that we heard on other occasions.

I remember earlier this year having testimony from people who lived in a part of Dallas, TX which was contaminated by lead and what was happening in those communities and the type of physical and emotional response problems that the people had in that area is quite similar to what you have described today, and as Congressman Molinari said earlier, we are concerned, we want to help, and we want to do it in a responsible way, and that is why we must pursue these investigations and hear from all parties, not just from some, and we will do that, and when we are through, to the extent it is possible to know what to do, we will know it.

You have made certainly a great addition to our knowledge as we pursue that.

Mr. Molinari, do you have something?

Mr. MOLINARI. I just have two remaining questions I would like to address to Mr. Hollingsworth, if I may.

Mr. Hollingsworth, in your testimony, you say you were advised—Mr. Ranier, if you have any objection to the questions, please interrupt me—advised by doctors to remain off work indefinitely.

Do you know what prognosis they have for your condition?

Mr. HOLLINGSWORTH. That is what the biopsy is for, whatever they find out what it is, then I will know.

Mr. MOLINARI. Other than the biopsy and any other indication of what your future health problems may be, assuming that the biopsy is negative, any further prognosis on where you are going, healthwise, that is?

Mr. HOLLINGSWORTH. What little bit I can do is pretty painful, so I wouldn't know.

Mr. MOLINARI. There is no indication at this time as to when you will be able to resume working?

Mr. HOLLINGSWORTH. No, none.

Mr. MOLINARI. I have no further questions. I thank you for your patience. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you very much.

Again, we are grateful and you will have helped countless thousands of other people as a result of what you have done today at great personal sacrifice to yourselves. Thank you. You are excused.

Tomorrow, the hearings will resume on this portion of our oversight and we will hear from Dr. Bertram W. Carnow, director of Occupational and Environmental Medicine of the University of Illinois at the Medical Center in Chicago.

He is an epidemiologist, toxicolist and author of numerous publications on health effects of hazardous waste materials. He will be joined on a panel by Dr. Bruce W. Karrh, who is the general director, medical safety and fire protection, of DuPont Industries. And also Mr. Courtney Riordon, Acting Assistant Administrator for Research and Development of the U.S. Environmental Protection Agency.

It is also possible that on tomorrow's panel they will be joined by representatives of the American Medical Association. The American Medical Association, at a meeting held of its House of Delegates in Chicago last month, adopted a resolution with respect to the subject of dioxin.

A headline which caught my attention appeared in The Washington Post of June 23 of this year which says, "AMA Votes to Fight Dioxin Witchhunt," and article proceeds to say, "The American Medical Association accused the news media yesterday of conducting a witchhunt against the toxic chemical dioxin and decided to mount a public relations campaign to counter the hysteria."

And, obviously, that is a rather interesting position. Given the prestige of the American Medical Association, I feel it is not only appropriate but essential that we hear from them to get their insight to this matter now that they have gone on record concerning this issue.

Mr. Molinari. I just wanted to mention that a day or two later there was a backpeddling on the part of AMA I don't know if you have copies of those follow-up articles indicating that they had some second thoughts about the earlier statements, which I think were drafted by an ophthalmologist.

I don't know how that qualified him to pass upon dioxin. But my recollection is that 1 or 2 days thereafter, the AMA put out a further statement recanting their position and taking a less harsh position on the so-called witchhunt.

Mr. Levitas. Well, I am just really interested to find out what their position is. As the organized representative of the medical community in this country, their position should be known, whatever it is at this point. I would like to know what it is.

I would also indicate that the subsequent hearing we are going to be dealing with in this portion of the victim compensation risk assessment may, as a result of the testimony tomorrow, include other witnesses next week or at a subsequent occasion. We also are considering a much closer look at a parallel and what we can learn from the Italy incident that was referred to earlier today by the witnesses from Times Beach. I have asked the staff to look into that, to find out how we can get a better understanding of what happened there. It is one of the most notorious examples of major contamination as a result of dioxin. As many as 37,000 people were exposed to dioxin as a result of a fire and explosion in that incident.

Is there any further business to come before the subcommittee today? If not, the subcommittee stands in recess until 10 a.m. tomorrow morning.

[Whereupon, at 2:15 p.m., the subcommittee recessed, to reconvene at 10 a.m., Thursday, July 21, 1983.]

[Subsequent to the hearing, the following was received for the record:]

Browning-Ferris Industries,
Houston, TX, August 4, 1983.

Re hearings on hazardous waste victims compensation.

Hon. Elliott H. Levitas,
Chairman, Subcommittee on Investigations and Oversight, Committee on Public Works and Transportation, U.S. House of Representatives, Washington, DC.

Dear Mr. Chairman: On July 20th, testimony was presented to the Subcommittee by Mr. John Hollingsworth, of Lake Charles, Louisiana and Mr. Drew Ranier, Esquire, of Baggett, McCaull and Ranier, Lake Charles, Louisiana with reference to a chemical waste disposal facility operated by Browning-Ferris Industries Chemical Services, Inc., in Willow Springs, Louisiana.

Please find attached a statement in behalf of Browning-Ferris Industries Chemical Services, Inc. for inclusion into the hearing record.

We appreciate the opportunity to present this information to the Subcommittee.

Sincerely,

JAMES R. GRECO,
Division Vice President, Government and Industry Affairs.

WRITTEN TESTIMONY ON BEHALF OF BROWNING-FERRIS INDUSTRIES CHEMICAL SERVICES, INC.

On July 20th, testimony was presented by Mr. John Hollingsworth, Lake Charles, Louisiana and Mr. Drew Ranier, Esquire, Baggett, McCaull and Ranier, Lake Charles, Louisiana with reference to a chemical waste disposal facility operated by Browning-Ferris Industries Chemical Services, Inc. (BFICSI), in Willow Springs, Louisiana. Since a number of lawsuits have been filed on behalf of Mr. Hollingsworth, it is not appropriate at this time to discuss in detail the merits of issues that will ultimately have to be decided by a judge or jury. In order to complete the record before this Subcommittee, however, there are two points which need to be made.

First, BFICSI has an extensive Occupational Medicine Program. The key elements of this program include pre-employment and periodic health examinations; periodic medical surveillance to provide early detection of exposures to chemical, biological or physical hazards; consultation with area physicians concerning the diagnosis and treatment of occupational health exposures; periodic medical inspections of the workplace to evaluate the adequacy of first aid and emergency medical procedures; and periodic meetings with location personnel to discuss chemical exposure. The BFICSI Occupational Medicine Program is under the direction of a consultant, Dr. D. Jack Kilian, who has had 30 years experience in the chemical industry and is currently Professor of Occupational Medicine, University of Texas Health Sciences Center, School of Public Health, Houston, Texas.

Although Mr. Hollingsworth's condition was not considered to be work-related, at the request of BFICSI management personnel at the Willow Springs location, Dr. Kilian contacted Mr. Hollingsworth's treating physicians in early February of 1983. Dr. Kilian's involvement included the release of BFICSI medical history on Mr. Hollingsworth and medical consultation. Dr. Kilian also recommended testing procedures needed to determine potential chemical sources of the infections and it was agreed that BFICSI would pay for this testing. Dr. Kilian's assistance continue until suits were filed in April of 1983.

Secondly, BFICSI provides statutory Workers' Compensation benefits for job-related injury and illness and group health benefits for non-occupational injury and illness. Mr. Hollingsworth's initial position prior to April of 1983 was that his condition was not job-related. Medical bills were paid as provided under BFICSI medical plan and his salary was voluntarily continued. In April, a lawsuit was filed and a new claim form submitted contending Mr. Hollingsworth's condition was job-related. At that time, group benefit payments were continued for the initial condition, coverage was denied as it related to the condition contended to be job-related, and Mr. Hollingsworth's salary was discontinued.

In view of Mr. Hollingsworth's inconsistent positions, efforts were initiated through Dr. Kilian and BFICSI legal counsel to determine whether Mr. Hollingsworth's condition was job-related. In July, based on all available data, along with conversations with Mr. Hollingsworth's physicians, it was determined by Dr. Kilian that Mr. Hollingsworth's illness was not job-related. In view of these findings, medical plan benefits are being reinstated.

It is clear, therefore, that Mr. Hollingsworth does have a remedy. Complete development of all issues will be accomplished through the litigation he has commenced, by any difficulties he has experienced in connection with obtaining medical benefits have been occasioned by his own submission of inconsistent claims.

In the meantime we appreciate the opportunity to present this information to the Subcommittee.

HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

THURSDAY, JULY 21, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 10:10 a.m., in room 2253, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Mr. **Levitas**. The subcommittee will please come to order.

This morning, we resume the second phase of our hearings on hazardous waste and EPA's management of the Superfund and related programs. We are focusing during this segment of our hearings on the subject of victims compensation. The subject matter is really one that lies at the heart of decisionmaking, both in the private sector and in the public sector, and will ultimately evolve into what is very likely a new area of the law itself in the toxic tort area. Next week we will continue this set of hearings.

As I indicated yesterday, the subcommittee intends be have hearings during the August congressional recess. The specific subject matter is not yet clear, but I would suspect there would be at least 1 or 2 days of hearings on matters involving the hazardous waste program, either on the Department of Defense aspect or the Seymour, IN site. We will make an announcement about that hopefully next week so people involved and the interested parties can make their plans accordingly.

This morning, we are particularly pleased to have a very distinguished medical-scientific panel here who will be in a position to give us their views as experts in the field; and, as well, hear from the Acting Assistant Administrator for Research and Development of the U.S. Environmental Protection Agency on the subject and also on EPA's views of the whole question of risk assessment, and what policies the agency has in that regard.

It is for that reason that we are particularly pleased to welcome Dr. Carnow, Dr. Karrh, Dr. Riordan, and Dr. Riordan's colleagues from EPA, Dr. Elizabeth Anderson and Dr. Casey Jason, and one other name not on my list.

Ms. **Lewtas**. Joellen Lewtas.

Mr. **Levitas**. Thank you.

We are pleased to have all of you here today. We will be looking forward to hearing your testimony.

At this time, I want to inquir whether my colleague, Mr. Molinari, has an opening statement to make?

Mr. **Molinari**. I do not, Mr. Chairman. I am just looking forward to the hearing today and hope that we get some answers to the questions we have been raising for some time.

Mr. **Levitas**. Thank you.

Dr. Rowland?

Mr. **Rowland**. I have no opening statement, Mr. Chairman.

Mr. **Levitas**. With that, if the witnesses would raise their right hand, please.

[Witnesses sworn.]

Mr. **Levitas**. Thank you very much. I don't want you to think you have been singled out to have the oath administered to you. This is an investigating and oversight subcommittee. All the witnesses provide their testimony under oath.

This morning, I think it would be best to hear from each of the members of the panel before the members of the subcommittee put their questions. So, why don't we just begin with you, Dr. Carnow. We look forward to your testimony. We welcome you here today.

TESTIMONY OF BERTRAM W. CARNOW, M.D., PRESIDENT, CARNOW, CONIBEAR & ASSOCIATES, LTD.; BRUCE W. KARRH, M.D., GENERAL DIRECTOR OF MEDICAL, SAFETY AND FIRE PROTECTION, E.I. DU PONT DE NEMOURS & CO.; AND COURTNEY RIORDAN, PH.D., ACTING ASSISTANT ADMINISTRATOR FOR RESEARCH AND DEVELOPMENT, ENVIRONMENTAL PROTECTION AGENCY, ACCOMPANIED BY ELIZABETH L. ANDERSON, PH.D., DIRECTOR, OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT; CASEY JASON, M.D., REPRODUCTIVE EFFECTS ASSESSMENT GROUP, OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT; AND JOELLEN LEWTAS, CHIEF, GENETIC BIOASSAY BRANCH, HEALTH EFFECTS RESEARCH LABORATORY, EPA

Dr. **Carnow**. Thank you very much, Mr. Chairman.

My name is Bertram Carnow. I am a physician, president of Carnow, Conibear & Associates, an occupational-environmental health consulting firm, and professor and director of the Great Lakes Center for Occupational Safety and Health at the University of Illinois, College of Medicine, at Chicago, and professor of occupational-environmental medicine at the School of Public Health at the university.

I am appearing at the request of the committee. I represent only myself, no organization, and the views that I present and express are likewise my own. I have been asked to discuss generally the extent of exposure as I see it, and particularly risks of acute chronic or long latency health effects, the actual incidences in my experience; and, second, the medical and scientific certainties and uncertainties associated with establishing a link between exposure and disease.

Now, diseases from toxic waste, which is the matter under consideration, are part of a group of environmental diseases. I think for the purpose of discussion, I would like to define it as I see it, because in order to establish whether such a disease is present, one would have to fulfill at least some of the definition. I feel that these are diseases that are caused by toxic chemical, physical or biological agents present in the community at levels or under circumstances which permit undue absorption into the body, this undue absorption then causing an altered cellular and organ response which adversely affects organ function. And, it is as a result of this, the health of those who are affected.

I think that toxic wastes are present in some water supplies, soils and airs in this country is not in question. I think that thousands of toxic sites have been identified. The degree of loss of containment or health impact, however, has not been quantified, and in most cases has not even been addressed. Since many stored substances are extremely toxic and many were placed in areas without regard to packaging, soil quality, or water tables, with many of these formerly unpopulated areas which are now residential communities, the problem, while unknown, may be very great.

In examining this, you have to examine how diseases surface, how the disease problems surface in relation to communities. First of all, either the community may be aware of or professionals may be aware of a large number of people, more than would normally have a disease, developing a common disease, either a respiratory disease or infectious diseases or mass diarrheas, and so on. And in these situations, a cause is sought that goes beyond individual susceptibility.

For example, in Australia, an epidemic of high blood pressure in young people was found. In pursuing this, it was found that they consumed water which contained lead. The lead affected the kidneys, which is the major target organ of lead. And so there was this epidemic of what has generally been considered a "natural disease."

I should say also at the outset that an environmental or occupational disease is a natural disease, but from an unnatural cause. That is one of the problems that we have with considering these diseases.

The second way it may emerge is that increased numbers of individuals with an uncommon disease may appear, as for example the leukemia increases in Massachusetts. Another case in point earlier, and not in this country, was the appearance of mesothelioma which is a disease caused by asbestos. Everybody is pretty well convinced of that.

Now, this is a disease that might appear in one in a million individuals in the population. In South Africa, some 40 cases were found in the community. It was found that the roads in that community were paved with tailings from the asbestos mine, and the diagnosis and association was very rapidly made.

Another way in which this may surface is increased deaths from many causes, reproductive abnormalities, premature births or increased spontaneous abortions. With regard to that, by the way, I should note—I am sure it will come up in discussion—that one way of looking at this is with the development of registries, national registries or State registries. There are very few of those in this country that address either cancer, birth defects or others of these abnormalilties They would be very useful in pinpointing problems.

Another way of looking at environmental diseases is to find toxic agents in a place and then develop a medical surveillance protocol to look for those diseases in those organ systems known to be affected by the toxic substance which may be present. And, to a large degree, this is what is happening, for example, in Times Beach and other communities that have been found to have had hazardous materials in them.

Or another way is, of course, to try to measure the amounts of the contaminant in the body once it has been established that there are contaminants in the community. Now, in many cases this is difficult or impossible, because a lot of chemicals leave the body rapidly. Many solvents do. And they do their thing as they go through. In others, you can't measure them in body fluids; for example, asbestos, because it enters the body as a dust in the lungs. The only way to measure it would be to remove lungs and look at those. Others, for example, like dioxin, are present in such small amounts in tissues that they are virtually impossible to quantitate.

Now, the problems in measuring the impact on health are many. That is one of the reasons why I think this is being addressed today. First of all, these diseases can mimic natural diseases, so-called natural diseases, because the body can only react in a certain way. The lungs can only react in certain ways—shortness of breath or cough or recurrent infection. The same is true with the liver, and so on. So that when an inflamed liver is found, it is sometimes difficult to make a determination of what has caused this inflammation.

The second problem, is that many diseases are multifactorial. That is, they can be caused by more than one cause. Many cancers, like lung cancer, can be caused by cigarettes. They can also be caused by asbestos. Each of those will increase the number of cancers in the population by 8 to 10 times. When the 2 are present together, they may increase the incidence in the population by from 50 to 90 times.

So another problem is not only that diseases are multifactorial but multiple factors may contribute to a disease. So it is not either smoking or asbestos; it is obviously both.

Another difficulty is that many of the diseases we are concerned about have long latency. That is, they may take a long time before they appear. And, because of this, they may be hard to relate to a cause.

The other problem with long latency diseases is that when they do appear, unfortunately they are frequently chronic, irreversible and, as in the case of cancer, fatal. And so every effort has to be made to find them before they appear, or at least at a point where they may be reversible, or prevented, of course.

Let me give some examples. In one community in Alabama over many years, DDT was released into the water, thousands of tons. The people in the community drank the water, bathed in the water and ate the fish from the water. A large number of individuals were affected and many of them developed diseases which were not recognized as being environmental diseases but were seen by different physicians and called many different things.

Another, for example, related to lead. We found in a number of instances downwind from lead smelters of lead poisoning in communities. Now, the children in those communities were lethargic. Some of them had anemias. Some of them were mentally retarded. Some of them had other symptoms, none of which were related to lead until they were looked for, and it was determined that these children had high concentrations of lead, those that were living downwind from this smelter.

So the problem of risk and the problem of how many people have been affected, I believe, has barely been raised. I think it may be akin to the problem that we had with black lung where we thought there might have been a few thousand cases in the community. When compensation was offered to black lung victims we find ourselves now with more than 300,000 miners or their families being compensated. So that it may well be that some of the things that we are seeing may be the tip of an iceberg.

Certainly, most of us feel that cancer is an environmentally related disease. One has only to look at the National Cancer Institute maps to understand this, because there are hot spots of cancer in this country, unquestionably. If we can find those factors which go into the development of cancer, or causation of cancer, then we can eliminate this group of diseases which kills 1 out of 4 Americans, 650,000 new cases each year, 350,000 or more deaths.

Let me then address the second question, that of medical scientific certainties and uncertainties. Dr. Shirley Conibear, my colleague, and I have developed models or ways of looking at the problem. If you can do an epidemiologic study, you can come up frequently with statistically related associations which may be very useful.

The problem we now have in the work place as well as the community is that there may be just a few cases, or there may be subclinical cases. And so what we need to do is to establish methods for estimating the probability that a disease is related to an external or environmental cause, because all we are dealing here with are levels of probability.

When we talk about causation, what we talk about is how probable is it that a disease is caused by an exposure. To do this, we have to consider a wide range of parameters. The first is to make a toxicological assessment of the materials, the substances, that are found. This tells us first about how toxic an agent might be and how much of a problem it might be; and, second, it tells us also about the possibility that it might be absorbed into the body. I will address that in a moment.

Second is to make an environmental assessment because that helps us determine the probability that a material is available—if you consider the definition—that a material is available for absorption and that it is present in a place and in a way that it may be absorbed by individuals.

The third major component is to look at the community at large and try to identify those individuals in the community who may be at high risk either because they are children, pregnant women, aged individuals with chronic disease, cigarette smokers, and so on and so on, because those are the individuals who might be expected to show the first evidences of disease from an external cause, because they are at higher risk.

Finally, to carry out medical procedures will help to determine a relationship. And the way to do that is, after we determine what materials or agents are in the environment, we set up a set of tests which will look at those organ systems which may be affected by those substances in the environment. If they are, then that enhances the level of probability.

Now I would like to take just a few moments to run through briefly some of these parameters and use as an example one which I believe you have already been discussing and one which has become a public household word. That is, the problem of dioxin. In making a toxicological assessment, the first thing you might look at is intrinsic toxicity of the agent. Dioxin, as we know, is probably the most toxic of all man-made chemicals. It is capable of killing a fetal animal at five parts per trillion, an extraordinarily low concentration.

Second, we would look at how can this chemical get into the body. In the case of dioxin, it can get in through the skin, through the respiratory tract, it can get in through the mouth. So there are multiple portals of entry. What the body sees, of course, is the total body burden. That is the one that is of concern to the body.

The third is the form of the agent. Is it in the form of something which can be breathed, that can be drunk, and so on? We then look at the whole question of how long it stays in the body. We call that half-life; how long does it take before it is broken down. Well, something like dioxin, because it has a chlorine at the 2378 position, is very difficult to destroy, because those are positions that this chemical is destroyed at and these positions are blocked by chlorines and therefore it can't be destroyed easily. So it stays in the body for a long time. It is not easily degradable.

We then look at the question of where is it stored. We find dioxin is stored in multiple organs, including pancreas, adrenals, kidney, the brain, and so on. We then ask the question: What are the known target organs? From a huge collection of data which has been assembled since 1953, we know that dioxin can affect virtually every major organ system in the body.

Finally, we look at what are the rates of excretion or how is it excreted. We find that with this chemical, it is excreted very slowly, if at all, in humans once it fixes in tissue. So that would be a toxicologic assessment.

Based on the answers to this question, one might present, on a scale of 0 to 10, the probability that toxicologically this may be of significance to the community in terms of health. The second thing that would be considered are the environmental and industrial hygiene assessments. For example, emission and production data of plants in the area; changes in processes and materials and controls; when these occurred in industries whose wastes were dumped or which were in the area.

We would look at the degree of containment of substances which are already in the ground by looking at air, dust, water, soil samples, to see whether or not it has gotten out of where it was originally. We would look at the probability of containment by looking at the depth of burial of waste, at the porosity of the soil, the level of the water table in relation to the dump, the packaging of the waste and at the length of time it has been there. All those are important questions in making a determination of whether or not it is available to the community.

With dioxin, we know that this was in one place. It was removed; it was spread over large areas. And it still is present in many of these areas.

Then we look at the probability of physical contact. For example, did people who are exposed—were they wearing protective clothing, and so on. In the case of communities, generally this is not the case. You have individuals moving around, children playing in the streets, food and gardens, all things we do in our communities.

We then try to look at the community itself. Is this a community that has many children? Is it a community that has many women who may become pregnant or who are, or who have been? Is it a community which contains large numbers of older people who may have chronic disease? All these are important in making a determination of what portions of that community we might want to survey in order to make a determination of what the impact might be of a toxic material which has been discovered in that community.

Finally, as I pointed out, we would do measures of either the effects on organ systems, which might be affected by this material, or measurements of body burden; that is, to look at the body and body fluids to make a determination of what the lead levels might be of mercury or cadmium or whatever, if that is possible. In some cases, it is. Then, after all of this is found, we would then ask a number of questions. There are many questions.

But, among those, we would ask the question: Is the relationship between the disease and exposure reasonable and logical? Was the dose sufficient to cause disease in the individuals who are diseased? Is the organ affected and pathology we find what might be expected from such an agent? That becomes very important. And, are multiple organs which are known to be affected by this agent affected, because there are many factors which can cause a disease of the liver? But if you have a disease of the liver and a disease of the brain and a disease of the peripheral nervous system and disease of other systems that we know, for example, that can be affected by dioxin, then it becomes one of these questions of, if it walks like a duck and talks like a duck, it in fact is. This is the way we get to a diagnosis of relationship of disease to a cause.

Then we ask the question: Are those who are expected to be at highest risk due to dose or other factors, the ones who develop the disease? Finally, we ask the question: Are others—not all others, because there are differences in susceptibility—but are others who are similarly exposed, similarly affected? If you can answer those questions in a reasonable way positively, then one has a responsibility to make an assumption that there could be a strong association which is akin to causation in disease between an exposure and a toxic agent.

I think this is, in brief, my statement. I have prepared what is a larger document which you may look at. One of the things that this document points out—and I would like to call the committee's attention to it are the few trained individuals, including physicians, in this country, in this field of environmental and occupational medicine. And while this is not a major problem in toxic waste, it is one of the problems that we have in finding individuals who may have been affected, because physicians generally are not trained to look for cause, but are trained to diagnose and treat disease. This is another area which I think this committee may not be addressing, but one which needs to be addressed.

Thank you very much.

[The statement of Dr. Carnow follows:]

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MEDICAL AND TOXICOLOGICAL CONSIDERATIONS IN DETERMINING PROBABILITY OF CAUSATION OF ENVIRONMENTAL DISEASES

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MEDICAL AND TOXICOLOGICAL CONSIDERATION IN DETERMINING PROBABILITY OF CAUSATION OF ENVIRONMENTAL DISEASES

Bertram W. Carnow, M.D. and Shirley A. Conibear, M.D., M.P.H.

Clinical environmental and occupational medicine is at the interface of diagnostic and therapeutic medicine on the one hand and preventive medicine, including epidemiology, toxicology, and biostatistics on the other. It extends diagnosis of disease to include identification of cause. It applies the data base of epidemiology and toxicology to a single or relatively small number of cases in order to accomplish this end. In the absence of large cohorts or groups of cases, standard methods for establishing significant relationships and extrapolating probable causation do not apply. New ways of assessing the level of probability that an environmental or occupational agent and a disease are related must be utilized.

As the basis for discussion, the definition of environmental or occupational disease is one caused or significantly contributed to by one or more toxic chemical or physical agents found in the environment or in the workplace, present at a level and under circumstances which permit undue absorption and retention in the body and which result in bringing these agents into contact with cells or organs sensitive to their toxic effects. These agents then cause an altered cellular and/or organ response which adversely affects organ function and as a result, the health of the person involved.

I. Clinical Medicine: State of the Art

An assessment of the state of the art requires a description of both the clinical medicine model as it has been taught and practiced over the years and the occupational (and environmental) medicine model. Most of the clinician's training, knowledge and skills, relate to the diagnosis and treatment of disease. Until recently little or no consideration was given to seeking a cause except in infectious diseases where the organism had to be identified in order to treat the patient. More recently genetic and dietary factors and cigarettes and drugs have been causally associated with diseases of the heart, lungs, liver and other organs.

In seeking a diagnosis, the physician attempts to determine if a disease is present, the organ or organs involved, and the nature and extent of involvement. The method used follows a specific pattern which all physicians learn during medical school and residency training. This consists of data gathering, drawing a diagnostic conclusion based on choosing the most likely disease, intervening with treatment, and observing the patient to determine whether treatment is successful. Data gathering consists of questioning the patient regarding past medical history, time when first symptoms were recognized, patterns and severity of symptoms, and other information such as family history and lifestyle. For some diseases, i.e. chronic pulmonary, hepatic, and lung cancer, physicians frequently take a history of family disease (genetic), diet, cigarette smoking, alcohol, drugs or trauma with a view towards establishing these as significant factors in the causation of the disease. Physical examination is then performed and based on it and the history, laboratory tests are ordered. These generally consist of tests to determine levels of organ function or dysfunction except in the case of infectious diseases where blood, urine, feces, or sputum analyses may be performed in a search for causitive biological agents. Tests are generally carried out with specific organs in mind and are to a large degree oriented toward defining levels of organ function. The diagnosis of disease is based on matching these findings with a known clinical syndrome, taking into account the pattern of disease prevalence in a geographic area within a particular socioeconomic or ethnic population known to the physician through clinical experience. Treatment is then prescribed and patients followed to determine whether treatment intervention has been successful. Reassessment of diagnosis generally occurs when the treatment does not appear to have resulted in a return to a healthy state. Since the key to diagnosing a disease as environmental or occupational is the establishment of cause, where this is not done through relevant history or laboratory tests, and particularly under circumstances where physicians are not trained in toxicology, epidemiology, or biostatistics, the diagnosis is only rarely made.

This is not true for all medical disciplines. The majority of occupational diseases are dermatologic in origin, so that dermatologists have long been aware of the cause or relationship between many skin irritants and sensitizers and dermatologic diseases. Allergists, very early, came to recognize that the environment along with genetic and other factors played an important role in disease causation. The development of scratch tests, intradermal tests, and RAST were all part of the search for etiologic agents. Physicians specializing in pulmonary disease and radiology also have known about pulmonary disease from the fibrogenic dusts and have related abnormal x-rays to occupational exposures. Psychiatrists also probe for environmental causes of behavioral abnormalities, but generally these do not include the physical or chemical environment.

II. Occupational and Environmental Medicine: State of the Art

The clinical methods used to diagnose occupational and environmental diseases include the identical components described in the state of the art in clinical medicine. In addition, however, the sciences of epidemiology, toxicology and biostatistics, and the occupational and environmental data base are utilized in arriving at a clinical diagnosis and, in addition, in finding the cause.

The problems involved in establishing environmental and/or occupational factors as causative are many and provide serious challenges to the physician. These will be discussed in detail, but two types of problems should be noted. Many agents can cause similar, if not identical, diseases of a particular target organ and when present together may greatly increase the possibility that disease will result. For example, the lungs of a welder who smokes and lives in an urban environment are assaulted by metallic and other fumes at work, and cigarette smoke and air pollution at home. The result is often chronic obstructive lung disease. Additionally, some toxic agents are available from multiple sources with each source playing a role in causing disease. Carbon monoxide is one of those and is present not only in many industrial settings but also in cigarettes and automobile exhaust. Where exposure is to relatively low levels of the toxic agent over a long period of time, the symptoms and abnormal findings may present very subtlely. In such cases there is also the problem of long latency between onset of exposure and disease. Since many toxic agents affect the lungs, liver or nervous system, synergistic effects of cigarettes, alcohol and drugs increase the difficulty of establishing major environmental causative factors. The problem is also made more difficult when the cohort under study is relatively small since many of these diseases, for example cancer, usually require large members and a long period of observation to separate "background" incidence from an excess due to exposure. The differences in risk of various populations because of cultural or socioeconomic factors may also be significant and are compounded by the variability of risk in groups of individuals within a population based on many factors, which will be discussed. The occupational and environmental medicine clinician faces all of the above problems when attempting to diagnosis a disease and its' cause. There is the added problem for the clinician of carrying out this activity on a single or a few cases where no statistical assessment is possible in the classical sense.

III. Clinical Diagnosis of Environmental and Occupational Disease and Its' Cause

The diagnosis of cause is based on establishing a high level of probability that a disease syndrome is related to an exposure. This can only be done by considering a multiplicity of factors which include the availability of the hazardous agent, the probability that it has entered the body, evidence that it is present in undue amounts and whether the individual is at enhanced risk for other reasons. In carrying out this estimate of the level of probability, factors which must be considered in the history include a toxicologic and environmental exposure assessment and the individual as a biological risk. Other data obtained include laboratory tests which measure levels of undue absorption of toxic agents, tests to determine the presence of abnormalities of function of organs or organ systems, and the physical examination.

A. Toxicologic documentation

Toxicology data gathering will be considered in detail in other presentations. Discussion here is confined to examining those components obtained as part of the medical history which help to determine if undue absorption has taken place, what the excess body burden might be and its' potential for causing disease.

The ability of an agent to enter the body through one or more portals should be determined in order to assess whether undue absorption might have occurred. Some chemicals in liquid or solid form enter only through the GI tract. Others, for example dioxin, methanol and many solvents, can enter through the skin and the lungs as well as by ingestion. The percent absorbed into the body after entry is also of considerable importance. For example, lead is absorbed to the extent of five to ten percent of the total ingested in most individuals, although in young children it may be much higher. On the other hand 30 to 50% of inhaled lead may be absorbed into the body and therefore represents a much greater threat to health.

The physical characteristics of the agent are also important factors in estimating body burden, particularly when considering aerosols and particulate. In most cases it is necessary for particles to be five microns or less in size in order for them to penetrate beyond the mucociliary defense mechanism into the lower portions of the lungs where retention time and therefore absorption may be markedly enhanced. This is not necessarily true, particularly of asbestos which, because of its' great length to width ratio, acts as a small particle. Aerosolization of substances may also enhance the penetration and dissemination of a toxic agent through the lungs. The temperature, vapor pressure and the molecular weight are additional factors acting to increase or decrease absorption into the body.

The ability of the organism to degrade, detoxify and excrete a substance determines to some degree the substance's availability to various tissues and organs, and its' ability to accumulate and to cause toxic reactions in the body. Accumulation and storage of toxic agents depends to some degree on the rate of excretion. Where the major routes of excretion are known, for example, through the kidneys, quantitation of body burden may be carried out by examining the urine for the agent or its' metabolic products. This has limitations since with many substances there appears to be poor correlation of urine levels with health effects and even with true body burden.

Tissue or organ storage is another factor in determining the probability that a significant health effect will occur. In the case of a substance like lead, 90% of it is stored in the bone, where it has little effect. The important site of action in this case is not the organ of storage, but other organs including the nervous system, kidneys and blood forming organs which are the major target organs for lead. This may also be true with other chemicals. This knowledge is critical since the measurement of effect will focus on target organs.

B. Environmental and Other Exposure Factors

It is essential to consider environmental exposure factors, specifically levels of toxic agents in air, water and soil, in estimating the probability that undue absorption has taken place and that a toxic agent is present in the body at a level sufficient to produce disease. Measurements of all three may be necessary not only to determine the levels of toxic agents but the areas contaminated and in some cases the source, which may be an industrial plant or stored toxic wastes. Where an industrial plant or other point source is suspect samples must be taken up and down wind from such facilities using prevailing wind speed and direction to predict areas of high and low concentration. We found soil and dust samples to be particularly useful in determining long term deposition and therefore chronic exposure of populations in an area. Soil samples taken at 16 compass points, at variable distances from a lead smelter and at variable depths, were highly correlated with the effects on the population around the plant.

Emission and production data may be extremely useful in predicting problems. In El Paso, Texas we predicted that children with lead poisoning would be found as a result of exposure to lead emitted in high concentrations from a smelter. This prediction was based on examining emission data which were reviewed for a completely different reason. Testing revealed large numbers of children living down wind to have undue body burdens of lead with many showing evidence of lead intoxication.

Changes in production process, materials used and emission controls are additional necessary pieces of information. Another group, a community of Mohawk Indians, were exposed to an estimated 25 million pounds of fluoride dumped on their island from an aluminum smelter. Information about changes in process with subsequent reduction in emissions made it possible to provide a reasonably good estimate of the levels of fluoride available to the population. As a consequence a study is being carried out to examine the health impact of fluoride on this exposed community.

Additional environmental factors which require consideration include the general characteristics of an area such as temperature, humidity and rainfall. El Paso's warm and dry climate enhanced the amount of lead dust present on surfaces and in the air, resulting in increased exposure of children to the lead. Most of the houses had dirt floors and the roads and playgrounds were unpaved, which also increased exposure. Other environmental observations which may be useful in predicting that a problem exists includes looking for damage to fish, plants or animals in the region, rapid deterioration of metals, fabric, and paints, etching of glass, or the presence of odors. On the island exposed to fluoride, much of the vegetation had died along with virtually all of the cattle, suggesting a high level of exposure of all life on the island.

C. Biological Host Factors

In every population there is considerable variability of risk among its members even at the same dose of an agent. While many factors affecting risk differences are unknown, some are well established and need to be assessed. An important consideration in assessing the probability of a causal relationship is whether or not those expected to be at highest risk as a result of factors other than intensity of exposure, are also the ones who appear to be the first and most seriously affected.

Genetic factors in risk determination have received little consideration but may be of importance. For example, individuals with enzyme deficiencies of G6PD or sickle cell trait are more susceptible to the effects of some toxic agents on the red blood cell. Others with alpha-1-anti-trypsin deficiency, asthma or allergies might be more sensitive to toxic inhalants. Another example, the finding that those with increased aryl hydroxy hydroxylase (AHH) induceability, a genetically determined trait, are at greater risk to PAH induced lung cancer is of considerable importance. It is important that much greater research efforts should be made to find other factors which increase or decrease individual susceptibility to disease. Elaboration of additional genetic risk factors will enhance our ability to quantitate the variability of risk in population groups as has been done with cigarette smoking, cholesterol and hypertension in cardiovascular diseases.

Age must be considered since the very young and the aged, particularly those with chronic diseases, appear to be at considerably higher risk. Studies showing the causal connection between various dyes and bladder cancer also revealed that those initially exposed before the age of 25 as compared to those who began their exposure after this age, even where the duration of exposure was the same, may have been at increased risk for the development of cancer. Very young children appear to have increased sensitivity to many agents, particularly metals and solvents. In the case of lead for example they exhibit much more serious acute and chronic disease effects at lower blood lead levels than adults. The reasons for this include greater absorption from the GI tract (up to 40% of ingested lead), a poorly developed blood-brain barrier, and nutritional factors (decreased calcium or iron). The fetus is probably the most sensitive of all particularly when the toxic agent can cross the placenta - for example, lead, mercury, and lithium. Sex differences may be important where individuals are exposed to hormones, drugs, or chemical wastes or fumes which may affect reproductive organs. Additionally the physiologic stress imposed by pregnancy may be a factor in altering risk.

Those suffering from chronic diseases also represent a cohort generally at higher than normal risk when exposed to a wide variety of toxic agents depending on the organs affected and whether they are also target organs for the toxic agents. This is not only true in relation to causing further damage to organs with already reduced functional capacity but in the case of organs serving as excretory routes, such as the kidney, may result in increased body burden as a result of increased retention of a toxic agent. Chronic liver disease may lead to decreased detoxification of toxic substances, again increasing retention in the body.

Habits and cultural differences may alter the probability that an individual exposed to a toxin will develop a disease or that the disease developed is primarily related to a toxic exposure. These include cigarette smoking (possibly the most confounding variable of all), alcohol intake, nutrition, and socioeconomic status. Each may enhance the potential for developing disease due to exposure to environmental toxic agents. Where the exposure is small and the "habit factor" is great, there may be difficulty in estimating the role of the environmental factor in disease causation.

There are many other factors but only two will be mentioned. The first is a positive past medical history before exposure. The history of disease present before exposure doesn't necessarily reflect reduced impact by the toxic agent. It only suggests that health problems were present before exposure and that the person may in fact be at high risk because of lowered adaptive capacity. It does not mean that the environmental agent has not caused disease, because in fact those with previously diseased target organs generally are at enhanced risk. The second is drug intake, since these may be synergistic, or antagonistic with toxic agents or may themselves be major causative factors in disease.

D. Effects on Biological Systems

Examinations for the effects on biological systems usually follows the determination that an individual or community has been exposed to higher than normal levels of a toxic agent and that the toxic agent may have been absorbed into the body. It also usually follows measurements of undue absorption, although with some materials this is extremely difficult if not impossible, for example, asbestos. The presence of toxicants in the body in undue amounts suggests that there has been undue exposure but gives no information regarding negative health effects, only the probability that such effects may have occurred. The absence of findings of undue absorption, particularly of a toxic agent which is stored where it cannot be measured, rapidly metabolized, or excreted from the body does not even provide that kind of information, so that frequently environmental data have to be relied on to conclude that enough had entered and passed through the body to predict that undue effects on health may have occurred.

A determination of whether or not negative health effects have resulted requires taking the history of exposure or residence in an area in which toxic wastes were present, a past history of health status, and the development of symptoms. From these, and with some knowledge regarding which toxic agents were present in the individual's environment and the major target organs generally affected by this agent, relevant diagnostic procedures can be carried out. This is then followed by a physical examination and from all of these a determination made that a disease is present or not, which organ systems have been affected, and the nature and extent of the effect. These constitute a disease syndrome and, particularly where multiple organs appear to be involved, an attempt is made to relate all of them to a common or single etiologic factor. In some cases, there are unique factors or findings present which permit the determination that a "classic" environmental or occupational disease syndrome exists.

E. History

The classic clinical history obtains information about the time of onset, the progression of disease, and the duration and/or periodicity of symptoms by asking these and other questions used in standard medical practice. In addition, however, a history is taken to determine if others in the community have been affected, the relationship between the onset of symptoms and date starting residence in the community as well as information regarding environmental exposure and toxicology previously discussed. A history of past health and one which may elicit genetic abnormalities is important in assessing the risk of the individual as a biological entity.

Symptoms are critical in making a diagnosis and eliciting them is an area where most physicians possess considerable skill. Where it is suspected that a particular substance may be involved, questions can be directed toward determining whether symptoms known to be caused by such a substance were or are present. This is often the area which is not considered in general medicine. Many cases of lead colic are diagnosed as cholecystitis, ulcer or appendicitis and in some cases individuals are subjected to multiple surgical procedures before a diagnosis is made. Where symptoms suddenly surface, are not continuous or have a temporal pattern, possible changes in environmental exposures should be sought. In one situation, children who were unknowingly suffering from chronic lead poisoning suddenly developed acute symptoms. This was eventually related to the discontinued use of a baghouse with a marked increase in airborne lead in the air of the community. Under some circumstances individuals leaving a community who may have "accommodated" to an environmental stresser, that is, do not have acute disease because exposure occurs over a long time period, suddenly become ill on returning to the area. In these cases a syndrome similar to fume fever may occur. This is a disease which occurs on Monday when workers who have been away for two or more days are exposed to zinc, brass or other metal fumes. Where numbers of people are found to have onset and progression of symptoms at about the same time and where the symptoms are similar, a common etiologic factor should be suspected. This is certainly the case in infectious disease epidemics and may also be the case where environmental agents are the cause.

F. Diagnostic Test

In considering the types of diagnostic tests to be performed many factors must be considered. These include:

(a) Numbers of individuals to be tested.

This is the first and often, the most critical consideration when carrying out diagnostic testing particularly in a community where a common environmental etiologic agent is being sought. If it is felt, based on all of the considerations previously discussed, that a large number of individuals may have been exposed, or, if there appears to be a high incidence of clinical disease in the community, it may be necessary to test many hundreds of people. Where this is the case sharply reduced questionnaire length in taking histories, sophistication of tests and time that the tests take to perform, become critical considerations. Where only small numbers of individuals are being tested this does not obtain. The decision of whether to test only those who appear to have been most heavily exposed to determine whether there has been negative health impact on them as "test cases" for the community is frequently a difficult one. There may be some danger in having the rest of the community wait until initial testing on the smaller group is carried out.

(b) The location of the community.

The location of a toxic dump, contamination of a water supply or other environmental problem is frequently in a community which is physically isolated. Limitations of personnel, transportation of equipment and required storage of specimens are among many factors which must be considered in deciding which tests will be carried out.

(c) The cost of the test.

This may often be the limiting factor in studying a community particularly when the numbers to be tested are large. For example in screening for lead a zinc protoporphyrin test (ZPP) which requires only a drop of blood, costs only a few cents to do and is easily reproducible on site may be much more desirable than a blood lead which costs considerably more, or a test for ALAD which in addition to the cost must be carried out promptly. There are many other examples of ways in which cost can be reduced without sacrificing to any signficant degree the usefulness of a test. The use of a PA chest x-ray instead of including a lateral view reduces cost considerably in a first round of testing as is the use of a hematocrit or hemoglobin instead of a CBC in screening for anemia.

(d) Acceptability by the community.

There are many test that groups and communities will not accept easily, particularly those of an invasive nature, such as tissue biopsy or bone marrow aspiration. Tests which may be painful such as peripheral nerve velocity conduction, EMG, or proctoscopy will also encounter considerable resistance. Others which may cause embarrassment or carry a stigma, for example production of a sperm specimen or "psychological tests" also often encounter difficulty. In one study, people informed that a neurobehavioral battery of tests was similar to the testing of their reflexes accepted testing while mention that psychological tests would be performed resulted in mass refusal.

(e) Time needed to complete the test.

A neurobehavioral battery may require two or more hours of time to perform, trained personnel to administer the tests, and skilled professionals to score and interpret them. Where large groups are being tested this becomes almost prohibitive because of cost, personnel and time.

(f) Morbidity and mortality.

Most testing procedures carry with them a very small risk. Some, including invasive procedures or those where dye injections are required, carry an increased morbidity and in some cases even a small mortality risk. Invasive procedures, tests such as EDTA challenge and others should only be carried out after serious consideration of their need, and only by skilled and experienced professionals and where some follow-up observation is possible.

(g) Test sensitivity and specificity.

The determination of whether or not to carry out tests which may result in many false positives as compared to many false negatives is generally determined by the importance of not missing any cases in the community. During the initial screening of large communities of individuals, those tests which are more "sensitive" that is which include all positive cases in addition to some false positives are generally more desirable. During a second round of examinations after the entire community has been screened, tests specific for a disease in which only few false positives result may be more useful.

(h) Personnel and equipment.

Depending on the location of the community to be examined these may be important considerations. Equipment carried into the field must be durable, stable and portable. In urban communities where facilities and equipment are easily available and repair of equipment obtainable this is not a serious consideration. In many cases, however, toxic waste victims are in out-lying or rural areas and this becomes important. The required number of personnel and their levels of training and expertise frequently determine the cost of testing as well as the numbers of individuals that can be examined. Local individuals in communities being studied often can be trained to assist in carrying out many necessary procedures if large numbers of people are being examined.

(i) Test preparation.

Those test which require special procedures such as restricted diets or a 24 hour urine collection are often very difficult to obtain. In examining groups of workers exposed to arsenic it was necessary for them to stop eating fish or seafood which might contain this chemical for three days before the test in order that the urine might be appropriately examined. Since, in this community, fish and other seafoods are an important part of the diet this was very difficult to achieve. Fasting blood is frequently difficult to obtain in working populations particularly if they are on shift rotation, as are meat free and other types of special diets.

A wide variety of diagnostic tests are used in clinical medicine. To a large degree they examine the level of function of organ systems and when these are inappropriate or abnormal a conclusion is reached that disease of this organ may be present. Where multiple organ systems are found to be abnormal, an attempt is generally made to integrate these findings into a single disease diagnosis. Environmental and occupational medicine utilizes in addition a wide variety of tests to determine whether toxic agents are present in the body and the levels at which they are present.

The first group of tests include those which measure total body or organ concentrations. These are the most specific tests since they look for the presence of the toxic agent in order to determine its' presence and concentration in the body. They are carried out by examining the appropriate body fluids including the urine, blood, spinal fluid, saliva, sputum, hair, nails, feces, sweat or exhaled air. While these are specific in determining whether the agent is present in the body they may not provide accurate information about dose or any information about the toxic effects on body organs or tissues. Lead for example may be measured in the blood, urine, feces, hair and nails, but in fact 90% of all the lead in the body is contained in the bone where without carrying out a bone biopsy it cannot be directly measured, although x-ray findings of density in the long bones provides qualitative evidence of a heavy bone burden. While there is some evidence that blood lead provides important information regarding whether levels currently present are toxic or not, it too only indirectly provides information regarding how much is in the body and it also, fluctuates depending on when exposure took place so that levels which may have been high in the past may be normal when obtained. For this reason other body tissues have been used to assess chronic exposures such as teeth in children, which is a measure of lead during tooth development or zinc protoporphyrin which reflects the replacement of iron by zinc in the red blood cell and therefore a measure of body burden over a period of months. Hair lead provides some information regarding past body burden but has not been shown to correlate highly with toxic effects. One additional method for assessing levels in the body is a provocative chelating test where urinary lead is measured before and after a dose of EDTA. A sharp increase generally means that other tissues, particularly the bone, may contain large amounts of this metal.

A considerable amount of work has been done on the usefulness of measurements of toxic agents in various tissues in estimating body burden. Studies carried out on Minamata Bay victims, individuals who ingested large amounts of mercury from contaminated fish, found that blood and hair provided reasonable estimates of body burden. In a study of workers in multiple plants who were exposed to lead, we found that ZPP correlated reasonably well with exposure and at an FEP level of 50 micrograms per dl of blood no workers were found with blood lead levels above 40 micrograms per dl of blood. In another study, workers exposed to arsenic and with arsenic intoxication were followed with multiple examinations over a three year period. We found a highly significant correlation between exposure and hair levels of arsenic. We found no correlation with urine. Urinary arsenic appears to be a useful measure only in determining whether this chemical had been absorbed into the body over the past 72 hours. The hair on the other hand was a much more accurate reflection of the amounts absorbed by the workers in our study. While previous studies claimed that external arsenic contaminated hair specimens and made it impossible to use hair as a tissue for measurement, we did not find this to be the case. This entire area is one where a considerable amount of research effort should be expended. Studies, particularly those in which levels of toxic agents in various tissues and fluids are compared with exposure of individuals and symptoms and disease, should be carried out.

A second group of laboratory tests provides us with an indirect measure of body burden by examining urine and in some cases feces for metabolic degradation or by-products. Toluene, for example, exits through the urine as hippuric acid which can be measured. There are many other similar examples. Others which in fact are measures of interference with biochemical function also provide indirect estimates of body burden. For example, lead at almost any concentration interferes with ALAD which is essential in the production of heme and processes delta aminolevulinic acid in the body. The reduction in the enzyme ALAD interferes with this process leading to an increase in delta-ALA in the blood and urine where its levels can be measured. Levels of ALAD can also be measured although with some difficulty and have been shown to have an inverse linear relationship to lead at a level as low as 10 micrograms g/dl in the body. Another example is acetylcholinesterase which is decreased in the body in the presence of organophosphate pesticides. These latter tests, measure body burden and also the level of interference with physiologic function and therefore increased physiologic stress if not disease.

The next group of tests, those which measure the health or disease of the major organs and organ systems, are familiar to all medical practitioners and are used with great frequency in the practice of clinical medicine. These measure organ function in a variety of ways in order to determine whether they are malfunctioning and therefore are diseased.

The first group are those which measure impaired or diminished function, such as muscle strength, nerve conduction or spirometry. In these tests, muscle strength is examined to see whether it is diminished, nerve conduction to determine whether there is slowing of the impulse and spirometry to look for the inability of the lung to contain a normal volume of air or to force it out of the lungs as rapidly as it should. Another example of reduced function includes audiometry, that is, hearing ability at certain frequencies.

Another group of tests examines organ systems for increases in function. These include hyperirritability of muscle or hyperactive reflexes on the physical examination. A third group measures deviation from normal patterns. These include EEG, and ECG as well as x-rays.

One other group that should be noted are tissue tests. These include lymph node, liver, lung, muscle or other biopsies or bone marrow examinations. They may be examined for levels of toxic agents, for example, by ashing tissue, or for histopathologic changes which, while frequently nonspecific, may aid significantly in diagnosis. An example of this is beryllium which causes a granulomatous lesion similar to that of sarcoidosis, where ashing of the tissue and testing for the metal can pinpoint the diagnosis.

The problem with tests of organ function is that they are generally nonspecific since organ systems can respond only in a limited number of ways to toxic and other disease producing agents and differentiation of cause is often impossible from tests alone. It is however an important component of the "clinical syndrome" when considered together with the history and all of the other data which have previously been discussed.

G. Physical Examination

The physical examination in our experience has proven to be the least sensitive group of tests in providing information about toxic exposures. This may be because surfacing of abnormalities in such examinations generally occurs only in severe acute, or far advanced chronic, disease. Physical examination of the skin, however, may be extremely useful in some conditions. An example is chloracne, particularly in children after exposure to dioxin, PCBs and other similar chlorinated compounds. Another is keratotic skin lesions and skin burns from arsenic exposure which can be useful in making a diagnosis. Examination of the skin can also be very important where exposure to carcinogens may be suspected or has occurred. More than a million skin cancers occur each year, more than all others combined and because they are visible they are frequently detected early. They therefore represent an extremely important signal lesion of effect as well as exposure since most carcinogens are carcinogenic for more than a single organ. Neurologic examination may also sometimes reveal abnormalities where there are few or no symptoms including abnormal reflexes or movements, or muscle atrophy. In general the physical examination at best serves to confirm the history, symptoms, laboratory and other test findings.

IV. Clinical Syndrome

The large number of variables operating in the development, intensity and extent of disease syndromes makes it extremely difficult to describe a "classic" syndrome resulting from absorption of a toxic agent into the body. For example, the form of the agent can often determine the level of absorption and action in the body. Organic forms of metals such as lead or mercury may cause a disease picture very different from that occurring from an inorganic form. Also in the latter group the more soluble the form the greater the absorption so that intensity and extent of disease may vary greatly. Further, absorption of a large dose over a very short period of time may result in a remarkably different "syndrome" than lower level exposure over a very long period although the total dose may be equivalent. The body generally reacts to toxic agents as it does to any other physiologic insult. For example, an acute blood loss from 16 to 8 grams in a period of minutes or hours frequently leads to shock, renal shut-down and often death. On the other hand individuals who develop anemias over a long period can be seen ambulating with 5 or 6 grams of hemoglobin. Children living close to and downwind from a lead-zinc smelter were found with levels of blood lead exceeding 170 micrograms per dl of blood. While some of them were anemic and most revealed evidence of brain damage, they appeared to be functioning physically. Many were lethargic, had low grade gastro-intestinal complaints, were irritable, and had headaches, but much of this was ignored because these symptoms were not dramatic. In Chicago on the other hand, a child eating flaking, heavily leaded paint and developing a level of 170 micrograms almost certainly would present with encephalopathy, cerebral edema and coma.

In addition to the differences between chemical forms and acute and chronic exposures, the populations at variable risk will exhibit very different syndromes from each other. The child with acute lead poisoning will exhibit a very different set of symptoms from an adult. The chronically exposed children that we saw, on the other hand, exhibited relatively similar symptoms to the adults except that the amount of brain damage found was considerably greater. The reason for this may be that in young children more than 5%, in fact up to 30 or 40%, of the lead ingested may be absorbed into the body. Also, in very young children the blood-brain barrier is not well formed, permiting more lead to enter the brain. Additionally, the increased metabolic activity of growing children is also a factor. In both groups, however, adults and children, the ultimate target organ does appear to be the kidney.

Some "classical" clinical syndromes have been described and are almost pathognomonic in that they have very unique features. Asbestos is certainly a classic case in point. Montague Murray in 1900 was able to relate the pulmonary disease he found on examination and at autopsy of a worker to asbestos exposure. The worker he saw pointed out that others working in the same area of the plant died from lung disease and had the same symptoms he had. Massive calcification of the pleura which Murray had never seen before led him to his conclusion that asbestos inhalation caused the disease. The clinical syndrome of a history of exposure to asbestos, asbestos bodies in sputum or tissue, progressive shortness of breath, a restrictive and ultimately obstructive defect of pulmonary function, a diffusion defect, bilateral lower lobe fibrosis on x-ray with, in some cases, honeycombing of the lung, pleural plaques on the diaphragm, thickening, fibrosis, and calcification of the pleura, clubbing of the fingers and rales at the bases are all components of the syndrome, although rarely are all present in one case.

Mesothelioma and cancer of the lung and other organs may also be considered part of this syndrome, that is diseases caused by asbestos. With some other agents there are also so-called classical syndromes. Fume fever's characteristics have been noted. Other fibrogenic dusts besides asbestos such as beryllium, silica and coal produce fairly characteristic syndromes when fully developed. Even in these cases however, other diseases many be mimicked. For example berylliosis looks very much like sarcoidosis and is a systemic disease involving the same large group of organs. History of exposure, ashing of tissue and examination of it for beryllium confirms the diagnosis. Lead encephalopathy in children would be considered "classical" except that encephalitis from other causes may present a somewhat similar picture. The history of pica in a child living in an old tenement with flaking paint, exposure near a lead smelter, finding of lead in the gastro-intestinal tract, evidence of increased blood lead, anemia and other similar findings would help to differentiate this syndrome. Some toxic agents tend to have a predilection for certain systems. Metals generally affect the cytochrome oxidase system, inactivate sulfhydryl groups and interfere with cell metabolism. The effects are related to this. Many of them are also excreted through the kidney so that this organ in addition to the central and pheripheral nervous systems is generally affected. Here too, however, there may be endless variations.

Solvents are another large group which have some general characteristics although there are wide variations in clinical syndromes. They tend to enter the body through multiple routes including the skin. Because of their chemical nature they also tend to defat the skin, affect the liver and as a group generally tend to have narcotic properties and affect the central and peripheral nervous systems. They can affect other organ systems as well, but particularly the liver so that a "classical" clinical syndrome for toluene might include a cracking of the skin, central nervous system depression, fatigue and dizziness and possibly liver enzyme changes.

A heightened level of consciousness of the possibility that environmental factors might be closely related to disease is critical to considering the "disease syndrome". During an evaluation and examination of a group of workers heavily exposed to chlorinated hydrocarbons medical records of evaluation and treatment by physicians who saw them during the first year following exposure were reviewed. Some had been diagnosed as having skin problems by a dermatologist, bladder problems by a urologist, hepatic disease by an internist and acute depression and anxiety by a psychiatrist. There was no intergration of these organ diseases into what was clearly a clinical syndrome resulting from exposure to a toxic agent.

It would seem appropriate to end this presentation with some recommendations in addition to those already made, for future activities in environmental medicine. The first is to qualitatively increase the didactic and clinical offerings in occupational and environmental medicine to medical students and residents in medicine. Continuing education programs for physicians should certainly contain lectures and other educational offerings dealing with the impact of environment on health, with suggestions about expanding the clinical history in office practice to include the work and community environment. This is particularly relevant for those who completed their medical education before it became generally recognized that environmental exposures were often causally related to clinical disease.

Finally a significant effort should be made to formally train physicians as specialists in environmental medicine. Since occupational medicine is a microcosm of environmental medicine it represents a discipline where such training is already being carried out. Until the development of the NIOSH educational resource centers, less than 700 certified physicians were practicing occupational medicine as a speciality and of those less than 200 received formal training in this specialty. Physicians trained in occupational medicine are required to develop skills in environmental health, biostatistics, epidemiology, and toxicology and to have some knowledge of industrial hygiene and industrial process. Physicians seeing individuals with diseases that could or might be environmentally related will attempt to answer questions such as:

1. Is the temporal relationship of exposure to developing pathology reasonable and logical?

2. Was the dose of the toxic material sufficient to cause the pathology?

3. Are measures of body burden in the range where disease is known to occur?

4. Is the pathology of the nature of what would be expected of this material?

5. Have other diseases that might cause the same pathology been ruled out?

6. Are those in the population expected to be at higher risk the ones who develop the disease?

7. Are others similarly exposed, similarly affected?

In a paper appearing in January 1971 (Arch. of Internal Medicine, Volume 127) on air pollution and physician responsibility, Carnow stated:

1. The physician should become knowledgeable regarding the relationship of environmental factors to disease.

2. The physician should search for clues to the environmental-disease relationship in the clinical patterns of disease so that definitive epidemiological studies can be carried out as extensions of these clinical findings.

3. The physician should support and participate in studies searching for the complex physical, biological and psychosocial etiologic factors in the changing patterns of disease morbidity and mortality by coupling medical insights with computer technology.

4. The physician should intensify research into the nature of man's interaction with his ecologic system as it relates to health and disease and lend support to the training of physicians who will become environmental specialists and develop this new discipline. These experts can then with engineers, biologists, ecologists, and others of like disciplines assess the health consequences of social and environmental changes and based on a cost benefit analysis (the cost to health as compared to the benefits and comforts given to man determine the value to man of these changes).

5. The physician should develop a therapeutic approach which will consider and utilize environmental etiologic factors. The evidence presented suggests that some high risk groups are already known and have been isolated. These are particularly the very young, the

> poor, and the older age groups with chronic heart and lung disease and those with asthma. Thus evaluation of a chronic bronchitic or cardiac patient should consider the risk factors including disease severity, pollution hypersensitivity, and the general degree of environmental degradation. This would enable the physician to classify the patient as to degree of pollution risk and to use this as a guide for regulating activity and medical dosage in relation to pollution levels. Individuals found to be environmental hyperreactors might even be advised to leave communities where pollution frequently achieves high levels.

Finally, we cannot procrastinate with pollution as we did with cigarettes, where our timidity has cost many thousands of lives. Physicians must become involved and begin to deal with the extremely complex problem of assessing the degree of environmental deterioration which has occurred along with its impact on health. We must begin to grapple with ways to structure a viable environment for future generations. We must see to it that our organizations are intimately involved in a positive way with these and other major problems of our times. Members of the medical profession must give time and energy to serve on legislative and advisory councils, committees, and boards in order to assure that human health will be a major consideration in the deliberation of engineers and legislators in defining legislative needs. Never before has society presented the physician with so wide a field of activity and with so much opportunity to play a key role. This is the time for the physician to become the asklepios politkos visualized by Plato.

Given the enormous toxic waste problems we now face and the increased community awareness and concern regarding the impact of environmental contaminants on health this course of action is even more relevant today than it was a decade ago.

MEDICAL AND TOXICOLOGICAL CONSIDERATIONS IN DETERMINING PROBABILITY OF CAUSATION OF ENVIRONMENTAL DISEASES

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DEFINITION OF ENVIRONMENTAL DISEASE - Caused by toxic chemical or physical or biological agents in the workplace present at a level or under circumstances which permit undue absorption into the body causing and altered cellular and organ response which adversely affects organ function and as a result the health of the persons involved.

PROBLEMS IN DIAGNOSIS

a) Multifactorial.

b) Long latency and low level long term development of chronic disease. Rarely acute.

c) Irreversibility.

d) Unlike occupational exposures - different physicians seeing individuals affected with different diagnoses.

e) Multiple sources of single agents.

f) Multiple substances impacting on a single target organ.

g) Inability to use epidemiology in the classic sense for the single or few cases.

DIAGNOSIS OF CAUSE - Based on level of probability.

How is this Done? By considering major factors relating to ABSORPTION - RISK - EFFECT.

1) Toxicologic assessment - Probability of undue absorption
2) Environmental assessment - Probability of undue absorption
3) Other exposure factors - Probability of effect - risk
4) Biological assessment of those exposed - Probability of effect - risk
5) Biological end point - assessment of effect

TOXICOLOGIC ASSESSMENT

1) Intrinsic toxicity of agent
    a) In relation to other chemicals
    b) In relation to other forms of the chemical

2) Portals of entry of agent and per cent absorbed

3) Form of agent - gas, particle - size, liquid

4) Biological 1/2 life

5) Tissue or organ of storage

6) Known target organ(s)

7) Metabolic products

8) Rates and modes of excretion

ENVIRONMENTAL & INDUSTRIAL HYGIENE ASSESSMENT

Determine:

1) Emission and production data of plants in the area.

2) Changes in processes, materials, controls and when these occurred in industries whose wastes were dumped or which were in the area.

3) Degree of containment through measurements of a) air, b) dust, c) water, and d) soil samples for toxic agents.

4) Probability of containment by noting a) depth of waste burial, b) porosity of soil, c) level of water table, d) packaging of wastes, and e) length of time it has been in dump.

5) Probability of physical contact and entry through inhalation, ingestion or skin by examining:

    a) Availability of water for bathing or drinking
    b) Use of land for gardening, recreation, landfill
    c) Proximity of homes, schools, walkways
    d) Characteristics of climate (e.g. dry, dusty)

OTHER EXPOSURE FACTORS

1) Time of first exposure
2) Duration of exposure
3) Temporal relationship between exposure and disease
4) Other exposures - particularly those affecting same target organ

BIOLOGICAL (HOST) FACTORS

1) Genetic Factors
2) Age
3) Sex
4) Chronic disease particularly of same target organ
5) Habits - cigarettes, alcohol, nutrition

BIOLOGICAL END POINT (HEALTH EFFECT)

1) Measures of body burden (not a measure of effect)
2) History of onset and progression of disease pattern - temporal relationship
3) Physical examination
4) Abnormal organ function - Laboratory and other tests
   - Pathologic specimens

QUESTIONS

1) Is temporal relationship reasonable and logical?

2) Was the dose of the agent sufficient to cause pathology?

3) Is the organ affected and the pathology what might be expected from this agent?

4) Have other agents which can cause the same pathology been ruled out?

5) Are those expected to be at highest risk due to dose or other factors, the ones who develop the disease?

6) Are others, similaly exposed, similarly affected?

Mr. LEVITAS. Thank you very much, Dr. Carnow.

Dr. Karrh, I think it would be well—I just had a discussion with Mr. Molinari about the procedure I announced. Since I take it you will be addressing from another point of view some of the points that Dr. Carnow has made, we will hear from you at this point.

Dr. KARRH. Mr. Chairman, members of the subcommittee, my name is Bruce W. Karrh. I am the general director of medical, safety and fire protection for the Du Pont Co. I am appearing today on behalf of the Chemical Manufacturers Association, CMA, a nonprofit trade association whose company members represent more than 90 percent of the productive capacity of basic industrial chemicals in this country.

We appreciate the opportunity to present our views on the compensation of persons injured by exposure to hazardous substances. This is an important subject and we are taking a constructive role in reaching sound public policy on this issue.

CMA strongly believes that individuals harmed by exposure to hazazdous substances should be compensated by those who have caused their injuries. We are concerned about anyone injured from such exposures, and we support the right of an injured individual to receive fair and speedy compensation. However, as I will discuss, we have not seen evidence of a significant health problem from hazardous wastes. We fully agree that concerns in this area require a thorough scientific review, but we also believe that in view of the review processes now moving forward, it would be precipitous for Congress now to enact pending proposals to modify existing compensation systems.

Compensation for nonworkplace injuries traditionally has been handled through the common law tort system of the various States. It has recently been asserted, however, that new approaches are needed because the existing tort law system is inadequate to deal with waste site injuries.

These assertions are based on the assumption of a major public health problem from waste sites. Proponents of this view have assumed that large numbers of people are being harmed by waste sites and that the existing legal system will not be able to handle massive numbers of claims to provide redress to those injured. However, this fundamental assumption of widespread harm from waste sites has not been closely examined.

Last year we created a special work group to determine if there was a relationship between waste sites and widespread harm. I served as chairman of this work group. We found that claims of such a relationship run counter to the experience of CMA member companies. Moreover, independent epidemiological surveys have failed to detect significant health problems.

In my testimony today, I will share information that led CMA to this conclusion. I also will describe what CMA is doing to increase our knowledge about potential health problems resulting from waste sites, and in particular I will report on two recent developments which should provide needed data and analysis upon which more informed judgments can be made in the future.

In our consideration of the issue, we found only two valid epidemiology surveys of populations relative to waste disposal sites.

Mr. MOLINARI. Might I interrupt for a second?

Is it your intention to read verbatim the report and testimony you have submitted?

Dr. KARRH. It is my intention to read my oral testimony I have here, Congressman.

Mr. MOLINARI. Are you going to be addressing in your statement the concerns raised by Dr. Carnow?

Dr. KARRH. I had not planned to in my oral statement. I would be glad to respond to questions. I did not know what statements Dr. Carnow was planning to make prior to the hearing, so I could not be prepared to address those.

Mr. MOLINARI. Well, Mr. Chairman, I thought that Dr. Karrh was going to review the statements made by Dr. Carnow and perhaps add to those by his own experiences. And, Mr. Chairman, we have a copy of the testimony here. We have had an opportunity, ourselves and our staff, to read it.

Subject to the biddings of the Chair, I would like to get into specific discussion between these two gentlemen so we can see where there are areas of agreement and disagreement, and then go on from there. We have the testimony.

Dr. KARRH. Mr. Chairman, I would like to respond by reading a paragraph out of your letter to me requesting me to testify.

Mr. LEVITAS. Yes.

Dr. KARRH. And show that I am trying to respond to what I was asked to do.

Mr. MOLINARI. You have responded. I am not suggesting you are not responding. Your response has been received by the subcommittee. We do have a copy of it before us. I think it is comprehensive. You are reading a digest of what you have said in this prepared report.

Now, if you want to continue, I have no objection to it. I just thought we might save time and get into the area that I would like to see us get into. If you prefer to read it, go ahead.

Mr. LEVITAS. What is your preference, Dr. Karrh?

Dr. KARRH. Naturally my preference is to continue as I was prepared to proceed.

Mr. LEVITAS. Then you may proceed in that fashion.

Dr. KARRH. The second study that we looked at investigated the health effects, on the general population, of chemical exposure in highly industrialized Contra Costa County, CA.

This 4-year study, which was conducted by the California Health Department cancer epidemiology unit, was financed by both State and Federal funds. It found that smoking, and not air pollution or chemical exposure at work or from dump sites, was the most likely explanation of higher rates of lung cancer in Contra Costa County.

During the course of the study, we discussed some of the studies that had been done including the studies of the residents in Triana, AL, that were exposed to DDT. CDC could not find any health effects attributed to DDT, but did determine that the residents had absorbed DDT into their system.

These residents have been identified as a group of people that can be studied prospectively, and CDC plans to do such a study to determine if health effects are occurring as a result of the previous exposures to DDT and absorption of the material.

The CMA group I chaired also reviewed a number of studies conducted by the Government and by industry to determine whether occupational exposure to toxic chemicals has resulted in an increased incidence of chronic disease. Based upon our own experience and the available exposure data, we believe that, in general, such employees would have been exposed to higher concentrations of chemicals than the population around waste disposal sites.

Hence, occupational exposure and health data should provide an indication of the health effects that might be expected if similar exposure patterns were encountered by the public. Since we saw no significant increased incidence of chronic disease among employees in the chemical industry, we concluded that widespread increased incidence of chronic disease among the public is also unlikely. Our studies have also found that chemical workers have a lower death rate from cancer and from all causes combined than the general public.

For these reasons, CMA's view is that evidence of a national problem deriving from public exposure to hazardous waste is not at hand. CMA also believes, however, that further scientifically sound information is needed. We also know the public needs reassurance that measures are underway to resolve the questions that have been raised. However, since we have seen no evidence of significant health effects, we have time to do the studies required.

CMA and its member companies are committed to cooperate in efforts to scientifically determine whether and to what extent there may be a relationship between waste sites and illnesses. We have recently undertaken two major efforts to accomplish this.

The first is the successful settlement of a lawsuit brought by the Environmental Defense Fund [EDF] and joined by CMA to get the Government moving on health studies. The second is the completion of a feasibility study on how the best available data and scientific expertise could determine the nature and extent of any such effects.

In section 104(i) of CERCLA, Superfund, Congress called for a scientific program to analyze waste site/disease relationships. Congress also required establishment of a new "Agency for Toxic Substances Disease Registry" to administer this program.

Two years after CERCLA's enactment, the Government had neither established the new agency, nor released significant amounts of Superfund moneys to support the study program by other agencies. It was in light of these failures that EDF and CMA filed their lawsuits.

This important case was settled on May 27. Even before the settlement, the Government had moved to resolve one significant point. On April 25, 1983, the Government formally established the new agency.

The parties then moved quickly to settle the remaining issues. Some of the most important provisions of the consent decree are listed in my testimony and will not be repeated at this time.

Another effort that CMA undertook was the feasibility study that we contracted for in March 1983 with the organization Universities Associated for Research and Education in Pathology. This study has now been completed. It contains important conclusions that bear upon the subject of assessing possible waste site/disease relationships and will be of great potential assistance to the new health agency in carrying out its function. This study was released on July 7, and a copy of the study will be submitted for inclusion in the record of this hearing.

Universities Associated for Research and Education [UAREP] is a consortium of 15 major universities. It was created in 1964 at the request of the National Academy of Sciences and the Department of Defense to do research in the field of human disease.

UAREP has concluded that within 8 to 10 months, it will be entirely feasible to carry out a comprehensive review to evaluate the substantial body of available data for the purpose of permitting an interim conclusion as to the nature of any health effects which may be associated with waste sites.

The objectives of this study are listed in my testimony.

The feasibility study also contains a detailed organizational and procedural outline for carrying out the comprehensive review study. This will require an executive scientific panel, two task forces, and a full-time scientific and support staff secretariat.

CMA is committed to the feasibility study's recommendations to carry out this important and comprehensive review. We believe this study deserves the broadest possible sponsorship and financial support. We are now actively engaged in seeking this support from environmental, academic, Government, and business groups. CMA is committed to beginning this study by September of this year.

Although we have not seen evidence of a significant health problem, and while the limited evidence we have seen makes us believe there is not likely to be a significant problem, we fully agree that the public concern in this area requires a thorough scientific review.

In light of the substantial scientific review processes that are now moving forward, CMA feels it would be precipitous for Congress to enact any of the pending proposals to modify the existing compensation system related to waste sites. These proposals would impose new taxes and establish new bureaucracies, and they tend to ignore established principles of causation. Yet they are based merely on assumptions of need for such measures, and are being proposed at a time when programs are actively getting underway to test such assumptions.

Dr. Carnow mentioned the Black Lung Act. We learned, I think, considerably from the black lung experience. When the legislation was passed, it was estimated there would be 50,000 cases of black lung at a total cost of $500 million. The experience to date has been about 600,000 cases of black lung that have received compensation at a total cost of about $25 billion, and a study by GAO that was recently released indicated that between 80 and 85 percent of these claims were nonmeritorious as far as having disease that was caused by exposure in the mine operations.

In conclusion, Mr. Chairman, I wish to emphasize that we are here to take a constructive role in the process of reaching sound public policy decisions in the public compensation area.

Thank you.

[The statement of Dr. Karrh follows:]

STATEMENT OF BRUCE W. KARRH, M.D., GENERAL DIRECTOR OF MEDICAL, SAFETY AND FIRE PROTECTION, E.I. DU PONT DE NEMOURS & CO., ON BEHALF OF THE CHEMICAL MANUFACTURERS ASSOCIATION

Mr. Chairman, members of the subcommittee, my name is Bruce W. Karrh. I am the General Director of Medical, Safety and Fire Protection for the Du Pont Company. I am appearing today on behalf of the Chemical Manufacturers Association (CMA), a non-profit trade association whose company members represent more than 90 percent of the productive capacity of basic industrial chemicals in this country.

We appreciate the opportunity to present our views on the compensation of persons injured by exposure to hazardous substances. This is an important subject and we are taking a constructive role in reaching sound public policy on this issue.

CMA strongly believes that individuals harmed by exposure to hazardous substances should be compensated by those who have caused their injuries. We are concerned about anyone injured from such exposures and we support the right of an injured individual to receive fair and speedy compensation. However, as I will discuss, we have not seen evidence of a significant health problem from hazardous wastes. We fully agree that concerns in this area require a thorough scientific review, but we also believe that in view of the review processes now moving forward, it would be precipitous for Congress now to enact pending proposals to modify existing compensation systems.

Compensation for non-workplace injuries traditionally has been handled through the common law tort system of the various states. It has recently been asserted however, that new approaches are needed because the existing tort law system is inadequate to deal with waste site injuries.

These assertions are based on the assumption of a major public health problem from waste sites. Proponents of this view have assumed that large numbers of people are being harmed by waste sites and that the existing legal system will not be able to handle massive numbers of claims to provide redress to those injured. However, this fundamental assumption of widespread harm from waste sites has not been closely examined.

Last year CMA created a special work group to determine if there was a relationship between waste sites and widespread harm. I had the opportunity to serve as chairman of this work group. We found that claims of such a relationship run counter to the experience of CMA member companies. Moreover, independent epidemiological surveys have failed to detect significant health problems.

In my testimony today I will share information that led CMA to this conclusion. I also will describe what CMA is doing to increase our knowledge about potential health problems resulting from waste sites. In particular I will report on two recent developments which should provide needed data and analysis upon which more informed judgements can be made in the future.

In our consideratin of the issue, we found only two valid epidemiology surveys of populations relative to waste disposal sites. The first was a study conducted by the New York Department of Health to determine whether the population in Niagara Falls, New York, which could have been exposed to wastes from the Love Canal site, had suffered any increased incidence of cancer. The data employed in this epidemiology study was obtained from the New York Cancer Registry for the period 1955 through 1977. The New York Department of Health concluded that the data showed no evidence of higher cancer rates associated with residence near the Love Canal site than in the entire state outside of New York City. Cancers reviewed included respiratory cancer, liver cancer, lymphoma and leukemia. The only increased incidence noted was for respiratory cancer, but the incidence in the population surrounding Love Canal was not significantly different from the rest of Niagara Falls. Thus, it was concluded there was no evidence that the lung cancer rate was associated with toxic wastes. Moreover, despite earlier assertions, a recently released study by CDC has not found an increase in chromosomal damage in Love Canal residents when compared to residents elsewhere in Niagara Falls. The study reported no increase in abnormalities among residents or former residents of the area surrounding the canal when compared to residents away from the site.

The second study we looked at investigated the health effects, on the general population, of chemical exposure in highly industrialized Contra Costa County, California. This 4-year study, which was conducted by the California Health Department Cancer Epidemiology Unit, was financed by both state and federal funds. It found that smoking, and not air pollution or chemical exposure at work or from dump sites, was the most likely explanation of higher rates of lung cancer in Contra Costa County.

Thus, the only epidemiology studies known to us which scientifically analyzed the effects of chemicals from waste disposal sites in the surrounding community suggest no causal link between proximity to the sites studied and cancer incidence.

We have also reviewed a study of residents living in Hardeman County, Tennessee, who used water from a contaminated aquifer. The study found that blood tests for some of the residents displayed a temporary abnormal liver function which, in most cases, disappeared with the removal of the source of exposure.

We also reviewed a number of studies conducted by the government and by industry to determine whether occupational exposure to toxic chemicals has resulted in an increased incidence of chronic disease. Based upon our own experience and the available exposure data, we believe that, in general, such employees would have been exposed to higher concentrations of chemicals than the population around waste disposal sites. Hence, occupational exposure and health data should provide an indication of the health effects that might be expected if similar exposure were encountered by the public. Since we saw no significant increased incidence of chronic disease among employees in the chemical industry, we concluded that widespread increased incidence of chronic disease among the public is also unlikely. Our studies have also found that chemical workers have a lower death rate from cancer and from all causes combined than the general public.

For these reasons, CMA's view is that evidence of a national problem deriving from public exposure to hazardous waste is not at hand. CMA also believes, however, that further scientifically sound information is needed. We also know the public needs reassurance that measures are underway to resolve the questions that have been raised. However, since we have seen no evidence of significant health effects, we have time to do the studies required.

CMA EFFORTS

CMA and its member companies are committed to cooperate in efforts to scientifically determine whether and to what extent there may be a relationship between waste sites and illnesses. We have recently undertaken two major efforts to accomplish this. The first is the successful settlement of a lawsuit brought by the Environmental Defense Fund (EDF) and joined by CMA to get the government moving on health studies. The second is the completion of a feasibility study on how the best available data and scientific expertise could determine the nature and extent of any such effects.

RESOLUTION OF LITIGATION

In section 104(i) of CERCLA superfund, Congress called for a scientific program to analyze waste site/disease relationships.Congress also required establishment of a new "Agency for Toxic Substances Disease Registry" to administer this program.

Two years after CERCLA's enactment, the government had neither established the new Agency, nor released significant amounts of Superfund monies to support the study program by other agencies. It was in light of these failures that EDF and CMA filed their lawsuits.

This important case was settled on May 27. Even before the settlement, the government had moved to resolve one significant point. On April 25, 1983, the government formally established the new Agency.

The parties then moved quickly to settle the remaining issues. Some of the most important provisions of the Consent Decree are as follows:

Registries.—By May, 1984, the Agency must conduct a peer review on the methods to be used in establishing registries of persons who have been exposed to toxic substances. The Agency is required to establish and maintain registries whenever inclusion of such persons "would be scientifically appropriate or valuable for long-term follow-up or specific scientific studies." The Agency must also recommend methods for establishing registries of serious diseases by November, 1983.

Inventory of literature.—By November, 1983, the Agency must establish a "publicly accesible" inventory of literature, research, and studies on toxic substance health effects.

Survey and screening programs.—Based on the registries and other information gathered from the Consent Decree implementation, the new Agency must conduct periodic survey and screening programs "to determine relationships between exposure to toxic substances and illnesses."

Mr. Chairman, I am thus happy to report that as a result of the joint efforts of environmental and industry groups, the important scientific study program called for by Congress in 1980 will now get underway.

COMPLETION OF UAREP FEASIBILITY STUDY

I am equally pleased to report that a feasibility study that we contracted for in March of 1983 with the Universities Associated for Research and Education in Pathology (UAREP) has been completed. This feasibility study contains important conclusions that bear upon the subject of assessing possible waste sites/disease relationships, and will be of great potential assistance to the new health Agency in carrying out its functions. This study was released on July 7, and we have submitted a copy for the record of this hearing.

UAREP is a consortium of 15 major universities. It was created in 1964 at the request of the National Academy of Sciences and the Department of Defense to do research in the field of human disease.

UAREP has concluded that within eight to ten months, it will be "entirely feasible" to carry out a comprehensive review to evaluate the substantial body of available data for the purpose of permitting an interim conclusion as to the nature of any health effects which may be associated with waste sites.

Such a study would have, among others, the following objectives:

To identify those chemicals associated with waste sites which could prove hazardous to humans as the result of excessive exposure;

To identify, if possible, the levels of human exposures likely to occur in association with waste sites;

To define and classify the probable health effects, if any, likely to occur in association with waste sites;

To identify the clinical and laboratory end results that would be useful in investigating problem sites; and

To identify the epidemiological criteria necessary to establish causal relationships.

The feasibility study also contains a detailed organizational and procedural outline for carrying out the comprehensive review study. This will require an Executive Scientific Panel, two Task Forces and a full-time scientific and support staff secretariat. The Executive Scientific Panel will consist of twelve to fifteen eminent academic scientists of international reputation from a variety of relevant disciplines. The task forces will also be staffed by experts qualified to deal with the needed health and environmental reviews.

CMA is committed to the feasibility study's recommendation to carry out this important and comprehensive review. We believe this study deserves the broadest possible sponsorship and financial support. We are now actively engaged in seeking this support from environmental, academic, government and business groups. CMA is committed to beginning this study by September of this year. While the costs of such a study will not be insignificant, we are convinced that the costs will be well worth the benefits to public knowledge and understanding.

Although we have not seen evidence of a significant health problem, and while the limited evidence we have seen makes us believe there is not likely to be a significant problem, we fully agree that the public concern in this area requires a thorough scientific review. In light of the substantial scientific review processes that are now moving forward, CMA feels it would be precipitous for Congress to enact any of the pending proposals to modify existing compensation systems related to waste sites. These proposals would impose new taxes and establish new bureaucracies, and they tend to ignore established principles of causation. Yet, they are based merely on assumptions of need for such measures, and are being proposed at a time when programs are actively getting underway to test such assumptions.

CONCLUSION

In closing, Mr. Chairman, I wish to emphasize that we are eager to take a constructive role in the process of reaching sound pubic policy decisions in the compensation area.

Mr. LEVITAS. Now Dr. Riordan, we would like to hear from you.

Mr. RIORDAN. Good morning, Mr. Chairman. I am happy to be here before you with my colleagues from EPA. In the interest of time, I will restrict my comments to a very brief summary of my written statement. I would also request that you accept for the record the copy of the earlier testimony given before Congressman Florio's subcommittee by EPA's James Barnes, having to do with the issue of toxic victim compensation.

Mr. LEVITAS. Without objection, it will be made part of the record.

[The statement referred to follows:]

STATEMENT OF A. JAMES BARNES, ACTING GENERAL COUNSEL, U.S. ENVIRONMENTAL PROTECTION AGENCY

Good morning Mr. Chairman and members of the Subcommittee. I am Jim Barnes, Acting General Counsel of the U.S. Environmental Protection Agency. I welcome the opportunity to appear before you today to discuss toxic victim compensation and the proposals aimed at addressing it.

The issue of victim compensation goes well beyond EPA in terms of impact and meaning, as it also relates to a multitude of issues, including health-related costs and income maintenance. In recognition of this, and to coordinate Administration efforts and to develop an Administration position, the President has designated a high level Cabinet Council Working Group, under the auspices of the Cabinet Council on Legal Policy. One of the major functions of this Working Group is to review the legislative proposals in the victim compensation area as they relate to existing tort law and existing compensation schemes, such as Black Lung and Social Security. As part of this effort, EPA is working closely with other Federal agencies to consider issues with government-wide implications. This effort will require some time; however, it is driven by the realization on all our parts of the urgent nature of the possibly unfilled needs of those affected by toxic exposure.

Those who suffer debilitating disease from hazardous substance exposure obviously face serious physical, financial and emotional difficulties. We at EPA, and especially those who work to clean up high priority Superfund sites, are familiar with the ways hazardous substances can contaminate our environment by affecting the water we drink, the air we breathe, and the homes we live in. Of additional special concern are the anxieties and fears concerning possible health effects caused by exposure to hazardous substances. At EPA, we believe it is extremely important to provide accurate environmental and health risk information to the residents of areas where exposure to hazardous substances may have occurred. Indeed, openness about what we know, what we suspect, and what we do not know regarding the risks of injury and disease in these situations is perhaps the most important single factor in assisting individuals to cope effectively with the exposure. Ironically, it is this very area of the relationship between exposure and injury where we have the most uncertainty, and it is this uncertainty that is the major cause of frustration when the victims attempt to obtain compensation for their injuries.

EARLY LEGISLATIVE CONSIDERATION OF VICTIM COMPENSATION

In 1980, the Congress considered including provisions for compensation for personal injuries in the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, also known as the "Superfund." It was decided, however, that the initial legislation would involve a Fund to pay the costs of cleaning up releases of hazardous substances and to compensate claims arising from damage to natural resources. Nevertheless, the sponsors of Superfund agreed that there was a need for more information regarding injuries to persons from exposure to hazardous waste. This, in order to provide some of the data and studies that would be needed for future consideration of personal injury-related issues, the Superfund legislation required that, under Section 301(e), a study be conducted of the adequacy of the legal remedies relating to injuries and damages from hazardous wastes. The results of that study have been available for approximately one year and form the basis for a number of legislative proposals currently being considered.

While the analysis of the legal issues has been extensive, I feel that there are other issues which are equally, if not more complex, and require equally thoughtful attention and analysis. All of the victim compensation proposals which are being considered rely heavily on an amdinistrative compensation system to relieve the inadequacies and cumbersomeness of the judicial system. However, we must carefully consider the need for and the feasibility of the administrative schemes and whether the scientific knowledge necessary to implement them is available.

GENERAL SUMMARY OF MAJOR VICTIM COMPENSATION PROPOSALS

The purpose of all these proposals in to improve the chances for recovery for injuries resulting from exposure to hazardous substances. All the bills provide compensation for personal injury resulting from exposure to a range of hazardous substances. Some bills also allow compensation for economic loss or natural resource damage.

All of the bills set up an administrative compensation program to handle victims' claims with payments from a fund. Some rely on extension and/or expansion of the Hazardous Substance Response Trust Fund established under CERCLA while others would create a Toxic Victim Compensation Fund through loans from the Hazardous Substance Response Trust Fund and transfers from the U.S. Treasury, when required. Money for the Fund may be obtained through recoveries from liable parties. H.R. 2582 sets up a separate Victim Compensation Fund with essentially the same tax base and the same tax rate as the Hazardous Substance Response Trust Fund. Most of the bills allow either judicial action or administrative claims, or both, as a forum for relief. Most of the bills would drastically reduce the burden of proof on claimants to establish their injury and its cause. In general, they would require three major findings to establish presumption of injury: (1) that there was a release of a hazardous substance(s) and that the claimant was exposed; (2) that the claimant was exposed for a duration and at a level that is reasonably likely to cause or contribute to claimant's injury; and (3) that it is reasonably likely that such exposure caused or significantly contributed to the type of injury alleged.

There are two types of scientific analyses that responsible agencies may have to prepare. One type is incident specific investigations to evaluate individual claims. The second type of scientifc analysis requires assessments of the health impacts of specific hazardous substances, levels of exposure that would result in specific injuries, and associated levels of risk. As such assessments are completed they would serve as a basis for determinations in claims.

ISSUES RAISED BY THE PROPOSALS

We are carefully reviewing the victim compensation proposals introduced in this Congress. While EPA is not ready to take a position today, I would like to comment on some general issues that we believe we all need to address with respect to various victim compensation proposals.

At the outset, I should note that compensation programs such as the ones proposed by these bills involve many issues other than environmental considerations. We need to determine how such schemes will affect existing systems of compensation, particularly tort litigation. It has been suggested, for example, that the presumptions and evidentiary standards that may be part of a compensation system will eventually be incorporated in our tort litigation system; we obviously must consider the full implications of such a development. We also will have to consider the long-term fiscal implications of compensation programs. Experience with many government administered compensation programs has been that they have grown far beyond initial expectations; we must consider what we realistically can anticipate in terms of growth, both as to claimants and overall cost. We must carefully consider the administrative mechanisms for awarding compensation, and whether these mechanisms actually accomplish the ends sought, and at what costs. In evaluating the costs of compensation systems, we must consider the role of the legal profession and the portion of the compensation that would go toward payment of legal fees. We must also look at the economic consequences of compensation programs, and at who will bear the ultimate costs. And, we must consider how any compensation program relates to other compensation programs already provided by the Federal and State governments, including Social Security disability insurance, medicare/medicaid, and other forms of income maintenance.

These are all issues which go well beyond this Agency's mandate of environmental protection, and necessarily involve other Federal agencies and other Federal programs. In this regard, I would note that the Federal government has considerable experience with compensation programs, including the Black Lung miner's compensation program, Social Security disability insurance, and the Federal employee's and the longshoremans' compensation systems. There are valuable lessons that have been learned with regard to compensating individuals for injuries and illnesses, and we must look carefully at our experiences with these programs in determining whether a particular compensation program is warranted, and, where it is, how it should be structured.

The considerations I have identified above are but a few of the larger policy issues that will have to be dealt with in reviewing compensation proposals. These issues, and the many others we will have to consider, cut across the responsibilities and expertise of many Federal agencies and many Federal programs. As noted in the recent editorial in the Washington Post of this past Saturday, the issue of creating a compensation system involves much more than just determining the best environmental policy, and must be considered in this broader context. For this reason, the Administration has created the high-level interagency Working Group, referred to earlier in my testimony, that will review the broader implications of compensation systems, and the basic questions of social equity at issue.

I also wish to touch on four additional general issues that are particularly important to EPA: first, the scope and definition of the problem; second, the scientific and administrative issues which we must come to terms with in any victim compensation scheme; third, the potential impacts on EPA programs; and finally, the implications of the various funding mechanisms.

A basic question which many people raise with regard to toxic victim compensation is: what is the extent of the victim compensation problem? How many people are injured, what injuries do they suffer, and what and how many substances cause those injuries? Currently, the Agency is assessing the data we have to contribute to answering these questions.

In further viewing this issue, the complexity of defining the problem and its scope must be considered.

Toxic victim compensation has been most broadly defined to cover any injury or disease or death caused by any exposure anytime to any substance any place. On the other hand, it has also been limited to compensation for the injury, disease or death of any person due to exposure to hazardous waste not occurring in a workplace.

A number of questions arise in exploring the definition of toxic victim compensation; to follow are but a few of the most critical. For example, do we agree that only non-workplace exposures are intended to be covered, or would there be instances where occupational exposures should be included? Does "exposure" cover exposure only to hazardous wastes or does it extend to any release of a hazardous substances? Does "hazardous substance" cover only those substances traditionally found in waste sites, or does it extend to the broad range of substances (including those regulated under other laws) in the air, water, drinking water, and consumer use? What more need we know about possible mulitple causes of some conditions that may be linked to exposure to toxic substances? What harms (i.e., death, injury, illness) are to be covered or compensated as a "toxic tort"? Should the proposals only address chronic, but not acute, health effects? What is the relationship of this type of action to product liability law?

The scientific assumptions and requirements which underlie all the victim compensation proposals are among the most important issues in the whole victim compensation area. All victim compensation proposals assume some ability through science to establish a cause and effect relationship between particular substances, exposure levels, and diseases. This may not be the case. We must look closely at the questions we are asking scientists in these proposals, to assess whether or not the ability to implement the legislative scheme exists.

In addition, although EPA has extensive experience regulating toxic chemicals under RCRA, FIFRA, TSCA, the Clean Air Act and Clean Water Act, the case specific scientific analyses and conclusions required in the proposed legislation differ significantly from our experiences with existing laws. The scientific data and analyses that would be required to answer the questions above may not currently exist. We intend to assess the ability of the Agency to gather such data and answer these questions. We must assess the scientific feasibility of implementing these potentially broad statutory requirements. Some of the proposals, including H.R. 2582, require the Federal agency, either EPA or HHS, to produce toxic substance documents. We are attempting to assess the magnitude of these requirements, but based on EPA's experience, these documents could be very costly and each one could take up to several years to complete.

A third area of concern for EPA is the possible impact of victim compensation proposals on existing programs and what the possible effect might be on our ability to carry out current responsibilities. Assigning a regulatory agency such as EPA responsibility to compensate people for injuries due to environmental exposure to hazardous substances might well create serious conflicts with EPA's existing responsiblities to regulate environmental contamination under the many statutes the Agency administers and to respond to hazardous substance releases under Superfund. Furthermore, EPA is not involved in administering any programs of this nature and therefore, all its administrative implications would require further analysis. Other agencies with expertise in this area might be able to provide valuable information to the Congress.

Implementation of Superfund is exemplary of this issue. Victim compensation claims and Superfund response actions could exert significant influence on each other. In making decisions related to taking response actions, the Agency gathers extensive data about a site and determines the most cost-effective response that mitigates any public health or environmental hazard. The data required to make these determinations may be very different from data the Agency would collect to assess victim compensation claims. Similarly, the Agency might decide that the appropriate response would be site closure and permanent relocation of residents without extensive cleanup or study of the area. Detailed health assessments would require much more extensive work, and expenditures from the Fund, than would be taken in solely a response action. This competition of interests and purposes between victim compensation and cleanup needs must be assessed before decisions are made with respect to appropriate legislation.

Finally, the proposals contain some variation in funding mechanisms. It is critical that we determine whether the funding mechanism is viable and funding levels are adequate to pay claims. An additional funding issue relates to the CERCLA Post-closure Liability Fund. CERCLA established the Post-closure Liability Fund to assume virtually all liability, including for personal injury, and for certain eligible closed land disposal facilities which operated under final RCRA permits. H.R. 2582 sets up a Post-closure Victims Fund which duplicates the Post-closure Liability Fund both in purpose and in tax base. Both Funds would apparently exist side by side with some inconsistencies and differences which would be unresolved by the proposed bill.

EPA EFFORTS IN THIS AREA

Faced by the urgency of some of the issues in this area, EPA has undertaken to further analyze and better understand this critical area. Accordingly, we have commenced a review of legislative proposals focusing on administrative and scientific issues as well as assessing the impacts upon the Agency's substantive programs and its resources.

An important issue that we are investigating is EPA's ability to conduct the kinds of health effects studies required by the various legislative proposals. Our ability to develop these studies relates directly to our capability to answer the scientific questions I highlighted earlier in my testimony.

WHAT LIES AHEAD

The Congress faces a substantial undertaking in considering the range of toxic victim compensation issues. At this point, the only conclusion that is clearly evident to me is that we all share a large degree of concern for the victims of hazardous substance exposure, and we at EPA stand ready to assist you and them in any way possible.

Adequate consideration of the full range of toxic victim compensation issues will require coordination, cooperation, and good will within EPA, the Executive Branch, and between the Congress and the Administration as well as with the States, which have a great interest in what is finally decided here in Washington.

It is certainly worth the effort of us all to assure that all parties with an interest in this issue are heard during the Congressional deliberations which will follow today's hearing.

We at EPA pledge to do what we can to assist in this important effort and will keep this Subcommittee and Congress apprised of our activities as well as progress toward the goal of developing more scientific certainty in the area of health effects resulting from exposure to hazardous substances.

This concludes my prepared statement. I would be happy to respond to your questions.

Dr. RIORDAN. I have with me Dr. Betty Anderson, head of the Office of Health and Environmental Assessment. That office has the responsibility for providing guidance on carrying out risk assessment as well as actually carrying out risk assessments in the agency. To her left is Dr. Casey Jason, a medical officer and expert in pediatric risk assessment, and Dr. Lewtas, who is head of the Genetic Bioassay Research Branch of EPA's Health Effects Research Laboratory at RTP and is prepared to answer questions on our research program. We are hoping to elucidate on the problem of risk assessment in the future.

I would like to start off by saying we do carry out risk assessments in EPA for the purpose of setting regulations and standards. We have been involved in this activity for several years. I think that the methodologies that we have adopted have met the test of legality. They have met the test of science and they have met the test of policy. However, I would like to elaborate a bit on the risk assessment activities, so that I don't provide you with a misapprehension that risk assessment may be terribly useful in terms of implementing a toxic victim's compensation program.

The utility of risk assessment has been demonstrated but the efforts have only been able to give us crude indicators of potential adverse effects and the extent within the population. They do not pinpoint in any specific way whether ill effects will occur and, if so, in which exposed persons they will occur, nor do they allow for an increase expected by chance alone in the population based upon a sample study.

Present risk assessment activities in EPA have centered on analysis of single chemicals. The difficulties there are less than they are from chemical mixtures such as diesel emissions.

When we come to hazardous waste sites, we have multiple problems in performing and interpreting risk assessments. There are potentially thousands of waste sites nationwide, each containing hundreds of possible different chemicals.

I would like to briefly refer to Love Canal to demonstrate the problems that we face. It is illustrative because of the great number of substances that might be involved in a particular site. With Love Canal not only do we still have some incompletely described compounds in terms of their health effects but even those for which we have health information, the data are limited. Well over 400 substances were found to be present in Love Canal.

I would like to say that in those substances that were found at Love Canal, just to give you an idea of the problems that are involved, of the 400-plus compounds found in Love Canal, following analysis, 10.8 percent were tentatively classified as mutagens, 12.8 percent as carcinogens, 7.2 percent as fetotoxic, 5.8 as neurotoxic, 9.7 percent as hepatoxic, 9.5 percent as renal toxicants, and 8.6 as pulmonary toxicants, from animal and *in vitro* tests.

We have heard earlier testimony by the second witness that it has been very difficult to find any effects in the Love Canal population as a result of epidemiological studies. There is one study that is yet to be published in the peer reviewed literature which does indicate an impact on birthweight of children born in what we call the wet areas in Love Canal.

But the problem with that study is although we get a statistically significant relationship with low birthweight from the babies born in the wet areas of Love Canal, it is difficult to interpret what that statistically significant increase in low birthweight means in terms of potential health effects. So even when you come out with a study which identifies an indicator effect, it is hard then to relate that to some hard effects that might be at discussion in a victims compensation proceeding.

It is very difficult, given the multiple chemicals and given the multiple sites that one deals, to develop a risk assessment that would be useful in the victims compensation area.

I would simply like to close, then, by saying that we have risk assessment procedures that we use in setting standards and regulations in EPA. They essentially provide us with rough indicators of potential effects on a statistical basis in an exposed population.

If we examine the underlying science behind those procedures, they would not probably hold up very well in a cause-and-effect relationship determination that is necessary, in what we would expect to find in these proceedings for toxic victims compensation. I won't get into some of the details.

I think the initial witness elaborated quite a bit on the scientific questions that have to be addressed in that kind of a proceeding. We are here I think to answer questions that might help you in understanding some of our experiences.

Thank you, Mr. Chairman.

[The statement of Mr. Riordan follows:]

STATEMENT OF COURTNEY RIORDAN, ACTING ASSISTANT ADMINISTRATOR FOR RESEARCH AND DEVELOPMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY

Good morning, Mr. Chairman and members of the Subcommittee. I am Courtney Riordan, Acting Assistant Administrator for Research and Development of the U.S. Environmental Protection Agency. I welcome the opportunity to appear before you today to discuss scientific and health research issues which relate to the problem of toxic victim compensation as well as EPA's current research work.

On June 29, 1983, Jim Barnes, Acting General Counsel of EPA, appeared before Chairman Florio's Energy and Commerce Subcommittee on Commerce, Transportation and Tourism to discuss the general issue of toxic victim compensation. In his statement, Mr. Barnes indicated EPA's and the Administration's concern for those exposed to or living in areas subject to the potential risks due to hazardous substances and outlined our efforts to address this critical issue. Additionally, he discussed a number of concerns we have regarding various proposals in this area—including concerns relating to science and health issues. I respectfully submit to you a copy of Mr. Barnes' statement and request that it appear as part of the record of today's hearing.

I share Mr. Barnes' concerns over the plight of those exposed to hazardous substances as well as those who live in areas faced with potential hazard. I feel that it is important that we in government—at all levels—do what we can to inform and, if necessary, assist those faced with the likely or potential effects of toxic exposure due to improper use, release, or disposal of hazardous substances.

A critical part of any attempt to consider this multi-faceted problem is to understand the current state of scientific knowledge and efforts to obtain adequate scientific data and appropriate methods for useful analysis. I should note at the outset that EPA is but one government agency involved in this area. For example, EPA's "Superfund" statute assigns to the newly created Agency for Toxic Substances and Disease Registry responsibility for health monitoring and health assessments. EPA is also working closely with States, and in particular State Health Departments, on these matters.

In my statement, I will first discuss EPA's risk assessment activities under our existing statutory mandates. Second, I will address a number of the key scientific issues which must be considered when examining the various proposed compensation schemes, including: exposure, likelihood of injury, and causation. Finally, I will outline the research efforts currently underway involving EPA which may offer some assistance in this area.

RISK ASSESSMENT IN EPA

Within the federal government, EPA's risk assessment responsibilities are unusual in that they emphasize assessments of health risks for regulatory decision making. The manner in which risk assessments are used depends upon the statutory provision being implemented. By contrast, the National Institutes of Health, for example, and within it, the National Institute of Environmental Health Sciences, places a much greater emphasis on basic research. These entities do not have regulatory responsibilities. Although the EPA conducts research and risk assessment in many health areas, our cancer risk assessment approach provides insight into the complexities and limitations of performing risk assessment generally.

In assessing the possible risk of carcinogens posed by individual chemical substances, EPA follows a two step approach. Risk assessment attempts to answer two questions: 1) how likely is the event to occur? 2) on the assumption that the event does occur, what is the magnitude of the public health impact? Since only rarely do we know for sure that an agent is indeed a human carcinogen, the first step involves an evaluation of all the biomedical data to determine the weight-of-evidence that an agent might be a human carcinogen. The second step involves the quantification of risk, that is public health impacts, in terms of rough estimates for current exposures as well as estimated exposures for various regulatory options.

To answer the first question regarding likely carcinogenicity, EPA weighs the biomedical evidence as ranging from the strongest evidence based on human data, backed up by animal bioassay tests; to suggestive or supportive evidence provided by positive results from short-term in vitro test. This weight-of-evidence is described in terms of the nature of the responses, the quality of the data, and an evaluation of both positive and negative results.

The second step, that of presenting some quantitative estimates of public health impacts, is presented in terms of rough estimates describing the individual increased cancer risk for exposed sub-population groups, (i.e., increased risk of, for example, one chance in a thousand or 1×10^{-3}) and the nationwide impact in terms of annual increased cancer cases. This second step is intended to give the regulators a feel for the potency of the suspect carcinogen and some rough idea of the magnitude of the public health problem. It seems useful to provide these rough estimates as a measure of potency, because the potency of carcinogens can span a range of fifty million fold. There is an obvious desire to take this disparity into account in making public policy decisions even though scientific methods for precisely describing quantitative risk associated with environmental exposure was and still is a very underdeveloped field. For most suspected carcinogens, there are no methods currently available to accurately describe risk outside the dose range where effects have been bracketed between a plausible upper bound and a lower bound approaching zero. The upper bound is calculated using conservative exposure estimates and the linear, non-threshold dose response model at low doses.

In many ways the approach has worked reasonably well. The qualitative (i.e., biomedical) evidence coupled with quantitative, rough estimates are useful in setting priorities; and balancing risks against benefits, and in considering residual risk after the application of best available technology.

Risk assessment approaches have evolved over the years and will continue to do so. This is a rapidly developing field but there are large gaps in scientific knowledge which must be filled in order to make real improvements in risk assessment methodology.

In short, the utility of risk assessment has been demonstrated in the EPA, but there are many scientific uncertainties associated with risk assessments. Our experience is helping to shape our current research efforts. These efforts are only able to give us crude indicators of potential adverse effects and the extent of harm within a population. They do not pinpoint in any specific manner whether ill effects will occur and, if so, in which exposed persons, nor do they allow for an increase expected by chance alone in a population based sample.

Present risk assessment activities at the EPA, in response to our statutory mandates, have centered upon analysis of single chemicals. The difficulties there are less than they are for chemical mixtures, such as diesel emissions. When we come to hazardous waste sites, we will have multiple problems in performing and in interpreting our risk assessments. There are potentially thousands of waste sites nationwide, each containing literally hundreds of different chemicals. We must determine which chemicals are present, in what concentration, and we must estimate the proportion of chemicals that humans are exposed to and what proportion may enter the body. Existing hazard data on each of the chemicals must be assembled and analyzed to determine if there may be a problem. Exposure and hazard information need to be combined to estimate in some quantitative manner our degree of concern about potential risks. To deal with 400 or more chemicals per site becomes a difficult task, and we really have no means presently of intelligently evaluating possible interactions, for example, synergism among chemicals. Each toxic waste site is a unique microcosm of potentially hundreds of chemicals and spontaneous reaction products. To help illustrate some of the pitfalls in performing assessments at waste sites, let me give some examples.

Love Canal, New York is especially illustrative of the great number of varied substances which might be involved in one site. For Love Canal, not only do we still have some incompletely described compounds but even those for which we have described health information, the data are limited. Well over 400 organic substances were found to be present.

EXPOSURE INFORMATION AVAILABLE TO THE AGENCY

Environmental exposure to hazardous substances outside of the workplace is generally quite low but may be persistent through time. Depending upon where and when environmental sampling is conducted, exposure estimates of varying accuracy may occur. For example, measurements of PCB's dioxins, or metals in the immediate area around dumpsites may be representative of levels which can be used to determine exposure. However, the same may not always be said for volatile organics. Here the transport through the dumpsite and neighboring areas as well as through man may be quite rapid. Thus, in the case of volatile organics, even if a health effect were the result, documenting and quantifying the exposure which may have contributed to this outcome may be difficult if not impossible.

Body burden will be dependent upon more than isolated environmental measurements. It must also take into account specific routes of exposure and habits of certain population subgroups. For example, a child who eats soil might have much higher levels of certain toxic substances than an adult under seemingly the same exposure conditions.

LIKELIHOOD OF INJURY

It is difficult to determine whether exposure to a hazardous substance has caused a particular adverse effect. Of the 400 plus compounds found in Love Canal following analysis, 10.8 percent were tentatively classified as mutagens, 12.8 percent as carcinogens, 7.2 percent as fetotoxic, 15.8 percent as neurotoxic, 9.7 percent as hepatoxic, 9.5 percent as renal toxicants, and 8.6 percent as pulmonary toxicants from animal and in vitro tests. Little or nothing was known about the potential toxic effects of the remaining substances—over half of those found.

Levels of exposure will generally be quite low when compared to animal or even industrial exposure settings. It appears that most health effects resulting from hazardous substance exposure are likely to be chronic effects involving commonly occurring diseases, rather than rare diseases. Thus, it will be necessary to separate increases in various relatively common outcomes from the expected rate. Low birth weight and chronic lung disease, for example, rather than mesotheliomas which can more confidently be associated with a certain exposure to a specific chemical agent.

In order to get an idea of what this means on a site specific basis, we should consider the relevant experiences at Love Canal and Woburn, Massachusetts.

The only study on Love Canal which so far seems to have demonstrated a statistically significant difference between exposed and comparison groups related to birth weight. Low birth weight is strongly associated with major malformations, high neonatal mortality, and development problems. In the surrounding "swale" area a highly signigicant increase in the rate of low birth weight was seen even after correcting for many maternal risk factors, including maternal alcohol consumption, maternal cigarette consumption, and other maternal characteristics. This result is also consistent with the literature on animal embryo toxicity with lower dose chemical and pesticide exposure. However, low birth weight is expected to occur in roughly 6 percent of the white and 12 percent of the black births. In addition, a base line expected low birth weight rate that takes maternal risk factors into consideration, is difficult to establish.

In general, the Love Canal experience is valuable in considering victim compensation issues as it emphasized several important points, including the following.

1. Looking for rate health outcomes probably will not be helpful except in specific circumstances, such as with sufficiently large population groups to be able to detect the event.
2. Looking for an increase in chronic health effects which requires an understanding of the expected rate for a similar unexposed population is much more likely to be fruitful.
3. Exposure types and amounts especially for volatile substances will change through time and will be difficult to document.

Finally, I would also like to offer some comments on Woburn, Massachusetts, a place where a cluster of adverse health outcomes has been seen and evaluated in terms of associated environmental pollutants. This area has also been cited in support of legislation for toxic victim compensation.

In the summer of 1979, the citizens of Woburn, which is located about 12 miles northwest of Boston, became quite concerned over the health impact of 100 years of dumping various arsenic and other compounds in the Northeast sector. The initial concern arose when an apparent increase in childhood leukemias was discovered. In eastern Woburn over the period 1969–1978 a 12-fold increase over the expected rate was seen in childhood male leukemias.

Historically the area of exposure was a leading producer of arsenic compounds from 1900–15 as well as other chemicals and glues. The main route of suggested exposure was through two particular wells supplying drinking water throughout the 1950's through the 1970's. Unfortunately, historical information on drinking water toxic substance content was lacking. The problem was evaluated by the Commonwealth of Massachusetts and the Center for Disease Control and the following conclusions were drawn. Although there was an increase in leukemias around the Woburn area, case-control questionnaire data failed to identify any lifestyle differences among the cases and controls. For the one compound known to have heavily contaminated the area, arsenic, previous human epidemiologic studies have shown increased rates of lung and skin cancer following arsenic exposure but not leukemia. In other words, investigators were unable to discern whether any of the potential chemical exposures could account for the clustering of leukemia cases.

CAUSATION

The above discussion helps to indicate that determining a cause of an observed health effect is a very difficult problem—one with which science is currently struggling. This is especially the case with chronic effects, which are the types we most expect to occur from exposure to hazardous waste sites. Our risk assessments can only give us some rough indications of how much levels of exposure may increase the chance of injury. Applying these kinds of test results to the compensation setting is problematic. In chronic exposure cases, we are usually confined scientifically to determining association of exposure and disease, not causation. Furthermore, we cannot confidently distinguish between individuals in the background population who would develop illness anyway and those whose illness is attributable to hazardous substance exposure.

Problems continue to affect our ability to predict health effects in humans. The scientific community involved in risk assessment is aware of these problems and is continually seeking to improve the state-of-the-art of risk assessment. To summarize, we have a long list of waste sites. Each contains a myriad of hazardous substances that must be identified and quantitated. Not only is our information on health effects limited for many of these substances, but our testing procedures cannot account for multiple risks, synergistic, or antagonistic reactions among chemicals. Second, although a substance may clearly injure both animals and humans, the manifestations of illness may be quite different in humans than in animals. This problem makes human health effects difficult to predict. Third, many chronic health effects occur after years of low-level exposure. Modeling this would be very time-consuming and expensive, so instead we test in a shorter period using large doses. This procedure yields sound information for regulatory purposes, but whether the conclusions are appropriate to decisions that would have to be made in a compensation system is not clear and is, fundamentally, a policy decision.

RELATED RESEARCH EFFORTS

Some of the efforts now underway at EPA which may be of future help to prioritize and evaluate hazardous substance risk include the following:

1. Establishing the expected rates for sensitive reproductive health outcomes in children at the county level.

A. County level: As mentioned before, birth weight is a sensitive indicator of toxic substance effect on animals and as suggested in Love Canal may be a good outcome to assess for humans. We are presently developing national county level birth weight mapping techniques using vital statistics records which adjust for maternal risk factors using specific statistical techniques.

B. Local level: EPA in collaboration with New York State and now with Missouri will be developing a local census tract level assessment of birth weight using similar techniques to those developed at the national levels. In addition, adjusted childhood cancer rates are also being added at the local level. It is hoped that besides a comparison to national mapping techniques, more and better risk factors can be considered to refine the assessment technique and aid in priority setting for dumpsite clean up. Potentially the techniques being developed in these two states could go down to the level of Love Canal areas. This is also being developed to complement ongoing state efforts in this area.

2. Animal/Human Correlation Studies: The currently available animal toxicity information on reproductive problems associated with toxics substance exposure is being compared to all available human data on the same substances to begin to address the problem of generalizing animal study information to human risk assessment.

3. Correlation of exposure induced genetic damage in somatic (body) and germinal (reproductive) cells resulting in heritable changes which might involve risk to future generations of exposed individuals.

4. Significant gaps in the risk assessment methodologies are being addressed along with validation of methodologies. Although past efforts have been essentially limited to cancer risk, we are currently working on both qualitative and quantitative procedures to improve risk extrapolation methods for animals to man for mutagens and carcinogenesis, as well as for potential adverse specific health effects such as: neurobehavioral, pulmonary, hepatic, reproductive, and teratologic. Methodologies to predict heritable genetic effects of chemicals found in the environment will also be addressed.

5. ORD is now verifying a screening test system to evaluate the potential health effects of complex waste mixtures. The proposed program uses in vivo and in vitro assays of increasing complexity to estimate the biological activity of the complex mixture.

CONCLUSION

Adequate consideration of the full range of toxic victim compensation issues will require coordination, cooperation, and good will within EPA, the Executive Branch, and between the Congress and the Administration as well as with the States. Everyone will be affected by what is finally decided here in Washington, whether they be from business or from affected or potentially affected areas.

We all feel a deep concern for the victims and potential victims of hazardous substance exposure. We at EPA pledge to do what we can to assist in this important effort and will keep this Subcommittee and Congress apprised of our activities as well as progress toward the goal of developing more scientific certainty in the area of health effects resulting from exposure to hazardous substances.

It must be understood that science can only provide statistical probability of cause. Given that limitation, it must be left to the policy maker, not the scientist, to decide whether to award compensation.

This concludes my prepared statement. I would be happy to respond to your questions.

Mr. LEVITAS. Let me say this at the outset. I know that members of the subcommittee, myself included, have a large number of questions that we would like to put to various members of the panel. What I intend to do is go through a certain number of questions that I have at the outset, and then pass it along to some of the other members, and then come back around a couple of times, so that we let everybody have an opportunity to begin to focus on some of these issues.

Let me start with Dr. Carnow. In making both legislative decisions or in governmental agencies making enforcement or action decisions, or in litigation, we need to operate on the basis of relating the existence of a phenomenon, namely, a hazardous waste site to reported health effects in a surrounding population.

In your judgment is there reliable evidence upon which either we as legislators or courts or enforcement agencies, regulatory agencies, can operate to relate the presence of a linkage between the existence of the hazardous waste site and the health effects which are reported in the surrounding community, or is it so indefinite that we are not able to make those decisions, based on the evidence available?

Dr. CARNOW. I think in some cases there may be some lack of definition. I think, though, that much can be done. I think using the kind of models that Dr. Conibear and I have developed, for example, might be useful in making an assessment of the probability of causation. One of the problems is that very standard tests are done. Anybody knowing American medicine knows we have many tests for the same thing.

We must have 20 tests to determine whether or not the liver is healthy. The reason that this is the case is because we don't have one single test that tells it all, so we do many. Our tests are also calculated to find overt disease.

Preventive medicine is something which is just coming down the road, so most of the time we look at clinical disease and our tests measure clinical disease. One of the things that we have to do is to address—and some of us have—much more the problem of developing tests which show preclinical disease or subclinical disease. Let me give you one example.

We know that lead will cause a slowing of conduction in motor nerves, and so we began to set levels for safe levels of lead based on that. What was not recognized at the time was that the kidney is the major target organ, and so to a large degree that was ignored.

The second thing that happened is that scientists who were looking for early tests began to look even more carefully, and Dr. Seppalinian found if you looked at slow rather than fast fibers, you could pick up damage at a much earlier point in time. So one of the problems that we have is that the tests that we do may not be sensitive enough. We have to develop more sensitive tests.

The second thing that we need to do is to look at the development of new or extended tests. One of the things that we believe, for example, is that an important early effect of many toxic agents is on the central nervous system, particularly the brain, and we have developed a very extensive, in some cases complex neural behavioral batteries, which we feel can find early evidence of neurological brain damage, so that new tests have to be developed.

The other problem—and you allude to it when you say to reported health effects—is that some of the health effects of toxic agents are not known. It was, for example, thought in relation to dioxin that if you did not have chloracne, which is a disfiguring kind of acne, you didn't have the disease. Well, the problem was that the disease was first looked at by dermatologists.

We now know that very few people exposed to dioxin may develop chloracne, but there may be a wide range of other problems that individuals have, and so the first thing that has to be done is to gather all that is known about the scientific literature about an agent or a group of agents. Then the second thing is, again, to make an assessment of what we don't know about those agents.

For example, we have now examined hundreds of individuals in communities who may have been exposed to dioxin. We find in more than normal numbers individuals with immune deficiencies, which is not a common phenomenon in the general public, of peripheral neurological damage, of central neurological damage, of liver damage, abnormalities of lipid metabolism.

Dr. Karrh noted in Triana, that CDC found no disease. Now, that is in a sense true, but it is not quite true, because they found abnormalities of liver activities in these people, and they did not do a comprehensive examination. It was a survey. We did an examination of some, I don't remember, 25 or 30 people in Triana, and we did find damage. CDC did not report on teen-age males who had female breasts, or teen-age females who had precocious puberty, or other abnormalities.

So, again, I think what has to be done is I think more intensive effort. Again, I don't believe that CDC did a bad thing. They did a survey, and a survey only gives you a certain amount of information. But when you have a group of individuals who may have been eating and drinking DDT for a period of 20 or 30 years, in my opinion that deserves very intensive investigation, and our examinations were quite labor-intensive.

Again, you look for what disease is present, and what organs are affected. When you say do you look to reported health effects, yes, but there may be others which have not been reported, and if you find abnormalities you cannot pass that off because they were not reported.

Mr. LEVITAS. Let me put it in a specific context. Dr. Karrh, in his testimony, says, "We have not seen evidence of a significant health problem from hazardous waste."

Now, at the same time, just taking two specific well-known examples, the Federal Government has spent millions of dollars at Love Canal and at Times Beach to respond to a situation where people in the area of the abandoned hazardous waste sites complained of all manner of health consequences, and in response, a decision was made to spend a lot of the taxpayers' money to do something.

The question that I am interested in, was this simply a political response to quiet down some hysterical housewives in the area, or was it justifiable in terms of scientific information.

Specifically, I know you are familiar with the Times Beach situation. Could you describe what medical conclusions you have drawn from your experience in the Times Beach situation, and was the government response appropriate, or should we have simply said, well, as I read Dr. Karrh, yes, there is a hazardous waste site there, and, yes, you do live near it, and you may have these illnesses. But they are not caused by that hazardous waste site, and therefore the Federal Government, the taxpayers, ought not to be spending these millions of dollars.

Dr. CARNOW. Well, I don't know what CDC has done. They have not discussed any of this with me, certainly, and I am not privy to their activities. I believe that one of the physicians in charge of this activity is a physician who was involved in legal activities in relation to others exposed to dioxin, and who testified and the others that although they had a variety of diseases, none of them could be related to dioxin.

I don't know whether that in any way influenced the conclusion that there was no problem. I can only assure you that I have some 1,000 scientific reprints which represents studies of various kinds in relation to dioxin. We have examined now more than 300 people, none of them at Times Beach. We have not examined the Times Beach folks, but we have examined people who have been exposed to dioxin. I can assure you that it is toxic.

I can assure you that it has caused a wide variety of diseases, and I have 30 years of medical and scientific activity which says that those are related to and caused by the exposure to dioxin.

To find 40 or 50 percent of the people in a community with evidence of neurological damage would be extraordinary in a normal community of particularly young, healthy working people. Again, I think that these are not as great mysteries as we make them to be.

I think the problem with Times Beach is an enormous problem. I think dislocating people, moving people out of town, doing all sorts of things like this is a terrible thing to do, and should not be done without cause. But I think that the kind of study that consideration of such a dislocation demands may not have yet been done in Times Beach.

Mr. Levitas. I throw the question to you, Dr. Karrh. Let me phrase it, if I may.

Dr. Karah. All right.

Mr. Levitas. Based on your professional expertise, and as I read the portion of your testimony I just did, do you believe it was an appropriate decision to move, evacuate Times Beach or Love Canal or any of these sites which are now well known to us, based upon the medical and scientific evidence available, or do you believe the contrary, that it was mostly a political response, not related to the healthy impact on the surroundang community resulting from that hazardous waste site?

Dr. Karrh. Mr. Chairman, I have not studied the people at Times Beach or at Love Canal. As a physician, I have not examined any of those people. I have reviewed the data that are available from those two sites, the data that have been presented both in scientific documents and in the lay press.

Based upon that, it is my opinion, and it was the opinion of my work group that I chaired that was made up of professionals also, that there was not scientific data that these people had health effects that had resulted from their exposure in the Love Canal or the Times Beach area, but that they perhaps had had some exposures that did need further investigation.

I think earlier when you characterized my testimony in your question of Dr. Carnow, I am not sure that your exact statement was what I had said because I have not said that we have a site and we have a disease and the disease is not caused by exposure. What I have said is, based upon the data that we have reviewed, we have not found any valid scientific data that indicates that hazardous waste disposal sites have caused a significant health problem.

We feel, though, that there needs to be more study, and we would hope that people like Dr. Carnow, who are working in this area, would publish their studies so that when we try to pull the data together and determine if we have a problem, and if we do have a problem what is the extent of it, we will have access to those data so we can utilize them in our decisionmaking process.

Mr. Levitas. Dr. Riordan, EPA Is not a think tank. It is a regulatory agency. At Times Beach a decision was made to spend $33 million Superfund money to evacuate that community, buy it out. Yesterday we heard some very poignant testimony about the impact on that community as a result of that decision.

Is EPA in this regard making a political decision in response to the outcries from the citizens and the congressional delegations and the Governor of those affected areas, or is the decision that is made, whether it be Love Canal or Times Beach, made upon a scientific judgment that there is something that is affecting the health of people resulting from that nearby hazardous waste site?

Dr. **Riordan**. Mr. Chairman, I can't speak directly from personal experience, but I think I can speak from being in the agency and being peripherally involved with some of the decisions that have been made at Love Canal and at Times Beach. I would characterize the decision as a prudent public health decision being made in the case of Times Beach. I say that for two reasons, and then I will defer to Betty, if you would like some explanation about some of the risk assessment work that was done as part of the decisionmaking process.

First, the Times Beach situation was somewhat unique in the sense that we had potential for a very ubiquitous exposure to dioxin since the materials had apparently been spread quite widely in the community. It wasn't a contained site where one could perhaps restrict the potential for adverse health effects, but rather it was a potential, extremely ubiquitous contamination, in some cases at fairly high levels of concentration. That led us to be fairly cautious and led us to, I think, decide quickly that we had to take action.

The other thing is that dioxin has the potential for chronic health effects, and although effects at the Times Beach situation may or may not have been expressed as of today, nevertheless the risk assessment activities indicate that in fact long-term exposure may result in some chronic health effects.

That added into the equation, we worked closely with CDC in making that decision. I don't know what levels they were talking about in terms of justification for actual evaluation, but perhaps Betty could fill us in on some of those response studies that were done as part of this.

Mr. **Levitas**. I would like that response, Dr. Anderson, and in addressing it, perhaps you could also answer what I consider to be a simple question. I am sure it is not, but do you consider that there is evidence of significant health problems from hazardous waste sites or not?

TESTIMONY OF MS. ELIZABETH L. ANDERSON

Dr. **Anderson**. To respond to your last question first, in the EPA we have always taken the position that it is our responsibility, as was just said, to pursue a policy that is proven, which means we don't expect to find disease before we act. It is well known that epidemiology methods are not particularly sensitive, and they are expensive to conduct. It takes a very long time, and in some cases is absolutely impossible to identify appropriate cohort groups to study.

So I think from an environmental protection standpoint, we don't look for—you probably heard the term—"dead bodies," before we act. We don't necessarily rely on the human evidence before we take action, but of course, any human data are highly valued and it helps us enormously in making decisions. But I think the whole purpose of the risk assessment in EPA is quite different.

That is, since we don't know for sure what might happen, our purpose is to formulate an approach to taking great uncertainties in the scientific area, and having some consistent way of organizing that information to get some idea how likely it is that disease might occur, and then some feel for if they do occur somewhere down the road from long-term, low-level exposure, what would be the magnitude of the impact.

This has been done in the agency, as Courtney said, I think quite successfully now for the last 6 to 8 years. We have considerable experience in the area of carcinogenesis. There are great scientific uncertainties, there is no question about it.

In many of the evaluations we do, we necessarily have to just take arbitrary positions, because the underlying science is not sufficiently advanced to do anything else. But we think that the fairest thing we can do is to spell out exactly what the basis for those decisions are, and that is exactly what we have been doing.

Certainly these issues are quite debatable in the scientific community. I think to leave any other impression would be incorrect, particularly for purposes of your considerations here.

A risk assessment that would address the issue of whether a person's illness is associated with a particular waste site exposure is different from the risk assessment we have been doing, where we are trying to predict what might happen. Here we want to know the probability that a disease that has occurred is associated with a prior exposure.

I think this becomes even more difficult. That is not to say that one couldn't formulate guidelines for doing it. I think that could be done. I think the guidelines would certainly get into, again, necessarily, arbitrary decisions, necessarily arbitrary rules, and there are going to be enormous data gaps.

For example, we probably would have very little information on what the real exposures had been in the past even if we knew some substances were present, and so forth. I don't know whether this responds to all of your questions here, but I think the flavor of a risk assessment is that when it is predicted, when it is done with nuclear powerplants, when it is done in financial markets, it is much the same. It is a way of organizing great uncertainties, and I think here we are dealing with a very difficult area. That is not to say something couldn't be done.

Mr. **Levitas**. Mr. Molinari.

Mr. **Molinari**. Let me start off by saying that having listened to the testimony of some of those who are victims, outlining the extensive health problems they have had, it is frustrating when you see people who are obviously ill, and there is obviously a connection to a condition of toxic waste exposure. Yet the chemical industry will say there is no scientific evidence to show that in fact those illnesses were caused by dioxin or other toxic waste. It leads me to believe that the attorney who testified yesterday, in suggesting that perhaps we should consider shifting the burden of proof after there is the establishment of an exposure of a serious magnitude, may have correctly identified the way to go.

I would like to just ask you this question, Dr. Carnow. We have a woman who testified yesterday, who had lived in Imperial, MO. She was right across the street from what we call the Minker property, which was contaminated by fill dirt laced with dioxin. In 1982 she went to the St. Joseph Hospital emergency room for bleeding kidneys and bladder problems. She was treated that year for sores that left scars. She had high blood pressure, was told that she had an extremely nervous condition, and was waiting for results from liver and immunity tests.

Her husband experienced swelling of the face, lips, and eyes. For 3 months his eyes were shut at times, originally diagnosed as allergy. In 1982 he completely lost his voice for over 5 weeks and was still hoarse for about 3 months thereafter. Throat continuously sore; bronchial problems.

Ron Jr., 17 years of age when they moved to this site, had respiratory troubles, severe stomach pains, repeated throat infections and colds, viruses, bronchial problems.

A daughter 11 years of age, nosebleeds, cauterization, severe chest pains, shortness of breath, rushed to the hospital with severe stomach cramps. Diagnosed as having hemorrhagic cystitis. Weight loss, the daughter, from 130 pounds to 116 pounds.

Son Ricky, 4 years old, throat infections, medication wouldn't clear it up. Blood clots.

Son Chad, 10 months old, vomiting, diarrhea, stomach problems.

And then interestingly enough, they sell. They get out. The son of the persons who bought the house, a 6-year-old boy, has a kidney problem, and on and on and on.

Now, Dr. Carnow, where you hear these symptoms, and you know that there has been a reported case of dioxin contamination across the street, would you say that there is a very good possibility that these problems, these health problems, have been caused by exposure to dioxin?

Dr. CARNOW. When you have that many people who are sick, and when you have some commonality, like commonality of abnormalities of bloodclotting, which we have also found, and kidney problems which we have known about at least since 1971, because in a horse arena in Missouri, one of the girls and a number of other people who were exposed had that as a first sign, and the 80 horses that died, many of those had hemorrhaging from the urine also, so that you have some kind of commonality. But again the way we would make this kind of a judgment is, first, by taking the kinds of history that you have related; second, looking at the kinds of damage to organ systems that are present in these people, to determine whether those are the kinds of damages that we see in individuals who are exposed, and put these together, because any one of these symptoms can be caused by a number of diseases, and one of the things that is always in contention is does this individual have nine diseases, which are related to one person, or do all of these symptoms and findings put together represent one disease.

One of the great men in medicine, Osler, said many symptoms, one disease. You don't try to make a separate diagnosis for each one. You can say that high cholesterol is due to eating fat and liver diseases are due to alochol and lung problems to smoking; and the kidney problem is common, everybody has kidney problems.

Again, if you see them as separate entities, then you see them as separate diseases. Unfortunately, that occurs. People go to a skin doctor and he says, "Well, you are using the wrong kind of a perfume," and then they will go to a urologist, and so on.

Mr. **MOLINARI**. Let me cut you off, Doctor. If they came to you, would you recommend that they move from the site?

Dr. **CARNOW**. I would recommend that they move from the site, because I know that dioxin is the deadliest chemical known to man. But the second thing that I would do is I would work him up in the way that I said.

Mr. **MOLINARI**. I understand.

Dr. **CARNOW**. And I would make a judgment. But what we are talking about is two things. One, is it safe to stay at Times Beach. I wouldn't go there even for 20 minutes. The second thing is, have these people been affected. I think that in order to make that kind of a determination, you would have to examine them and do the kinds of things that we said. The workups are fairly complex and extensive. If you just do simple tests, you may come away with no answer. I think that that is one of our problems. The workups on these people cost thousands of dollars.

Mr. **MOLINARI**. I understand.

Dr. Karrh, if somebody came to you—and this is not an isolated case; this is just one witness; the others that lived in the Times Beach area had similar symptoms—what would you recommend be done?

Dr. **KARRH**. I think I would take the same approach that Dr. Carnow said. I would like to examine the patients, I would like to talk with them about their symptoms.

Mr. **MOLINARI**. How about the recommendation that they move? Would you join in that recommendation?

Dr. **KARRH**. Based upon the information I have now, no, I wouldn't make that recommendation until I had worked them up and decided whether or not I thought the conditions they had could be related to where they live.

Mr. **MOLINARI**. How long would it take for you to make a determination that there is scientific evidence, as you put it, to indicate that exposure to dioxin was the reason for these people becoming ill?

Dr. **KARRH**. That is a question I don't think any physician can answer, because it depends upon each case and the amount of information you have from that case, and what results you are getting back from the lab tests and other tests that you are doing. But you have to do it based upon scientific determination, not on emotion.

Mr. **MOLINARI**. I understand that. But in the meantime the people are emotional because they are sick.

Dr. **KARRH**. I think we all get emotional when we get sick, yes, sir.

Mr. **MOLINARI**. And some of these nervous problems that people are told they have in fact have been diagnosed by others as not really being nerves. It is a manifestation of the problem. Indeed, it could be nerves, too.

Dr. **KARRH**. Yes.

Mr. **MOLINARI**. Triggered by the trauma that they have in the family and living under those conditions. The point is this: what I see, from what I have heard today, particularly from the chemical industry representative, is that we should not suggest that these health conditions are caused by exposure to known large quantities of dioxin in the area.

We have heard testimony about very small quantities being fatal to animals. Oh, yes, we can say, as the chemical industry will say, there is no relationship. In different animals, it takes different doses to die, and we don't know the human toxicity because we don't inject humans with the stuff to see how much of a quantity it would take to be lethal, if at all.

Dr. **Karrh**. Congressman, I don't think you have heard from the chemical industry today what you have said you heard. I think what you have heard us say is that we have not seen evidence of a significant health problem from hazardous waste. We fully agree that concerns in this area require a thorough scientific review.

We feel we ought to get on with that scientific review both through the efforts of the chemical industry, efforts of people such as Dr. Carnow, and from ATSOR that was set up by Superfund and then decide what types of regulations should be put in place.

Mr. **Molinari**. When did you come up with the decision that this study should be made?

Dr. **Karrh**. This was in my testimony that you objected to me reading earlier.

Mr. **Molinari**. You just made that decision. We just became aware that there could be problems associated with chemical substances? What I am asking you is, why did it take so long for the chemical industry to say we believe a study should be made at a particular point in time?

Dr. **Karrh**. We have been studying the toxicity of chemicals for many years. My own company started a toxicology laboratory in 1935 solely for that purpose, to study the toxicities. I think we only became aware that there could be some health problems in communities as a result of waste disposal sites within the last year or two. The Love Canal situation triggered a lot of the concern, people began to look more, and EPA began to look more.

The chemical industry has had a concern about this for quite some time. The concern culminated in November of last year when the study group, that I chaired, was put together to try to determine what was the extent of the scientific data that indicated that there were health problems as a result of waste disposal sites and waste disposal practices in the past.

We have concluded that we have not seen significant numbers of health problems, but we are not sure that there aren't problems out there, so we think that adequate scientific studies need to be done to determine if there are problems.

Mr. **Molinari**. That is where I have a problem. You are not sure that there are significant health problems out there. You are completely ignoring the empirical data that exist all across this Nation. Every place where you have a known dump site of a serious magnitude, the people living in the vicinity are exhibiting similar symptoms. I am no expert, but when I hear the same symptoms—nausea, severe headaches, severe rashes, urinary infections in the case of females, and some of the others worse than that, bronchial conditions, asthma in very high quantities, what I suggest to you is that you are blinding yourself to the obvious. You have an industry bias and you simply are not looking at reality.

Dr. **Karrh**. I don't agree with you, sir.

Mr. **Molinari**. Of course, you don't agree with me. I would fall off the chair if you agreed with me.

Dr. **Karrh**. As Dr. Carnow said earlier, occupational, environmental diseases are not unique diseases. They are the same as all other diseases. People all over the country have all the diseases you are talking about. Just because they have the disease doesn't mean they are related to waste disposal sites. They mean they are people, and people have diseases. We want to find out if they are related to waste disposal sites.

Mr. **Molinari**. If you weren't a representative but a member of Congress, or an EPA, state or city health department official, and you know there is a site and that the people surrounding that site are running to their doctors in high numbers, all having the same symptoms, don't you begin to say there might be a problem that should be addressed?

Dr. **Karrh**. Absolutely, and I would turn to EPA and CDC and the chemical industry, saying you ought to do adequate studies to see if there is a relationship to that. That is precisely what we are doing, studies encouraged by lawsuits and others to get CDC to do the studies to see if there are problems related to waste sites.

Mr. **Molinari**. Has there ever been a study conducted and completed which concluded that, in fact, the health problems were caused by chemical agents near the premises?

Dr. **Karrh**. Yes. I reported that in my testimony. The Hardeman County, TN, study indicated that people were suffering some change in their liver function studies as a result of a contaminated aquifer. Once they stopped using the water from that aquifer, their symptoms went away. Studies do show whether or not there are health problems related to waste disposal sites, if you do the studies properly and adequately.

Mr. **Molinari**. Regarding those who suffered from the liver problems during that time, whatever the time frame was, do you think they should be compensated for the problems that they had?

Dr. **Karrh**. Our position is that any victim, any person who is a victim of the action of someone else, whether it is a chemical company, a person or an individual or anyone else, that has a compensable type of condition, should be compensated by the person that caused the injury.

Mr. **Molinari**. I am happy to hear that.

Dr. **Karrh**. It was in my testimony.

Mr. **Molinari**. Yes; I know. You read it.

Dr. Carnow, do you agree——

Mr. **Levitas**. Can I ask one question on that point before you go on to another?

Mr. **Molinari**. Sure.

Mr. **Levitas**. What degree of scientific evidence or proof do you think should be required as a result of these studies, in order to bring about action, either in the case of compensation provided under the existing tort system, under a system of victims compensation which Congress might legislate, or for EPA to take action such as that taken on Times Beach?

How much information do you have to have before you would, as a scientist, be willing to say now is the time to make a decision or pay compensation?

Dr. **Karrh**. That is a question that you can't answer with an across-the-board answer, because it will vary a lot from the types of data that you have and the strength of the study that you do and the so-called power of the study, and other things.

It would vary tremendously. But under the tort system, the definition of scientific certitude is quite different than it would be under some other type of compensation scheme because there the decision is made by a judge or jury based upon their evaluation of the data that is presented.

Under a compensation mechanism, if such a mechanism was put in place, you would like to have the available data reviewed by the appropriate scientific expertise, whether it is an individual or a group of individuals, to come up with an opinion that, yes, based upon the data that are available, there probably is good scientific evidence there is a cause-effect relationship. You wouldn't want to just take anecdotal information and then fashion some type of remedy from that.

Mr. **Levitas**. Does it have to be scientific certainty or reasonable likelihood?

Dr. **Karrh**. I think that would depend upon what the individual who was either adjudicating the case or the scientist who was looking at it decided. In my own opinion, if the scientific data was adequate to lead me to conclude that there most probably was a relationship, a cause-effect relationship, then that would be adequate for me. Someone else may want to see 90 percent probability. Other people are satisfied with much less, 25 or 30 percent, but generally that is not a good scientifically valid type of relationship.

Mr. **Molinari**. But you wouldn't agree that the problems in Times Beach, the health problems complained of by the people there, most probably were caused by the presence of dioxin in the vicinity?

Dr. **Karrh**. I can only answer that, Congressman, by saying as I said earlier, that based upon the data that I have reviewed, both in scientific data and in the lay press, I have not seen anything yet that convinces me that the problems they have, or anything that even makes me think that the problems they have, are related to the dioxin contamination of Times Beach.

I hope that other studies are done such as Dr. Carnow talked about and these studies will be published, and then we can do, scientific peer review and let a group of scientists, not me but groups of independent scientists, decide whether or not there is a cause-effect relationship.

Mr. **Molinari**. But you would go along with the recommendation that the people be moved from the area?

Dr. **Karrh**. No, sir. As I said earlier, based upon the data I have seen, I would not go along with that recommendation at this time, but that is a decision that was made from an administrative standpoint that I had nothing to do with, and consequently was not consulted on.

Mr. **Molinari**. Dr. Carnow, Dr. Karrh said in his testimony that CMA "has found only two valid epidemiology surveys of populations relative to waste disposal sites." Do you believe that to be accurate, from your own background?

Dr. **Carnow**. Well, it depends on what your definition is of waste disposal sites, and it depends on your definition of study. If you are talking about full-blown studies, not many full-blown studies have been done.

Mr. **Molinari**. Surveys. I guess there is a difference, I don't know. I have to ask you people, the experts. Is there a difference between surveys and studies?

Dr. **Carnow**. Again, I recounted to you at least a half dozen. I believe that there are more. There have been reports in areas in the West, where people have been exposed to radioactive tailings of increases in leukemia. There have been other reports. Again, I have not done these studies, but I am firmly convinced that if we look very carefully, we will begin to make associations. One of the problems that Dr. Karrh alluded to is the question of scientific certainty.

I think that you are not going to find the kind of pure values you want if you look at cancer and only look at a thousand people, because 10 cancers in 1,000 can be epidemic, if it is a population that doesn't have a lot of cancer, so that you may have to wait 200 years before you can make a determination.

We estimated you have to use 600 animals, and so on and so on, for various valid experiments. If you are talking about cancer, you have to use thousands in order to make a determination. Again, the problem that we have, a physician will make a diagnosis on a patient with very little evidence, because the realities of life say that you have to, and it is based on a history of physical exam and some lab tests.

When you have more than one individual, you have groups of individuals, they have similar symptoms, again if you just take the dioxin business, there is such a repetition of these kind of findings that there is no question in my mind about what these people have, based on what you have told me. Again, I have not examined them, but those whom I have examined, who have those symptoms, have exhibited physical findings and laboratory abnormalities which are consistent with and in some constitute a dioxin syndrome.

Let me just mention in regard to remaining in Times Beach. I think that, certainly in regard to animals, we know that dioxin is the most potent cancer promoter known. It is 30,000 to a half million times more potent than 3 methylchlorolanthene, which has been used to spike research so that animals will develop lots of tumors. I have read—I have not seen this study—that NIOSH, National Institute of Safety and Health, has developed a registry of dioxin workers.

It has been in effect for 2 years. I understand that they will be publishing a report which suggests that they found in the first 2 years 25 times the normal numbers of sarcomas, which is a form of cancer, in these workers. Again, I have not seen the study, but in any circumstance where you find an excess of cancer, particularly this kind of cancer, and you know that there are levels, significantly high levels in a place—and I think they were measured at Times Beach and they were high, certainly 30 to 50 to 100 times higher than CDC considered safe—it would seem to me you would have a responsibility to remove people.

Again, recognizing the dislocations, the trauma to the people and so on, I cannot feel that those people should have been left there waiting for peer value to appear.

Mr. **Molinari**. Dr. Carnow, have you ever appeared as a witness for a plaintiff in a court suit based on health injuries that were allegedly caused by exposure to hazardous wastes?

Dr. **Carnow**. Yes, I have. In regard to dioxin, in Missouri there were 47 railroad workers who were exposed, and I examined all of those workers, my staff and I examined all of those workers, on two occasions. It was our medical opinion that these workers were suffering from a syndrome, a disease complex caused by dioxin; yes, I did.

Mr. **Molinari**. In deference to the other members of the subcommittee, I am going to ask one more question until it is again my turn. I would just like to ask you, Dr. Karrh, would you have any apprehension about moving your family into Times Beach and living there for, say, from 1 to 5 years? How would you feel about the possibility of a health problem affecting you there?

Dr. **Karrh**. Based upon the information I have seen, I always have to preface it in this way because I have not been to Times Beach, I would have no hesitation moving my family to Times Beach for 4 or 5 years.

I would like to point out on the incident that Dr. Carnow just mentioned, the Sturgeon, MO, incident where the 47 people were supposedly injured by a transportation accident, that precisely supports the position that the chemical industry has taken in that we are saying that we don't know if there is a problem or not.

If there is a problem, we don't yet know the magnitude. We want to do studies to find out exactly what that problem is, and the magnitude. But in the meantime, the existing systems, existing mechanisms, are adequate to cover the victims compensation issues until we find out what the magnitude of the problem is.

The 47 people in the Sturgeon, MO, situation, through the court system, received a settlement of $58 million, if you can believe in the American Lawyer Journal report, and that seems to have been compensation for any injuries that they may have suffered. That is in Sturgeon, MO.

Mr. **Molinari**. I have to ask you one more question, since you said that you would have no hesitation, based on what we know, to move your family into Times Beach, MO. Pardon me for being blunt, but if you did that, and your family started to experience symptoms like nosebleeds and rashes and kidney problems and nausea, sore throats, bronchial conditions—symptoms that everybody else is getting—how long would it take you to get the hell out of that area?

Dr. **Karrh**. I think that is a question, Congressman, that everyone would answer differently. I certainly would evaluate the data that I had, and I would look at it scientifically and objectively and decide whether or not it was related to the place where we were living or if it was something else. I wouldn't arbitrarily, because I was emotional about it, jump to the conclusion that it had to be the place we were living.

Mr. **Molinari**. I may disagree with you but I admire you for your consistency.

Dr. **Karrh**. I was sworn in when I became a witness, and I have tried to stay honest.

Mr. **Levitas**. Thank you, Mr. Molinari. We will go back around. I have a couple of other questions.

As I look at the nameplates out there, everybody has the name preceded by Doctor. We have our own doctor on this panel as well, and I now recognize the gentleman from Georgia, Dr. Rowland.

Mr. **Rowland**. Thank you very much, Mr. Chairman, and may I take this opportunity to welcome you, my colleagues, to this committee hearing, and tell you how much I appreciate your being here. We are confused, it seems, whether or not certain of these processes are caused by these agents. The Congress is somewhat confused by the contradictory testimony in the hearing.

This is certainly the problem we are trying to resolve at this time. For example, yesterday we heard from the mayor of Times Beach, we heard from her about many, many different disease processes that she related to exposure to dioxin. On the other hand, I have heard in the Veterans' Affairs Committee that there were veterans who were in Vietnam that were drenched with dioxon day after day, month after month, several years ago, and experienced apparently no ill effect from this having taken place. So you can see the contradictions that we are hearing.

The mayor yesterday testified, some staggering testimony to me, that in Times Beach there were teenagers who were experiencing breast cancer, which she related to the exposure to dioxin. I have been in medical practice for 30 years and I don't recall ever seeing a teenager with breast cancer.

She told of a family that had a child with neuroblastoma. Very unusual chances. The chances of that happening, again, were extremely rare, the next child she had was also born with this. She talked about one block with 15 families and several of those households had seizure disorders.

We heard this over and over, pelvic tumors, for which there was no diagnosis, and I inquired about pathologic reports. No pathologic reports. The doctor just said they didn't know what the cause was.

It is very confusing, the testimony that we have been hearing, and I realize that it is very difficult to have absolute determination of whether or not these chemicals are causing these disease processes, and it takes a long time to determine that.

I would like to ask about the Times Beach testimony that we heard yesterday. Do we have, to your knowledge, any documented evidence from physicians of some of those things that I mentioned that she testified about yesterday, to your knowledge?

Dr. **Karrh**. I have no knowledge, documented knowledge, documented information from physicians regarding what you describe as being testified to yesterday.

Mr. **Rowland**. Dr. Carnow?

Dr. **Carnow**. No. Again we don't solicit, as you know, physicians. I have not been called into Times Beach by anyone, so I have not been privy to the medical records. I have spoken with the mayor on a number of occasions. And I do know about some of the problems that she has expressed. I have not seen physicians' records.

I also know that the things that you recount, what we have seen in other communities and that others have reported in other countries in other circumstances, and so on, there is a commonality of symptoms and findings in regard to this chemical. I think sometimes, however, that, you know, this chemical is such a very toxic one that I think it is actually relatively easy to make associations. I think the problems we have are with those chemicals not quite as deadly, not quite as toxic, in trying to make determinations about relationships.

I have no difficulty in my mind with the relationship with dioxin given a good test protocol which includes neurophysiologic, central nervous system, liver, a host of other tests which look at many parameters that dioxin can affect. Again, what you are talking about is something that I am familiar with, not in Times Beach, but in other groups we have examined.

Dr. JASON. Dr. Henry Falk at CDC has been heading up a program to look at the chemical analysis of the individuals involved in Times Beach. They have a group of around 140 individuals thus far where they have looked even at immune function. I think he would probably help you out with some of your questions concerning their look at cancer incidence, also. So I think probably the most appropriate people in this case would be the CDC and Dr. Falk who has been looking into that area.

Mr. ROWLAND. Do you know if they have established a relationship?

Dr. JASON. Some of the analyses even of the blood at this point is just being done. So I think it would be premature to draw any real conclusions. He can tell you the status of the chemical analyses. I am not aware of how intensively they have looked at the cancer incidence. One of the jobs I have at EPA is risk assessment. Present data sets, whether they be the National Cancer Atlas or others, are usually a mortality indices. What we are dealing with here is a changing spectrum in the use of health outcome data.

If we look at the childhood cancers, these children are actually surviving. The majority of these children are not dying. So if you look at mortality statistics to address areas of concern, it can be misleading depending on age groups you are looking at. So the kinds of data sets we have relative to the areas of concern could be misleading in some cases depending upon the age group examined. It is a concern of our agency to go further in this area.

Mr. ROWLAND. Would you say at this time, then, that the disease process as it has been reported from Times Beach has not been established as a cause-effect relationship with dioxin?

Dr. JASON. I think the difficulty, speaking not only as a physician but a scientist, also, is the fact that isolated reports are very difficult to handle, mostly because of the fact that diseases do occur, even occasionally constellation of diseases occur but with rarity. As you begin to accumulate more and more data as Dr. Carnow was saying, it becomes less and less likely. I think one of these that would be helpful would be medical records, an occasion to look at those individuals and make sure that what we are seeing is consistent with what is being reported.

I think that is always the difficulty. The human tragedy of illness is something that is certainly in all of our minds. It gets down to the bare essentials of what is the disease process? Is there consistency of the specimens with a common etiology. I would say in answer to your question yesterday about whether a pathological diagnosis made, some diagnosis was made. Something may have been written that said it was impossible to make an absolute diagnosis, but there will be at least a histopathological description of the cells. So I think there is information one can still draw upon. At least that is generally the case in medicine. I would hope that Times Beach uses a similar medical procedure.

Mr. **Rowland**. I guess that we are impatient to learn about this. Mr. Molinari referred to that on more than one occasion. Do you have any idea of when we might learn of the relationship insofar as Times Beach?

Dr. **Jason**. I think the difficulty to a certain extent, perhaps because I am a pediatrician, is the fact that if we look at the turnaround time in events such as cancer, we talk about latency periods in many cases of 14 to 30 years. The mobility of a young population is such that even if cancer were to occur and that is the chosen outcome, many of the individuals will have already moved. So it will be difficult to look at an increase in a specific area, and trace it back to the appropriate exposure. If we look at others, such as Dr. Carnow's suggestion of major malformations, as one, it is extremely important to address that issue. As I am sure some of you may know, the birth defects monitoring program of CDC has been attempting to do that over 12 years.

There are many inadequacies of the BDMP data base which they realize. It was an organized attempt however, to look for areas of increase in birth defects. Thus far they have not been able to find a single chemical or drug related birth defect outcome using the birth defects monitoring program. This raises some concern over the sensitivity of this type of approach for our needs.

We at the Agency right now are using another one. That is the birth weight, which is an extremely sensitive indicator, perhaps overly so. But it is consistent with the animal evidence of embryo toxicity. In this case, in conjunction with New York, as a matter of fact, Mr. Molinari's area, we are working with the census tract level to look for statistical increases of low birth rate that may be correlated appropriately with exposure.

The reason I think that this is important is that it is very difficult with long latencies, even in chronic diseases to begin to associate or even correlate a given exposure with a given outcome. I think we have to be thinking in the direction of several indices that have been formulated. One is the National Death Index where given a person's name, if we have a list of individuals in a certain area, we can find out if they have died. And if so, what the cause of death was through the death certificate.

Thus we begin to refine our approach of finding mortality data compatible with the exposure. We have the Chronic Disease Index which is presently being developed through CDC. As you can imagine the problem there will be inadequate ascertainment for certain areas as well as an inappropriate diagnosis in certain cases. The same problems that plague major malformations reporting will be a problem here, not to say it isn't important, but that we have to realize the limitations of the data we are using. When we talk about body burden on an individual basis, as Dr. Carnow has already said, it is very difficult in many cases to get accurate data.

For example, to get enough adipose tissue to do a dioxin analysis would be on the order of a one-half gram of adipose. We would have to go in with a scalpel to be able to get enough tissue. So an adequate source of reasonable substance, whether it be blood, adipose tissue, or whatever, to begin to draw the important kinds of correlations can some times be very difficult. Not impossible, but certainly difficult.

Mr. ROWLAND. In that respect, the long turnaround time, Mr. Chairman, there are a few more questions I want to ask.

Mr. LEVITAS. Go right ahead.

Mr. ROWLAND. Dr. Lewtas, I may refer to this, would chromosomal analysis help us any and shorten that turnaround time with reference to dioxin?

Dr. LEWTAS. I think there are beginning to be in addition to chromosomal analysis, a number of genetic human monitoring tools available which we can use in population studies. CDC is using these tools, and did use them in Love Canal. I am not sure whether they are being used in Times Beach or will be used there. I think those tools are useful in that many of those methods are rapid and that should help. In addition to the chromosomal analysis method, several other methods are now available for screening long term chronic effects, e.g. genetic effects, by doing analysis on blood and possibly sperm and tissues in different body fluids——

Mr. ROWLAND. Not to interrupt you but would you give me a detailed description of those techniques for the record?

Dr. LEWTAS. Yes.

[The information received follows:]

GUIDELINES FOR STUDIES OF HUMAN POPULATIONS EXPOSED TO MUTAGENIC AND REPRODUCTIVE HAZARDS

Editor
Arthur D. Bloom, MD

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EXPOSED TO MUTAGENIC AND REPRODUCTIVE HAZARDS

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As part of our efforts to achieve these goals, we sponsor, or participate in, a variety of scientific meetings where all questions relating to birth defects are freely discussed. Through our professional education program we speed the dissemination of information by publishing the proceedings of these and other meetings. From time to time, we also reprint pertinent journal articles to help achieve our goal. Now and then, in the course of these articles or discussions, individual viewpoints may be expressed which go beyond the purely scientific and into controversial matters. It should be noted, therefore, that personal viewpoints about such matters will not be censored but this does not constitute an endorsement of them by the March of Dimes Birth Defects Foundation.

DEDICATION

To the residents, past and present,
of the Love Canal of New York State:
Lest you think it was all in vain.

PREFACE

In 1980, the results of a cytogenetic study of the residents of the Love Canal in upstate New York created extensive debate within the scientific community and profound anxiety within the studied population. In response to the scientific controversy and out of concern for the well-being of the residents of other communities contaminated by toxic wastes, four agencies of the U.S. Government (the Center for Disease Control, the Environmental Protection Agency, the National Institute of Environmental Health Sciences of the National Institutes of Health, and the Occupational Safety and Health Administration) and a private, non-profit foundation (the March of Dimes Birth Defects Foundation) jointly organized and sponsored the Conference on which this book is based. The Conference was held from January 26-27, 1981, at the Washington Hilton Hotel, Washington, D.C., and was attended by 90 invitees.

It was decided early in the planning stages of the Conference that the emphasis would be on genetically related end points, since those were clearly and properly primary concerns during the Love Canal episode. To that end, three Panels were established: one on the cytogenetic effects of genotoxic chemicals on human populations (Panel I); one on reproductive effects (Panel II); and one on testing for mutagens in human populations (Panel III). Draft reports were prepared by each Panel of scientists, and the reports were circulated prior to the Conference. The Conference itself was devoted to a general discussion of each report, followed by an in-depth critique and revision of the report by those attendees most knowledgeable in the area. Final responsibility for the content rested with the Co-Chairpersons for each Panel: Anthony Carrano and myself for Panel I, Zena Stein and Dorothy Warburton for Panel II, and Fred deSerres and Michael Waters for Panel III. James Neel presented a summary of these proceedings from a human population geneticist's viewpoint, and Robert Miller summarized numerous other toxic exposures of humans. Their papers are an integral part of this volume.

We wish to emphasize here that the reports are meant to serve merely as guidelines to the proper conduct of the types of studies discussed. The reports represent, generally, a consensus of views on the part of the panelists, as well as of the Conference participants; but they clearly do not mean to imply universal acceptance by the scientific community of the approaches described. Our intention was simply to provide guidance to those contemplating these types of studies in human populations. The recommendations may be regarded as reasonable, state-of-the-art suggestions as of 1981.

The Steering Committee for the Conference consisted of: Fred deSerres of NIEHS, Clark Heath of CDC, James Neel of the University of Michigan, Gabriel Stickle of the March of Dimes, Dorothy Warburton of Columbia University, Michael Waters of the EPA, and myself, also from Columbia. The March of Dimes served as the coordinating agency, and in large measure, the success of the Conference was due to the efforts of Gabriel Stickle, Phyllis Wright, and, especially, Natalie Paul of the March of Dimes. Mrs. Paul did a superb job as Associate Editor of this volume, and we are very grateful to her. Marci Allmond of my office ably assisted the March of Dimes, and especially me, throughout the Conference and the preparation of the book. To all of these people, and to numerous others, I am indebted.

A high degree of interaction among agencies of the U.S. Government and among scientists of diverse orientations is reflected in this publication. This book is our attempt as physicians and scientists concerned with the public health to avoid more Love Canals.

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Welcoming Remarks

Gabriel Stickle,
Sr. Vice President for Planning and Administration
March of Dimes Birth Defects Foundation

On behalf of the March of Dimes Birth Defects Foundation, one of the four sponsoring organizations, I welcome you to the Conference on the Evaluation of Human Populations Exposed to Potential Mutagenic and Reproductive Hazards.

It seems unnecessary for me to elaborate on the motives for organizing this conference. For many months in 1980, Love Canal shared the front pages with Iran, and our direct attempts at their solution had failed in both, or so it seemed at the time. The bitter aftertaste of our failure was tinged with the fear that we may fail again, if the scenario were to be repeated. If ever the time were ripe to formulate guidelines to study actual or suspected environmental hazards to a localized population, it is now.

We are not under the illusion that guidelines, by themselves, will solve the problem, that is, will achieve a consensus as to the presence, magnitude, and nature of the hazard or who in fact is at risk and to what degree. The interactions among ecologic, medical and sociopolitical factors involved in a situation like Love Canal are so complex and so little understood that we must know much more than we do now to be confident about what public health measures are needed to protect people from potential hazards of the high technology society.

What the guidelines could do, of course, is to minimize mistakes. This is the first and necessary step in achieving adequate control. Equally important is that the guidelines may help close the ranks of the scientific community faced with other Love Canals. Divided opinion among experts generated a kind of intellectual smog that still hovers over Love Canal. And not a little of the anxiety and heartache of Love Canal residents must have come from uncertainty about purported dangers which fanned the flames of uninformed speculation.

It goes without saying that whatever guidelines emerge from this meeting will be subject to continuous revision and amendment as we gain in experience and understanding. A standing interagency committee to carry on this task may be in order.

While science has much to do, the public has much to think about. Faced with a potential danger, many of us conjure up an apocalypse. Nuclear energy has conditioned us to this propensity, perhaps to a degree that may not be appropriate to most other environmental hazards. The public and its principal information source — the media — have to learn to balance the fear of missing hidden hazards against the harm of inflating non-hazards.

The public needs to learn that there is great variation not only in the way a mutagen or teratogen reaches human subjects but also in human capacity to detoxify these agents. Of 10 pregnant women exposed to a teratogen one may have a spontaneous abortion, another a growth-retarded baby, another a child mildly mentally retarded at age 3, and the rest may have normal offspring. The timing and intensity of exposure may have contributed to the outcome but so could the ways in which the 10 mother-fetus pairs disposed of the teratogen.

In these and other respects, therefore, there is a need for better public understanding that clearly matches the need for better professional judgment. The intention of the March of Dimes is to serve both these needs.

With that I shall wish you well in your labors and give the podium to the organizer of this conference, Dr. Arthur Bloom.

REPORT OF PANEL I

GUIDELINES FOR CYTOGENETIC STUDIES IN MUTAGEN-EXPOSED HUMAN POPULATIONS

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PREFACE

When cells are exposed to either physical or chemical mutagenic carcinogens, one thing that is readily seen is induced cytogenetic damage. If the damage is in germ cells, in theory it can lead to sterility in the exposed individuals themselves or to detrimental mutations that could be expressed in their offspring or descendants. If the damage is in somatic cells, it possibly could lead to somatic effects such as cancer or even premature aging. This somatic damage usually is manifested as increased levels of chromosome aberrations or sister chromatid exchanges (SCEs), which can be quantitatively related to the exposure.

With ionizing radiation, which is the most extensively studied mutagenic carcinogen, the induction of aberrations in long-lived circulating lymphocytes has proved its utility as a biologic dosimeter to estimate the absorbed dose.

With chemical mutagens the situation is more complex. For instance, the mechanisms of interaction with DNA differ, and such factors as the presence or absence of activating or deactivating enzymes, the biologic and chemical half-lives of the chemicals, the degree of DNA repair, etc., all can affect the yield of aberrations. Nevertheless, the formation of chemically induced cytogenetic changes in lymphocytes of exposed people has already shown itself to be a sensitive indicator of low level exposures, brought about by such factors as occupational exposure (e.g., uranium workers). The cytogenetic system, then, is among the most sensitive systems that can be used to monitor exposure of human populations.

It should be noted that, while chromosome aberrations are deemed undesirable, as are induced mutations in general, the presence of cytogenetic changes cannot be used to predict specific health effects in an individual. However, they do give an estimate of the magnitude of an exposure that could increase the risk of disease in the population.

Consequently, cytogenetic study of both chromosomal aberrations and SCEs has a clear place in the evaluation of human populations exposed to known or suspected mutagens. However, such studies must be done carefully, with appropriate controls and with proper methods for statistical analysis of the data. This document attempts to provide a consensus by cytogeneticists of how such investigations should be structured, what laboratory techniques should be used, and how the resulting data may be reasonably interpreted.

I. CYTOGENETIC INVESTIGATIONS: BACKGROUND

Determination of chromosomal aberration frequencies in peripheral blood lymphocytes from persons or populations exposed to ionizing radiation has become an established technique, and substantial agreement among cytogeneticists has been achieved as to methodology and confounding variables [1]. Unfortunately, the current state of our knowledge of the cytogenetic end points of interest in connection with other, particularly chemical, mutagens has not always been available to those sponsoring, planning, or evaluating such human cytogenetic investigations. This document attempts to provide those so involved with a consensus view by cytogeneticists of the current state of our knowledge of the technical and procedural factors that properly should be considered in such investigations.

While there is ample evidence of the usefulness of cytogenetic measurements to detect ionizing radiation exposures of human populations, (such as the survivors of the atomic bombings of Hiroshima and Nagasaki [2], uranium miners [3], nuclear dockyard workers [4], and others), experience with cytogenetic studies of populations exposed occupationally or environmentally to chemical agents is still very limited. Hence, studies of populations so exposed must be approached with caution. Our lack of knowledge about normal background levels of aberrations, about sources of variation and important confounding variables, and the sensitivity of different cytogenetic end points for detection of exposures to low levels of most chemical agents presently precludes a detailed description of just how a "proper" study should be designed, except in the broadest terms. Though it is doubtlessly going to be deemed desirable to do some studies of individuals or populations whose exposures are ill defined, a high priority must be given to those studies that can provide some of the information involving populations we now lack, such as those occupationally exposed to known mutagenic or carcinogenic agents, for whom at least some sort of dosimetry is possible.

End Points

Two end points, chromosomal aberrations and sister chromatid exchanges (SCEs), are now in wide use. Though the use of other tissues, such as bone marrow, is technically feasible, for practical purposes the only material likely to be used for population studies is the peripheral blood lymphocyte in short-term tissue culture.

Chromosomal aberrations constitute one class of mutational event, and thus their induction, even in somatic cells like circulating lymphocytes, clearly implies that there may be a germ line genetic hazard as well (providing, of course, that the inducing agent also reaches the gonads). Furthermore, many, if not most, agents causing aberrations, or clastogens, are also carcinogens, at least in animals. Determination of aberration frequencies has become a widely used and accepted radiation dosimetry technique. Spontaneous levels and confounding variables for the chromosome-type aberrations induced by ionizing radiation in peripheral lymphocytes are well established, and it has thus become relatively easy to design studies to detect particular levels of radiation exposure.

Unfortunately, attempts to use aberration frequency determination as a measure of exposure to potential chemical clastogens, especially those that induce chromatid-rather than chromosome-type aberrations, have begun only recently. Further, much of the information one would like to have to design and interpret either individual or population measurements is inadequate or unavailable. We particularly need information on spontaneous aberration frequencies, on interindividual variation, and on the persistence of chromatid aberrations and their derivatives. Until the necessary information is acquired, studies of persons or populations exposed to chromatid aberration producing agents, and the interpretation of chromatid aberration data must be approached conservatively.

SCEs, in contrast to aberrations, are not, as far as we know, mutational events, in the strict sense. We are aware of no health consequences of SCEs *per se*. Determination of SCE frequencies is, nevertheless, widely advocated as an indicator of potential genetic or carcinogenic hazard. Many believe the phenomenon to be a manifestation of some form of repair of damaged DNA or an indicator of mutation. A large number of mutagens and clastogens have been shown to induce SCEs and, for a number of agents, increases in SCE frequencies are associated with increases in gene mutation and are detectable at levels of exposure far below those required to produce detectable increases in chromosomal aberration frequencies. In addition, many persons find SCEs quicker and easier to score than aberrations. To the extent that SCEs are a measure of DNA damage which is likely to result in mutagenesis and carcinogenesis, it seems reasonable to include SCE frequency as an end point in cytogenetic studies, It is important, however, to note that there is a major class of mutagens and carcinogens (ionizing radiation and a few truly radiometric drugs) that are poor inducers of SCE, just as there are others (like diethylstilbestrol) that are poor inducers of aberrations.

Though there is controversy as to whether truly spontaneous SCEs exist, or whether all of those seen in otherwise untreated cells are in fact the result of the incorporation of the base analogs required for their expression, for practical purposes there is a background level of SCEs against which any increases in frequency must be measured. The levels seen are numerically far higher than spontaneous chromosomal aberration frequencies, with a number of SCEs generally being present in each metaphase spread examined. Furthermore, the study of variation in human peripheral lymphocyte SCE frequency and the factors influencing it has begun only recently. Thus, as with chromatid-type aberrations, we still lack much of the information necessary to design and interpret SCE studies as confidently as we would like.

Populations

Cytogenetic studies of persons or populations for increases in chromosomal aberration levels have been useful in two ways. As already suggested, aberration frequency determination has become a standard method of biologic dosimetry in cases of individual known or suspected exposure to ionizing radiation. It has proven particularly useful in cases where possible exposure has been suggested by physical circumstances, even though actual exposure seems unlikely. If no increase in aberration frequency is found in such a case, the cytogenetic findings suggest that no significant exposure did in fact occur, often providing significant relief of anxiety. It must be kept in mind, of course, that a negative finding does not rule out *some* exposure, any more than finding an aberration indicates definitively that there *was* some exposure. Though we now lack much of the background information necessary to derive a cytogenetic "biologic dosimeter" for chemical mutagens and carcinogens, it seems likely that we will ultimately be able to use aberration (and SCE) frequencies in a quantitative fashion in chemically exposed populations.

The second useful application of cytogenetic studies has been the study of populations exposed to ionizing radiation, both at low and high exposure doses. In this application it has proven possible to demonstrate statistically significant elevations in aberration frequencies for populations, and even subpopulations, grouped by broad dose categories, even though individual levels may not be elevated significantly above suitable controls. The extension of such studies to populations occupationally or environmentally exposed to other potential mutagens and carcinogens must be approached with caution, however. It remains to be confirmed that chromosomal aberration measurements are sufficiently sensitive to "detect" occupational (i.e., mostly low) levels of exposure. This confirmation will be dependent on the availability of physical dose estimates, and would, of course, be impossible for exposures to indeterminate doses. Needless to say, such confirmation studies must include carefully selected control groups, and attention must be given to the existence of both *known* (for example, other drug or radiation exposures) and *unknown* confounding variables.

Another possible application of cytogenetic studies of populations is for qualitative "triage," to sort out the more heavily exposed members of a population of individuals exposed to differing doses. This might be of use in tracking down the identity and source of some clastogenic or SCE-inducing agent, for example, or even as a basis for clinical management, in the case of uneven, accidental exposures of human populations.

II. METHODS FOR THE STUDY OF INDUCED CHROMOSOMAL ABERRATIONS

It is recognized that a wide array of protocols exist for the culture of human peripheral leukocytes for the purpose of doing cytogenetic analysis. It is not the intent of this Committee to recommend specific protocols to be followed in cytogenetic studies but rather to discuss the general problem areas that, if not considered, can invalidate a cytogenetic monitoring study. The problem areas that are the most easily identified are: 1) the assurance that the maximum effect is observed; 2) the introduction of extraneous factors that can modify the observed effect; 3) the identification of the appropriate cytogenetic end points to be screened for and the application of the best procedures for detecting them; 4) the randomization of both control and study cultures and the blind analysis of the slides; and 5) the possibility of not observing a clastogenic effect due to the inherent insensitivity of the human peripheral lymphocyte system to in vivo exposures of certain classes of chemicals.

Past experience with ionizing radiation has amply demonstrated, and reemphasized, the necessity of analyzing cells in their first posttreatment mitotic division in order to determine the maximum frequency of chromosomal aberrations. This is particularly true for exposures to purported chemical mutagens. It has been established that the great majority of chemical clastogens are S-dependent [5]. S-dependency, in the case of cytogenetic phenomena, means that the substance must either be present during replicative DNA synthesis, or the lesions it produces in the chromatin require replicative DNA synthesis to be translated into a structural aberration. The aberrations so produced are invariably chromatid-type and very transitory in that they are generally lost at the next cell division. This loss is either through cell lethal effects or through conversion to chromosome-type aberrations. Although, most chromatid aberrations are cell lethal, a significant frequency are expected to persist as balanced symmetric aberrations (e.g., reciprocal translocations and inversions). These aberrations are difficult to score with as high a degree of quantitative precision. Hence, the analysis of 2nd, or later, divisions will greatly underestimate the true level of damage produced by S-dependent clastogens, particularly if the effect is being produced by a long-lived lesion that was induced in vivo.

A method of insuring the analysis of 1st division cells that has been widely used in radiation studies is that of adding BrdUrd to the culture medium and performing one of the differential staining procedures that allows for the detection of 1st, 2nd, and subsequent divisions. The investigator can insure the observance of the maximum effect by analyzing only 1st division cells. If this procedure is employed, the investigator is urged to employ a BrdUrd concentration that does not significantly elevate the spontaneous aberration frequency. If the BrdUrd procedures are not utilized, care must be taken to insure a maximum frequency of 1st division cells. This can be done by fixing the cultures at an appropriately early time, usually 42-48 hours after initiation, depending upon the culture medium. It is noted, however, that fixation after 54 hours is unacceptable. By definition, S-dependent chemical clastogens are not expected to produce structural aberrations directly in the circulating lymphocytes as less than 0.1% of the lymphocytes possess an S phase. They may produce aberrations during the in vitro S phase if the lesions persist, as discussed, but they will generally produce their effect in the lymphocyte precursor cells. Therefore, the most common type of aberration that is expected to be observed is the chromosome-type lesion that is derived from an initial chromatid-type lesion. The majority of these are expected to be of the balanced symmetric type, and unless highly unequal so that obvious karyologic changes occur, will be difficult to quantitate. It has become a common practice in some laboratories to score banded chromosome preparations. It is felt that the use of banded chromosomes does not sufficiently increase the sensitivity of detecting the balanced aberrations. This, coupled with the difficulty of scoring chromatid-type aberrations in banded preparations, removes the necessity of scoring banded preparations, in the view of this Committee.

On the other hand, in those instances in which F_1 studies are conducted and only the balanced symmetric aberration types are expected to be seen, the slight-to-moderate increase in the efficiency of detecting them in banded preparations justifies the use of banding procedures.

There are many tissue culture media and medium compositions that are suitable for the culture of human peripheral blood lymphocytes. The precise medium composition is left up to the investigator. However, the use of virus-free serum is mandatory and care should be taken to avoid the addition of any other substances that conceivably could produce chromosome aberrations. Although the use of whole blood presents the possibility of carry-over of a mutagenic substance from the donor's serum, thus leading to the in vitro production of aberrations, this procedure is acceptable. Regardless of the culture method employed, it is advisable that duplicate cultures be established from each blood sample and that an equal number of cells be scored from each culture.

It is recognized that situations will arise in which it will be virtually impossible to initiate cultures immediately after blood sampling. In those instances, the blood samples should be stored at 4 °C and cultures should be initiated as soon as is practically possible. Storage time in excess of 5-7 days will probably result in poor growth.

In any study of this type, extreme caution must be taken to eliminate possible observer bias and subjectivity in the analysis of the slides. Perhaps the best method of preventing this situation is to randomize and code control and study group blood samples, and culture both simultaneously. In addition, it is also advisable to treat several cultures with a known clastogen to serve as a positive control. A further step that should be taken is to randomize, and code, all of the slides.

Because most chemicals are S-dependent and, therefore, are expected to be inefficient at producing structural aberrations in the circulating lymphocytes, the cytogenetic analysis of circulating lymphocytes is anticipated to underestimate greatly, if it even detects, the true effect a clastogenic agent will have on normally proliferating cell systems. Hence it can be anticipated that a small effect observed in the lymphocytes of exposed individuals may represent a minimum frequency estimate in relation to the effect that may occur in bone marrow, or spermatogonial cells. In order to avoid a gross underestimation of the potential true hazard posed by S-dependent chemicals, whose mode of action is not known, it seems advisable in those instances when exposure to only one or two known substances occurs, to perform an in vitro experiment to define the effect, and perhaps the mechanism of action, of the substance in pure culture. This experiment can be done prior to, or concurrent with, the population study. It should be carried out in a fashion that will, however, provide maximal information on mode of action and dose-response characteristics. Such experimental procedures are outlined in the EPA GENE-TOX Guidelines.

Finally, the possibility of chemically-induced mitotic delay in a sensitive subpopulation of cells might reasonably be considered in the design of a study. A precaution that can be taken to guard against this eventuality is to perform fixation at two or more time intervals. Of course this may increase significantly the work involved, but mitotic delay is of the same concern in vivo, as it is in vitro.

III. ANALYSIS AND CLASSIFICATION OF CHROMOSOMAL STRUCTURAL CHANGES

In the literature there are numerous classifications of structural chromosomal aberrations. We do not suggest that the classification system presented here is the only possible one, nor that it is necessarily the one that should be adopted. This system is a composite of those published earlier by Evans [6] and Savage [7], and is one which the panel members readily agreed was clear and accurate.

Induced chromosomal aberrations are divisible into two main classes: chromosome-type and chromatid-type. In general, chromosome-type aberrations involve both chromatids of a chromosome, and chromatid-type involve only one of the two chromatids (one exception is the isochromatid deletion which is discussed later). Also, in general, chromosome-type aberrations are induced in the G_1 stage of the cell cycle (i.e., prior to replication) while chromatid-type aberrations are produced during the S or G_2 stages (i.e., during or after replication). A few agents can induce aberrations in all stages of the cell cycle: chromosome-type in G_1 and chromatid-type in S and G_2. Examples of these chemical and physical agents are x rays, bleomycin, cytosine arabinoside, streptonigrin and 8-ethoxycaffeine. The vast majority of agents almost exclusively induce chromatid-type aberrations, even in cells treated in G_1. The fact that chromatid-type aberrations are induced from treatment of G_1 cells suggests that much of the DNA damage is not repaired before DNA replication, and that, as a result of replication of damaged DNA, chromatid-type aberrations are formed. This implies that the majority of agents to which humans might be environmentally or occupationally exposed will induce chromatid-type aberrations — an important point to be discussed again below.

Peripheral lymphocytes are noncycling cells and are in G_1, or to distinguish them from cycling cells, in G_0. Damage from most agents to the DNA of these cells will be repaired, albeit slowly, in G_0, or during the rather long G_1 of the 1st cell cycle, once the cells are stimulated to enter the cell cycle by a mitogen such as phytohemagglutinin. This low rate of repair makes the probability of producing an aberration low. (The repair of DNA damage by agents capable of producing aberrations in G_0 or G_1 appears to be more rapid.) The relatively long time interval between exposure in G_0 to the S phase in tissue culture means that a large proportion of the DNA damage will be repaired before the cell reaches the S phase. Thus the probability of chromatid-type aberrations being induced as the result of replication of damaged DNA is reduced. Since population exposure levels are likely to be low, there will be only a small probability of our observing chromosomal aberrations from the exposure of G_0 peripheral lymphocytes.

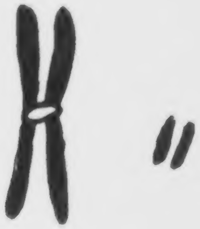
Those few agents that can induce aberrations in all stages of the cell cycle can induce aberrations in G_0 peripheral lymphocytes, and these will be observed as chromosome-type aberrations at mitosis. Since a significant proportion of human lymphocytes are long-lived, the cytogenetic effects of a dose accumulated from chronic exposures may be measureable.

The aberrations more likely to be observed will be those induced in lymphocyte precursor cells, which are cycling cells. Again, the aberrations induced by the majority of chemicals will be of the chromatid-type, but will not be observed as such at the first in culture mitosis. They will be seen as derived chromosome-type aberrations. This is due to the fact that there will be one or more cell divisions between induction of an aberration in a precursor cell and its observation in stimulated peripheral lymphocytes. Chromatid-type aberrations are essentially converted into chromosome-type aberrations as a consequence of cell division. The types of derived aberrations will be discussed later. As a result of cell killing caused by the loss of acentric fragments in mitosis, and of the mechanical interference with cell division caused by exchange aberrations, there is a significant probability that induced aberrations will be lost before the cell population is analyzed. This loss of aberrant cells will decrease the sensitivity of the assay, since the observed frequency will be less than the induced frequency. At low exposures, and consequently low aberration frequencies, this is of particular importance. Similar factors will influence the observed aberration yield from exposure to agents that induce appreciable aberration frequencies in all stages of the cell cycle. In the precursor cell, the chromatid-type aberrations induced in S and G_2 will either be lost at subsequent mitoses or will give rise to derived chromosome-type aberrations. The majority of chromosome-type aberrations (with the exception of reciprocal translocations and inversions) will be lost from the analyzable cell population, since the loss of acentric fragments or the mechanical interference with division by rings and dicentrics will often result, again, in cell lethality. Thus, for these potent clastogens also, the contribution to the total aberrations observed at the 1st mitotic division in culture, from induction of aberrations in precursor cells, will be small.

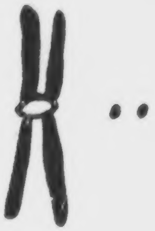
The following is a classification of the induced aberration types and the derived types observed at metaphase.

A. Chromosome-type Aberrations

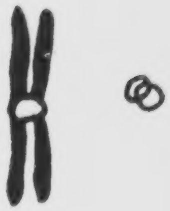
1. Terminal deletion



2. Interstitial deletions



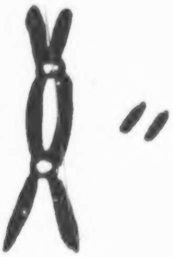
Minute



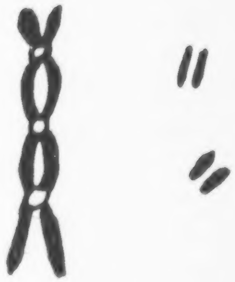
Acentric ring

The small interstitial deletions appearing as paired dots are classified as "minutes." The larger interstitial deletions in which there is a clear space in the center of the ring are classified as acentric rings. The distribution is not particularly clear-cut, and in general is merely an indication of the different sizes of interstitial deletions. The significance is not clear.

3. Asymmetric interchanges (usually dicentrics)



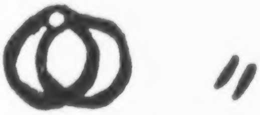
Dicentric



Tricentric

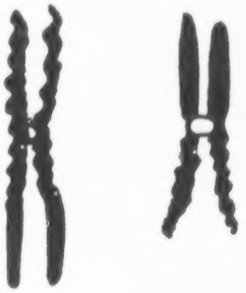
It is customary to assume that each dicentric, analyzed at the 1st mitosis after treatment, is accompanied by one acentric fragment. Thus, a cell containing one dicentric and two acentric fragments would be classified as one dicentric plus its accompanying fragment and one terminal deletion. Similarly, a tricentric is assumed to have two accompanying acentric fragments.

4. Asymmetric intrachange (ring)



As above, a ring is assumed to be accompanied by one acentric fragment.

5. Symmetric interchange (reciprocal translocation)



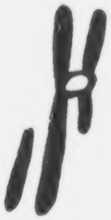
It is particularly difficult to observe this type of aberration, since the exchange often results in chromosomes that are only slightly different from the normal karyotype. It is possible to band the chromosomes, but even then the efficiency of scoring symmetric interchanges is not greatly improved. It is recommended that obvious symmetric interchanges be recorded with the proviso that their frequency is far from accurate.

B. Chromatid-type Aberrations

1. Achromatic lesions ("gaps")

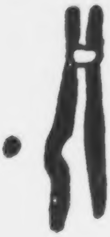
This chromosomal abnormality has been the subject of much discussion, and also confusion regarding its significance and classification. When chromosomes are analyzed at the 1st mitosis after treatment, nonstaining or very lightly stained regions are observed. These achromatic regions or "gaps" can be either in one chromatid or at identical positions on both sister chromatids. It is generally agreed that they should be recorded, but in a separate category from deletions, and should not be included in measures of aberrations per cell since their significance is unclear. It is often difficult to distinguish between achromatic lesions and true deletions. Two criteria are usually employed. First, if there is a distinct displacement of the chromatid fragment(s) distal to the lesion, the abnormality is classified as a deletion. Second, if there is no displacement, but the nonstaining region is wider than the width of a chromatid, again the abnormality is scored as a deletion. This latter definition is more difficult to interpret, and is clearly only a working definition.

2. Chromatid terminal deletions

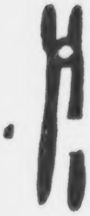


As discussed above, a displacement of the chromatid fragment, or a nonstaining region with a width greater than that of a chromatid, constitutes a chromatid terminal deletion.

3. Interstitial deletion (minute)

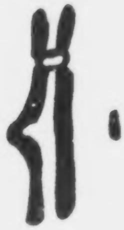


or



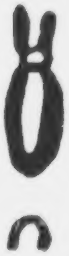
(minute + deletion)

or



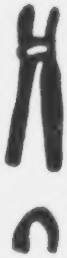
It should be noted that the deleted fragment often can be separated from the deleted chromosome, and, since it is small, will not be analyzed.

4. Isochromatid deletions



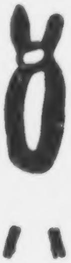
sister union

or



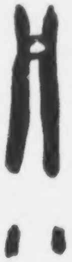
non-union proximal

or



non-union distal

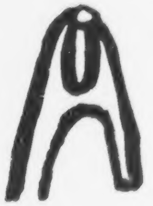
or



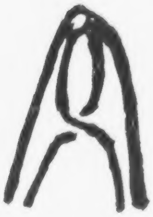
non-union proximal & distal

The acentric fragment is frequently not associated with the deleted chromosome. This can be the most ambiguous of the chromatid-type aberrations since non-union distal will lead to an acentric fragment that is indistinguishable from a chromosome-type terminal deletion. The presence of sister union would confirm an isochromatid deletion, since chromosome-type terminal deletions do not have associated sister union. However, in mammalian cells sister union is uncommon. Also the presence of clearly recognizable chromatid-type aberrations in the same cell as a double fragment would indicate that the double fragment is an isochromatid deletion. In the absence of any confirmatory information, a double acentric fragment should be classified with chromosome-type terminal deletions.

5. Interarm intrachanges



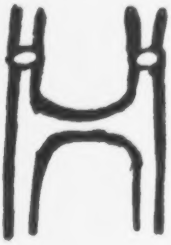
asymmetric



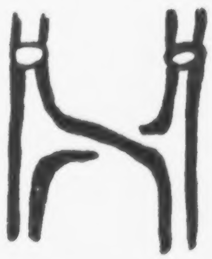
symmetric

There are in fact two forms of both the asymmetric and symmetric intrachanges, but for metaphase analysis only one of each is distinguishable. The symmetric form can be observed since chromatid pairing is maintained.

6. Interarm interchanges (chromatid interchanges)



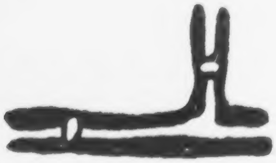
asymmetric



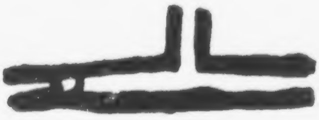
symmetric

These aberrations are the chromatid-type equivalent of the dicentric and reciprocal translocations. However, chromatid pairing allows the symmetric type to be analyzed efficiently, in contrast to the chromosome-type reciprocal translocation.

7. Triradials



or



This aberration can be considered as the interaction between an isochromatid break and a chromatid break to give a 3-armed configuration. There are several possible resulting configurations, but by far the most common are those indicated above.

There are also many additional categories of complex chromatid interchanges, but these occur infrequently, and, to avoid confusion, will not be dealt with here (see Savage [7]).

C. Other Chromosome Abnormalities

1. Shattering

In shattered cells the chromosomes seem to have broken into many small pieces. All the chromosomes can be affected, but usually only a small number are involved. Shattered cells should be recorded separately and not be included in the total number of cells analyzed.

2. Pulverization

This phenomenon is similar to shattering, but usually the whole cell is affected. The chromosome fragments are thin and unwound. There is similarity between such a cell and the chromosomes of an S-phase cell when fused with a mitotic cell (premature chromosome condensation). Pulverized cells should be recorded separately, and not be included in the total cells analyzed.

3. Polyploidy

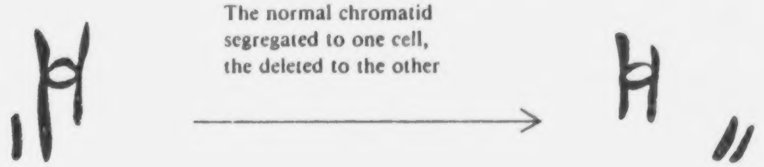
Polyploid cells are occasionally observed. Such cells are recorded separately, and are not included in the total cells analyzed. Aberrations observed in polyploid cells should not be included in the overall aberration frequencies.

D. Secondary or Derived Aberrations

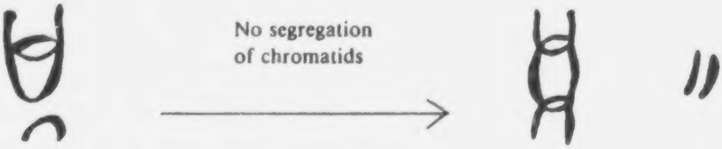
As mentioned in the introduction, the majority of induced aberrations observed at the 1st mitosis in culture will have been derived, as the result of cell division, from chromatid-type aberrations produced in stem cells. The following list shows the most common derived aberrations and the possible primary source. It should be noted that the probability of an induced aberration leading to a derived one is not high, especially when more than one cell division occurs between treatment and observation. Further, the primary source given is not the only possible one, but perhaps the most likely.

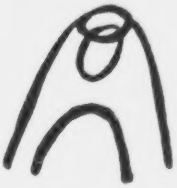
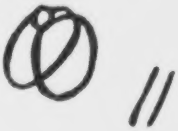
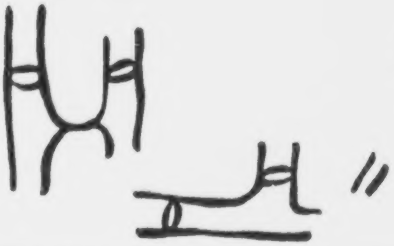
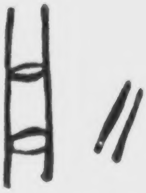
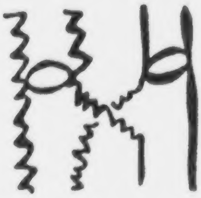
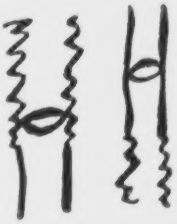
Chromatid-type Primary Aberration	Chromosome-type Secondary or Derived Aberration
Chromatid terminal deletion Isochromatid deletion Incomplete interchange Incomplete intrachange	Terminal deletion

Note: Following two divisions the acentric fragment usually will be lost, leading to cell death or a deleted chromosome without a fragment. This latter often will be detected only by banding.


The normal chromatid
segregated to one cell,
the deleted to the other

Interstitial deletion	Interstitial deletion often only detectable by banding
Isochromatid deletion (only when proximal union)	"Mirror-image" dicentric


No segregation
of chromatids

Chromatid-type Primary Aberration		Chromosome-type Secondary or Derived Aberration
Intrachange 	The normal chromatid segregates to one cell, the abnormal to the other →	Centric ring 
Asymmetric interchange Dicentric triradial 	One possible segregation gives one cell with normal chromatids, the other with the dicentric →	Dicentric (excluding "mirror-image" dicentrics) 
Symmetric interchange 	One possible segregation gives the translocated chromatids in the same daughter cell →	Symmetric interchange (reciprocal translocation) 

An important consideration that was mentioned earlier is perhaps best re-emphasized here in the context of derived aberrations. Cells should be analyzed at the 1st metaphase after culture initiation. This is discussed in the section on techniques. Failure to do this can lead to an underestimate of aberration frequency, and to more aberrations being included as derived chromosome-type, since chromatid-type aberrations arising spontaneously during culture will appear as chromosome-type if analyzed at the 2nd culture metaphase.

Recording of Aberrations and Analysis of Cells

All aberration types described above should be recorded separately on a score sheet. For each treatment, the results should be reported as the frequency of each aberration type per cell, and the frequency of aberrant cells for each aberration type, and not as total breaks per cell. For a statistical analysis, it is often appropriate to combine the frequencies of different aberration types: for example, chromatid-type deletions, chromosome-type deletions, chromatid-type exchanges, and chromosome-type exchanges.

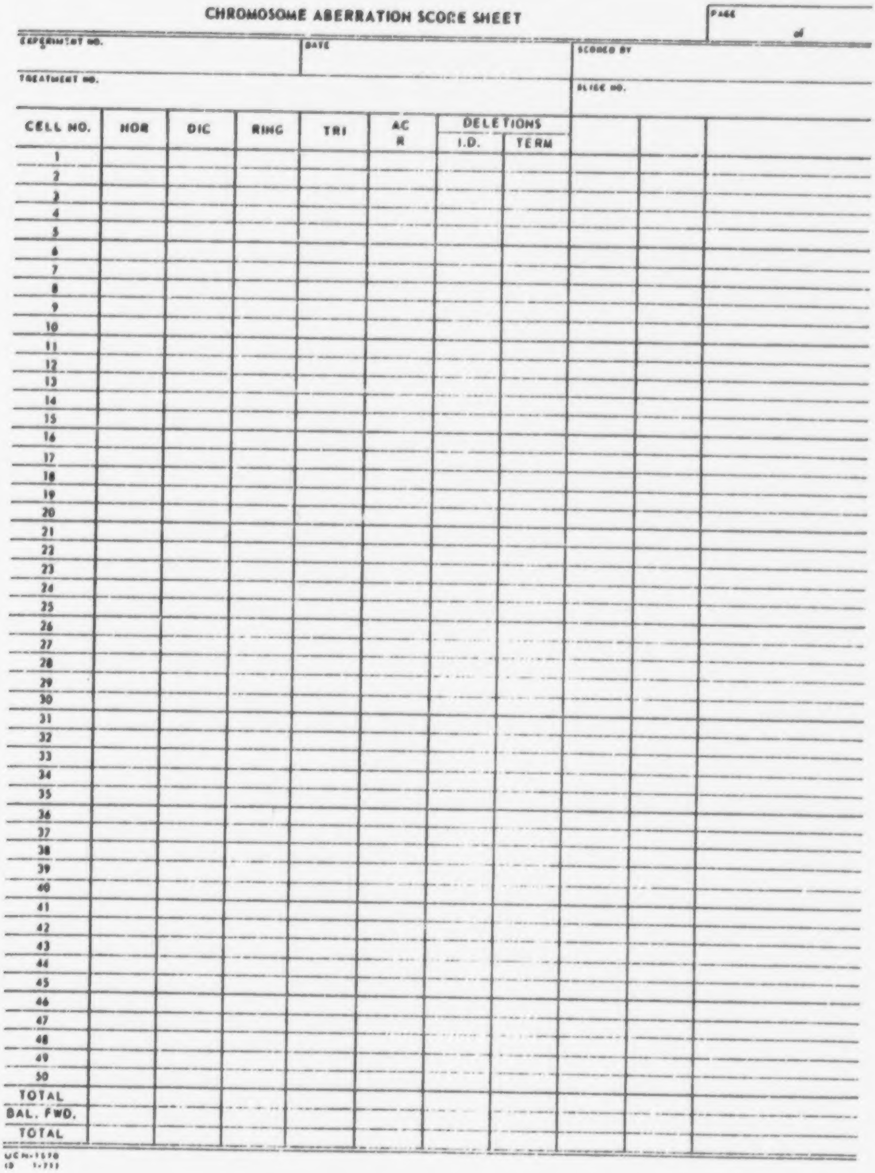
One important additional point concerns the recording of cells containing multiple aberrations. If at all possible, such cells should be analyzed and not simply recorded as "multiple aberrations," or abnormal cells, since this latter class causes potentially valuable information to be lost. If a cell cannot be analyzed, it should be recorded as containing too many aberrations to be classified, and not included in the total cells analyzed.

This leads to some comments on which cells should be analyzed. The acceptable method is to scan cells at low magnification. A cell which appears analyzable at low magnification, should be scored under oil immersion. The only reasons that a cell should be rejected from analysis once observed at high magnification are: 1) if it contains so many aberrations that it cannot be completely analyzed, when it is recorded as too many aberrations to classify (as above); 2) if it contains less centromeres than the chosen cut-off point — some individuals score only cells with 46 centromeres, others use 46 ± 2 as their criterion, where either is acceptable, provided that consistency is maintained; and 3) when the individual chromosomes cannot be seen clearly because of considerable overlapping or because nonchromosomal material (e.g., dirt or stain crystals) obscures chromosomes.

The number of cells to be analyzed from any particular individual or group will be variable, and is discussed below under "Analysis of Data."

The slides should be analyzed without prior knowledge of the group to which they belong, i.e., a blind study should always be done. This is particularly important in a population monitoring study. It is usual for the analysis to be shared between several individuals or between two or more laboratories. The study design should be such that possible bias introduced by an individual or laboratory will not influence the overall result. It is further suggested that an aberration observed or suspected by one individual be confirmed or negated by one or more other individuals. It is also highly desirable that a photographic record be kept of all aberrant or suspect cells.

Sometimes additional treatment of the samples has been advocated. It has been suggested that only banded preparations be analyzed. It is recommended here that normally this not be done, since it is very time consuming and, to date, it does not appear that any significant increase in detectable aberrations has been



CHROMOSOME ABERRATION SCORE SHEET

PAGE of

EXPERIMENT NO.

DATE

SCORED BY

TREATMENT NO.

SLIDE NO.

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observed. It can be seen from the aberration types induced that only derived terminal deletions, where the acentric fragment is lost, and reciprocal translocations can be better detected by banding. The increased sensitivity for detection of these classes obtained by using banded samples does not appear to warrant the large additional effort.

We also suggest that a banded karyotype be produced for each individual included in a study. The reason for this is to detect a congenital chromosome abnormality which could affect the analysis. This is not essential but is recommended so that at some later date the analyzer of the cells is not confronted with the information that he had analyzed a translocation heterozygote or other chromosomally abnormal individual without noting it.

This is an overview of the classification and analysis of chromosome structural changes. It cannot cover every possible eventuality, but it attempts to cover the more likely ones.

IV. ANALYSIS OF DATA ON STRUCTURAL ABERRATIONS

In analyzing counts of structural aberration frequencies, the simplest analyses of data on exposed and control groups lead to standard statistical procedures for the comparison of binomial distributions. However, real environmental exposures often may not be representable in such simple terms. The researcher should be prepared to consider any of a number of more sophisticated analytic approaches which are currently available, or to derive new statistical models to represent the particular situation encountered, if standard procedures are inappropriate.

The specification of sample sizes in terms of either numbers of persons or numbers of cells examined is also difficult, since it requires some prior knowledge or supposition of the results to be obtained. Such information will not generally be readily available in most instances of suspected environmental exposures. Some rough estimates and general guidelines are discussed.

A. Analysis

The analysis of collected data will be an attempt to ascertain whether there are indications of prior exposure to either ionizing radiation, chemical mutagens, or both. This is done by trying to rule out chance occurrences as an explanation of any differences observed in the frequency of chromosome aberrations. The raw data on counts and types of aberrations seen should be presented in as much detail as is practical. It should include the numbers of aberrations seen per cell as well as the number of cells with aberrations, for each of the types of aberrations scored. Ideally, the data summary should include sufficient specification of detail so that others may attempt their own analyses of the data, if desired.

Generally speaking, scored chromosome breaks and rearrangements in cultured peripheral leukocytes have been used much more widely as indicators of direct radiation exposure than of low-level chemical exposure. Upon exposure to whole or partial body radiation, the evidence indicates that circulating lymphocytes or their precursors are subject to some fixed probability of receiving "hits" to their chromosomal material. These "hits" will subsequently be observed as structural changes in metaphase preparations, as discussed above. The probability of such "hits" will be monotonically dependent on the radiation dose (though the aberration induction rate also may be affected by dose rate and other potential confounding variables).

For those cells which are affected, a sufficient environmental insult can result in one or more breaks in the complete chromosomal complement. It is generally assumed that all parts of the chromosomal complement are equally likely to be the site of a break. These assumptions imply that the number of breaks in the total chromosomal material of a given cell should be distributed as a Poisson variate with some average occurrence rate, λ , which is a direct function of the degree of intensity of the exposure. That is, the probability of a number, x, of breaks occurring as a result of an exposure which generates an average of λ breaks per cell should be given by:

$$P(x) = \frac{e^{-\lambda}\lambda^{x}}{x!}; \quad x = 0, 1, 2, \ldots$$

At very low levels of radiation exposure, it is unusual to have more than one break per cell, and extremely rare to have more than two. The probability of no breaks is given by:

$$P(0) = e^{-\lambda},$$

and, if the average rate of aberrations is on the order of 1 or 2 per thousand cells, then $\lambda = 0.001$, and $P(0) = 0.999$. That is, almost all cells will be unaffected, and only about one in a thousand cells, on the average, will display one or more breaks. In fact, the probability of 2 or more breaks per cell will be on the order of 10^{-7}. Even for a value of $\lambda = 0.01$, the probability of 2 or more breaks will be on the order of 10^{-4}.

Because of these numerical realities with low-dose radiation exposures, individual cells are classified most commonly as either affected or not affected with respect to whatever type of aberration being scored. Multiple aberrations in a single cell are occasionally seen, but this is not common in low-level radiation exposures, and is very rare in control data. When it does occur, it probably indicates that the data on the individual involved should be examined more closely for the possibility of heavier exposure or other potential confounding influences.

Published data on normal population controls are not extensive. It is usual to exclude persons who report recent or unusual radiation or chemical exposures or recent viral infections from the control group. The control data that are available do not suggest an effect of age or sex on structural aberration frequency. However, because of the current paucity of data on potential confounding variables, good histories should be obtained from all study subjects concerning their past work, medical, and residential experience.

For cytogenetic analysis, a number of metaphase cells will be chosen for scoring, presumably randomly selected from among those eligible for scoring by reason of their adequacy of spread and chromosome count. The number of cells scored will differ from investigation to investigation, depending upon the specific questions that are posed, the heterogeneity of the populations studied, and the degree of precision required for the circumstances at hand. Sample size considerations will be discussed below.

For an individual, therefore, the data will consist of the number of aberrant cells out of some fixed number scored. With a fixed sample size of cells, if the probability of finding an aberration of a given type in a cell is constant from cell to cell within the sample, and if the results on different cells within the sample are independent of one another, then the count of the number of cells with aberrations should follow a binomial distribution with parameters n = the sample size and p = the true proportion of aberrant cells in the blood of the individual sampled. Data are available (unpublished) which indicate that these assumptions are reasonable, at least for data from controls and those with low-level radiation exposures. For each individual scored, the fraction of cells which are aberrant is an estimate of the true proportion of aberrant cells within that person's population of circulating lymphocytes and that observed fraction will be subject to binomial variability. The task at hand will be to compare the collection of observed proportions, and to determine whether they are elevated, on the average, in the exposed sample relative to the controls.

The most powerful analysis (in a statistical sense) results when one can pool the data across individuals within exposure groups and compare the frequencies of aberrant cells between the exposure groups using the total number of cells examined in each group as the denominator for the overall estimate of aberration frequency. In order to do this, one must be assured that the amount of variability in the proportion of aberrant cells observed from person to person does not exceed the amount that would be expected in repeated sampling from a *single* binomial distribution. The usual chi-squared goodness-of-fit test for categoric data is not likely to be very sensitive to variation in proportion affected; Fisher's variance test is recommended as a much more sensitive alternative (see, for example, Cochran, 1954, p 417 [8]).

If the data are compatible with a single aberration frequency within each exposure group, then one of the statistical techniques for comparing two single binomial frequencies would be appropriate. The data can be displayed in a 2x2 table as follows:

	Exposed	Not Exposed	Total
Abberrant cells	a	b	a + b
Nonaberrant cells	c	d	c + d
Total cells	a + c	b + d	N
Proportion aberrant	$\frac{a}{a+c}$	$\frac{b}{b+d}$	

We want to determine whether the observed proportion in the exposed group, $\frac{a}{a+c}$, is sufficiently greater than the observed proportion in the control group, $\frac{b}{b+d}$, and that the difference is not likely to be due to chance. (Of course, if $\frac{a}{a+c} < \frac{b}{b+d}$ then no further statistical treatment is necessary, unless there are other factors to be taken into consideration.)

Again, the Cochran article lays out rules of thumb for appropriate testing. In general, if the smallest expected value is less than about 5, Fisher's exact test should be used (see, for example, Siegel, p. 96 [9]). Otherwise, a normal approximation is usually adequate. The square root of the usual (uncorrected) chi-squared statistic is probably the simplest way to calculate the appropriate normal statistic.

That is,

$$z = \sqrt{\frac{(ad-bc)^2N}{(a+c)(b+d)(a+b)(c+d)}}$$

is approximately distributed as a standard normal variate; and if it turns out to be greater than 1.645, for example, then the proportion of aberrant cells in the exposed group would be declared greater at the 5% level of statistical significance than that in the control group.

Of course, the foregoing discussion applies to only the simplest situation. If there is heterogeneity within either or both study groups, or if other potential confounding factors exist, or are thought to be exerting an influence, then more complex analytic procedures will have to be considered. Sometimes, the foregoing simple situation can be approximated by stratifying the study groups into relatively homogeneous subsets, subsequently combining the significance testing results by some appropriate technique (again see the Cochran article [8], or Fleiss, Chapter 9 [10]). In situations in which other variables must be accounted for (e.g., age, sex, time of exposure, actual exposure estimate, etc.), a variety of techniques are available, including logistic regression (e.g., Cox,1970 [11]), multiple linear regression (e.g., Evans, et al [6]), and many others (e.g., Bishop, et al [12]).

If, particularly for chemical exposures, it is thought that an individual's population of lymphocytes is actually a mixture of exposed and unexposed cells, then some estimate of the parameters of the mixed population would be a desirable first step in the analysis (see reference [13]). If there is reason to believe that the binomial parameter varies from person to person in some specific stochastic manner, then it would be appropriate to consider modeling the results with one of the so-called "contagious" or compound distributions (e.g., Johnson & Kotz [14]).

If a difference is found between the putatively exposed group and the controls, either in terms of frequencies of cells with aberrations or frequencies of aberrations per cell, then further analysis will be required to investigate whether a "dose-response" relationship can be established, and whether interactions exist with other potential confounding variables.

B. Sample Size Considerations

The size of available samples will have an important effect on the conclusions drawn, particularly if the results appear to be negative, for in that case we will want to be able to rule out with some degree of assurance the possibility of having missed an effect. The ability of a statistical test to rule out a difference of a given size is called the "power" of the test.

In order to estimate necessary sample sizes, one must specify the significance level at which the test will be done, the size of the difference (in proportion of affected cells, in this case) which would be of biologic importance or interest, and the desired degree of assurance to rule out the existence of a difference as large as, or larger than, that specified. Figure 1 shows the results of some calculations of the approximate sample sizes needed for comparing two proportions under a variety of circumstances. The bottom axis shows various potential values for the true aberration frequencies in the control group (e.g., p_1). Then, assuming a test will be done at a 5% level of significance, the graph indicates the approximate sample sizes required for each group in order to rule out an aberration frequency in the exposed group ($p_2 = kp_1$), and to do so with a probability of 0.80.

For example, if an observed frequency of about 1% were expected in the control group (as might be the case if all structural aberrations were being considered), and it was desirable to detect a threefold increase in the exposed group ($k = 3$), then about 600 cells in each group would be needed to give 80% assurance that such a difference would be detectable at a 5% testing level. On the other hand, if the aberration frequency were expected to be only 1/1,000 in the control group (as might be the case with dicentrics and rings), then about 3,500 cells in each group would be needed in order to detect a fourfold increase in the exposed group, with 80% assurance. (To guarantee 90% power, the sample sizes determined from this graph should be multiplied by about 1.5.)

The foregoing estimates were calculated on the assumption that the total sample within both the exposed and control groups could be pooled for analysis. It was also assumed that the two groups would be equal in size. Since either or both of these assumptions may be inappropriate in a particular case, the estimates given should be taken as indicating only the order of magnitude of the effort which may be necessary in a particular instance.

The design of a study, and, in particular the selection of controls, should be given careful consideration, taking into account the circumstances to which the investigators are responding. In some situations, the resources available would be utilized best by having fewer controls than exposed. In others, it may be necessary to have multiple controls per case in order to assure adequate power. The extremes, one cell per person, or the entire sample from one person, obviously are to be avoided; but the optimal intermediate point cannot be specified in general and must be determined on pragmatic grounds in response to the particular circumstances being confronted. The only general principle which should be followed is to have concurrent controls adequate to rule out laboratory artifacts or time factors as explanations for any differences observed.

Size (n) of Each Sample Necessary to Detect
a k-Fold Increase in the Exposed Group Over
the Control Group for Various Control Frequencies

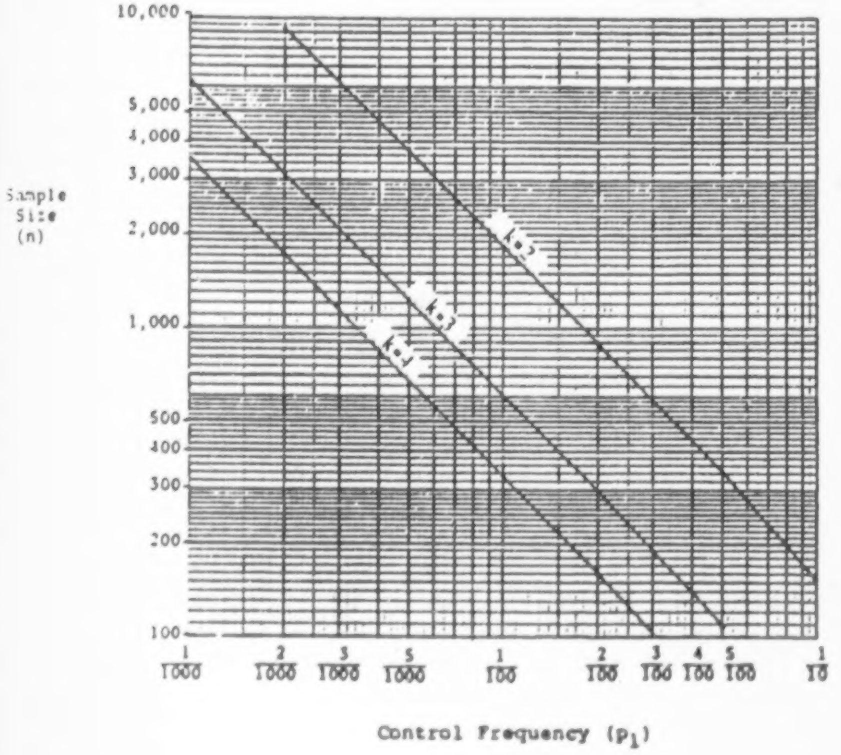

Fig. 1.

The quantification of structural aberrations (or sister chromatid exchanges) presents an opportunity which is not common in research and which potentially can be exploited to mitigate the absence of the prior knowledge required for precise sample size determination. If the total number of cells to be examined is allocated toward fewer cells per person and more persons in the initial investigation, this should leave a potential bank of cells for each individual which remain unexamined, but which are available to increase the sample size in either exposed or control group, if that should be found desirable after an initial analysis. Furthermore, such additional data acquisition can be done with a minimal expenditure of effort and cost.

One other general problem is the question of the degree of specificity with which an *individual's* aberration frequency can be estimated. This, of course, will be the question of interest once an elevated aberration frequency has been shown for the exposed group as a whole. In general, it requires considerable data to estimate a small frequency within narrow limits. For example, if 100 cells from an individual had been examined and *no* aberrations had been found, one could only estimate his true aberration frequency as less than 4% at the 95% level of confidence. With 200 normal cells, the upper 95% confidence limit on the true proportion is 2%, and it is not until about 375 normal cells are examined that 95% assurance can be given that the true rate in the individual examined is under 1%. This would represent an enormous investment of technician time, and in many circumstances, would not be considered justifiable from a cost viewpoint.

V. METHODS AND PROCEDURES FOR THE ANALYSIS OF SCEs IN HUMAN LYMPHOCYTES

As indicated above, the type of lesion induced in the genetic material will influence the cytogenetic alteration produced to a large extent. SCEs are more pronounced following exposure to those agents that form covalent adducts to DNA or chromatin, or otherwise induce distortion of chromatin structure relative to those agents that directly break the DNA backbone. Thus, although there may be an overlap between those agents that produce both aberrations and SCEs, both cytogenetic end points should be included in any population study. As an illustration, a population study involving known radiation exposure might be expected to yield an increase in aberrations. The use of SCEs in the same individuals would be useful to determine potential exposure from confounding factors such as medication or smoking. This section will address those points pertinent to the methodology for the evaluation of SCEs in human lymphocytes.

A. Blood Collection and Cell Culture

Whole blood obtained by venipuncture should be mixed with heparin, acid citrate, or another appropriate anticoagulant. This mixture can be stored between 4-37 °C for several days prior to culture, but this is not desirable. Lymphocyte loss, reduction of mitogenic response, and possible selective loss of SCE-forming lesions may occur during storage. Therefore, cultures should be established within 24 hrs, if possible. A white blood count and smear for a differential should be made from the blood sample just prior to establishing the culture. These end points serve as indicators of abnormally high or low lymphocyte counts which may influence the frequency of SCEs.

For the analysis of SCEs, it is imperative that each laboratory establish a standard-lymphocyte culture procedure. The detailed procedure may vary among laboratories but all should adhere to some basic requirements. Bromodeoxyuridine (BrdUrd) can induce SCEs in human lymphocytes, and with the culture conditions employed for most SCE studies, this induction is dependent on the ratio of the BrdUrd concentration to the number of lymphocytes per culture, as shown by Carrano et al [15]. This ratio should be kept as invariant as possible. In order to insure this, each laboratory should establish its own SCE induction curve for BrdUrd using a minimum of four individuals. In general, the frequency of SCEs will rise sharply at low ratios of BrdUrd to lymphocytes but may stabilize at higher ratios. Thus, each laboratory should select a ratio which is optimal for its conditions and maintain this as standard procedure. Selection of a ratio which is in the stable region of the curve may minimize the SCE variation produced by fluctuations among individuals in lymphocyte counts or in mitogenic responsiveness. Another factor to consider is the minimization of intra- and inter-individual variation. Each laboratory should select those conditions which minimize these sources of variation.

Selection of culture medium, serum type and concentration, whole blood vs purified lymphocyte cultures, and duration of culture are somewhat flexible and should be based upon historical experience. It is recommended that the serum lot be pretested for its potential to support lymphocyte growth and for its reproducibility of baseline SCE frequencies in at least two longitudinal control individuals (i.e., the same individuals repeatedly sampled with time). If feasible, the same serum lot should be used for each population study. The duration of the lymphocyte culture should be one that yields a high percentage of 2nd division mitoses. Each of these variables should be standardized prior to any study. Exposures of the cultures to other than yellow or red light should be avoided.

A minimum of two cultures should be established for each individual, which can be harvested at either the same or different times. The primary purpose of the replicate cultures is to insure a backup in case of culture or other technical failure. They may be used also to estimate culture-to-culture variation, if this is warranted. When a large number of cultures are processed, the inclusion of samples from longitudinal controls is important. Further, it is good practice for each laboratory to sample its longitudinal controls periodically to insure that the procedures remain satisfactory. Occasional addition of a positive control (e.g., an individual on chemotherapy or in vitro treatment with mitomycin-C) is useful to insure that the culture conditions allow for cellular responsiveness in terms of SCEs.

B. Preparation and Scoring

At the appropriate culture time, the cells can be arrested in mitosis by a 2-4 hr treatment with Colcemid, colchicine, or other suitable spindle inhibitor. Care must be taken to insure that the arresting agent does not induce SCEs, particularly if the longer treatment times are used. Any of the routine metaphase cell preparation methods are satisfactory. Several slides should be prepared from each culture, and the remaining cell pellet stored for future reference in absolute methanol:glacial acetic acid (3:1) at 4°C or lower. The slides can be stained for analysis by fluorescence alone [16], fluorescence plus Giemsa [17], or a reproducible variation thereof. The sister chromatids should be well differentiated, i.e., there should be a high contrast in staining intensity between the two chromatids, and there should be a minimum of staining debris. Once a staining procedure has been standardized and adopted, it should be used throughout the course of the study.

Only well-differentiated metaphases should be accepted for scoring. The slides must be coded as in the breakage studies, so that data collection is done in a blind fashion. The coded slides can be scanned under low magnification (100-200X) and selected for scoring on the basis of well-stained sister chromatids and an adequate number of chromosomes in the metaphase. Assuming that large chromosomes are not preferentially lost or gained during cell preparation and that SCEs are evenly distributed along the length of the chromosome, cells with other than 46 chromosomes can be analyzed. It has been established in at least two laboratories (unpublished data) that cells with 41 or more chromosomes are acceptable for SCE analysis, i.e., there appears to be no significant difference in the mean SCE frequency for an individual if cells with 46 vs 41 or more chromosomes are accepted. The experienced cytogeneticist can distinguish a cell with this number of chromosomes under low magnification. SCEs are counted for each chromosome in the cell with the final results tabulated as the number of SCEs per chromosome, or as the number of SCEs per diploid cell (i.e., the number of SCEs per chromosome X 46). The precise number of cells to be scored per individual will ultimately depend upon the desired relative sensitivity for the study.

Figure 2 illustrates the method for scoring. The exchange of stain must be reciprocal, i.e., a darkly stained region on one chromatid must be accompanied by a light staining region in the reciprocal region of the sister chromatid. It is not necessary that the entire chromatid width be involved in an exchange (Fig. 2H). Exchanges of stain occurring at the centromere (Fig. 2F) should be included as an SCE unless there is an obvious twist of the chromatids.

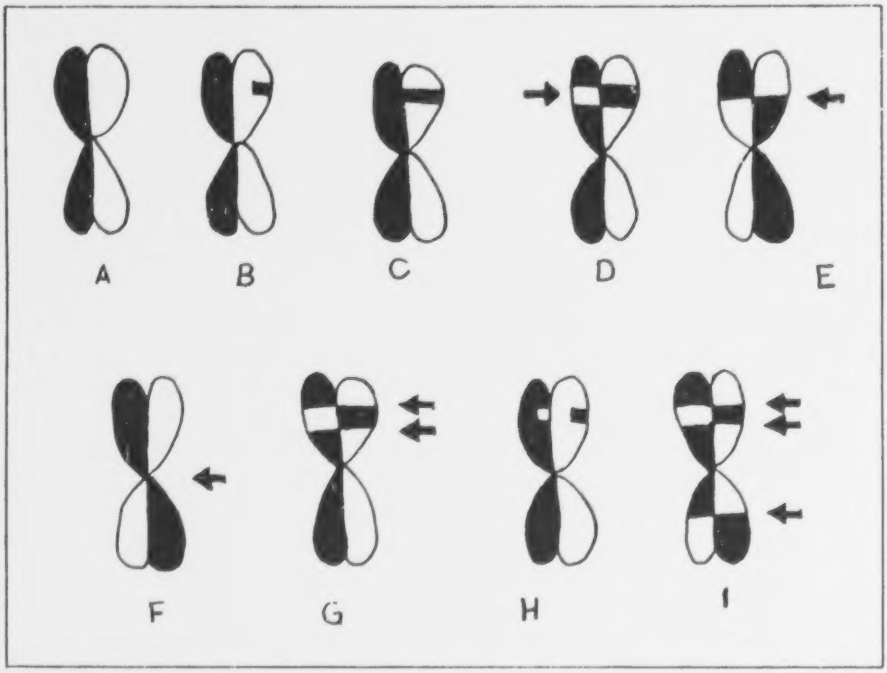

Fig. 2. Schematic representation of chromosomes differentiated for SCE analysis. The dark region represents unifilar substitution with BrdUrd, and the light region represents bifilar substitution. A) No SCE: complete differentiation of the sister chromatids. B) No SCE: incomplete differentiation or staining artifact. C) No SCE: staining artifact or possibly replication through less than 2 complete DNA synthetic periods in the presence of BrdUrd. This pattern should not be observed in metaphases from short-term lymphocyte cultures, if BrdUrd is added at the time of mitogen stimulation. D) No SCE: probably replication through more than 2 DNA synthetic periods in the presence of BrdUrd. E-I) SCEs are indicated by arrows. E) Single SCE. F) Single SCE at the centromere. G) Two SCEs. H) Two SCEs: reciprocal exchanges not covering the complete width of the chromatid I) Three SCEs.

C. Statistical Analysis of SCE Data

There are at least 2 statistical questions which arise as the result of the analysis of 2 or more cohorts. First, are there significant differences between cohorts; and second, does a specific individual in the test cohort differ significantly from the control cohort? As stated previously, the use of concurrent controls is mandatory and these should also be compared to historical controls. In order to optimize the statistical analysis, knowledge of the distribution of SCEs both within and among individuals would be useful. At the present time, such information is preliminary but suggests that no formal statistical distribution can adequately describe the within individual SCE measurements. The problem appears to be the existence within individuals of outlying cells, i.e., lymphocytes with very high SCE frequencies. The data for one population study do suggest, however, that the distribution of individual SCE means, at least in a control cohort, are normally distributed (Carrano and Moore, unpublished results). Obviously information on the distributions for other populations is needed before any statistical tests can be recommended as generally applicable. Until such time, each investigator should determine, for his own study population, the degree of homogeneity of the individuals within a cohort and then apply the appropriate statistical tests. Any analysis of data should consider the effects of confounding variables.

VI. SIGNIFICANCE OF ABERRATIONS AND SCEs

We are all exposed more or less constantly to agents which are capable of damaging the genetic material of our somatic or germ cells, and there are many mutagen-exposed human populations in which increased chromosomal breakage has been documented. These include the Hiroshima and Nagaski atomic-bomb survivors and numerous other radiation-exposed populations. Chemical exposures in human populations have been much less well studied genetically and cytogenetically, and most of our assumptions about these effects of chemical mutagens are derived from the radiation studies. Even when the exposure has been to a known mutagen (as ionizing radiation) at high doses (as in Hiroshima and Nagasaki) with considerable residual chromosomal damage, no increases in spontaneous abortion or birth defects have been found to date, and no heritable chromosomal abnormalities have been demonstrated. Our assumption must be not that such effects do not occur, but simply that their magnitude is very low, indeed.

Further, in human populations, no associations have been definitively drawn between those individuals with induced breakage and the subsequent development of cancer. This is true even in Japan, where thousands of persons have been shown to have chromosomal aberrations of radiation origin. Stable translocations and other aberrations have been shown to be useful in dosimetry assessments at high doses. Thus, although the presence of the chromosome breakage in the lymphocytes of mutagen-exposed persons is at times a marker of exposure, it is not by definition a harbinger of cancer for the carrier individual.

The two-step cancer hypothesis of Knudson et al., [18], however, suggests that not all individuals may be at equal risk of developing cancer. According to the hypothesized first step, some individuals are born with an inherited predisposition to cancer. These include: 1) persons who carry the autosomal dominant genes involved in disorders like retinoblastoma, Wilms tumor, and some forms of breast cancer; 2) persons who are affected with the autosomal recessive chromosome breakage syndromes, like Fanconi anemia, Bloom syndrome, and ataxia telangiectasia; and 3) persons with chromosomal anomalies of the karyotype, such as trisomy 21; or the recently described translocation family with a t(3:8) in which there exists an 87% risk of renal cancer by age 59 in carriers [19]. The second step of the hypothesis involves a somatic mutation of environmental origin (radiation, chemical, viral).

Further, we have a nonhomogeneous response to environmental mutagens in our species, with some repair-deficient persons (XP, AT), some immunologically underresponsive persons, etc., at increased risk of carcinogenesis for genetic reasons. These individuals may have an increased tendency to respond with enhanced chromosome breakage to environmental exposures. The identification and management of such genetically at-risk persons is a problem of major proportions for those concerned with the health effects of mutagens.

So, while we deem chromosomal aberrations as undesirable, as we consider induced mutations generally to be deleterious, we are unable to use them to predict specific human health effects in the individual. But, considerable evidence has accumulated that, at least for physical mutagens, chromosomal aberration frequencies may be reliable as biologic dosimeters for some individuals, but even more useful for subsets of a population exposed to various doses of a mutagen. This utilization of chromosomal aberrations as a biologic dosimeter has been shown to be valuable in radiation exposures, for both immediate studies of acutely exposed persons and many years after the acute high-dose exposure. Certain types of aberrations have proven particularly sensitive as indicators of dose, and these tend to be the aberrations which are rarely seen spontaneously, i.e., under "control" conditions. Such aberrations include, for example, dicentrics, rings, and balanced translocations.

This raises the question of the fate of various types of aberrations in cell division. Ample evidence exists in numerous systems that many sites of damage are repaired, though the mechanisms of this repair process in human cells remain to be elucidated. Nonetheless, the simple types of aberrations, such as chromatid breaks and deletions, are generally useful in acute exposures, much less valuable long after exposure, and of intermediate utility in chronic, low-level exposures. Some of the more complex aberrations may be eliminated during cell division (mitotic or meiotic), because of difficulties at the time of spindle formation or at other times; but they may nonetheless be more useful in biologic dosimetry than their simpler counterparts because they are so rarely seen normally, and because they are at times able to survive at least a modest number of in vivo or in vitro cell divisions. The long-lived nature of some lymphocyte subpopulations enables us, then, to analyze for rings, dicentrics, and translocations with dose questions in mind. In particular, the balanced translocations may be of help in such studies, since such translocations are often stable more or less indefinitely.

Another phenomenon requires comment, that of the amplification of subpopulations of aberration-bearing lymphocytes. The increase in frequency of cells which all bear the same aberration has been repeatedly documented in radiation-exposed populations. Reasonably this has been referred to as in vivo clone formation. Again, we cannot draw conclusions regarding the health of the clone formers, though we assume this phenomenon to be undesirable for the patients.

To this point, we have been concerned with somatic cell chromosomal aberrations. Agents which induce this kind of damage reasonably may be regarded as capable of doing so in germ cell chromosomes as well. However, even in the Hiroshima and Nagasaki populations, with all of their induced somatic cell aberrations, no consistent heritable aberrations (translocations, inversions, or the like) have been demonstrated to have been induced and transmitted to the F_1 generation [20]. Our concern for such F_1 effects must continue, but realistically the magnitude of the clastogenic effort is unlikely to be very great, based on what we have not seen to date, and somatic cell clastogenesis must be our primary concern.

The phenomenon of sister chromatid exchange has, we now believe, a different biologic, and therefore a different clinical significance from that of chromosomal aberration formation. Based on in vitro studies of recent years, largely with Chinese hamster cells, SCEs appear to correlate better with specific locus mutations than with clastogenic effects. Further, SCEs are more sensitive to chemical mutagens than to physical mutagens, as has been shown in studies with CHO cells exposed to a wide range of known chemical and physical mutagens including exposures to radiomimetic compounds, such as bleomycin. Assessment of in vivo SCEs is in its early stages, with only a few such human studies having been conducted, as discussed above. For these reasons, the Panel suggests: 1) that the usefulness of SCE studies in human populations is likely to prove to be a sensitive measure of exposure to chemical mutagens; but, 2) that such studies are in their formative, or developmental, stages and must be interpreted with caution. However, the Panel does recommend that such studies be done in instances of exposure of humans, acutely or chronically, to known or putative mutagens, with careful attention being paid to the inclusion of adequate controls. The same concerns described above for chromosomal aberrations, in terms of the 2-hit cancer hypothesis, would be applicable for SCE induction, and SCEs show promise of being an excellent reflection of point mutation in vivo as in vitro. Evidence exists for SCE formation in relation to repair and repair deficiency, but the mechanism(s) of SCE formation are not clearly understood at this time.

Throughout the discussion in this Chapter we have dealt essentially with aberration and SCE studies in the peripheral blood lymphocytes of exposed humans. Other tissues, such as bone marrow and skin, or even gonial cells, at times may be useful to study. But, for routine screening on a population level, there now seems little reason to go beyond the peripheral blood cell studies, ideally done under well-controlled conditions, with specific questions to be answered. We recognize full well, however, that such studies may be done at times when the agents and the doses are ill defined. Under those conditions our suggestion is that suitable control groups be simultaneously studied, with all microscope work being done blindly, i.e., without investigator knowledge of the putative exposure status of the person whose cells are being examined.

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REPORT OF PANEL II
GUIDELINES FOR REPRODUCTIVE STUDIES IN EXPOSED HUMAN POPULATIONS

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SECTION I

INTRODUCTION

In considering reproductive failure in relation to environmental exposures, we have defined reproductive failure (or, more properly, dysfunction), as an increase in one or more of a list of undesirable end points. There are shortcomings to this approach: many end points are not independent, and an increase in one deleterious end point, may be the result of decrease in another, e.g., neonatal mortality rates may increase as the late fetal death rate decreases and very low birthweight babies are born alive. Still, we content ourselves for now with the simplest possible definition.

For the exposure, we have hardly even attempted a definition. We simply imply that an adverse exposure is some physical or chemical contaminant of the physical environment (air, water, food or workplace). Usually, we visualize a concentration in one or several geographic areas, or in the workplace.

Section II provides the list of end points. Then, Sections III, IV, V, VI and VII describe principles and methods of study, and decision making. Section III discusses general statistical problems, especially relevant to investigations of the effects of environmental exposures. In Section IV, we set out the immediate steps usually taken by an investigation, alerted by a potential problem area. This account, based on the actual procedures adopted by the Center for Disease Control, emphasizes the uses and limitations of easily accessible sources of information. A description of some of these is included. The transition from what we term Level I studies, based on easily accessible data, to more prolonged and costly investigations (Level II studies) of a wider range of end points seems to be a point of decision making, and in Section V we discuss guidelines for movement from Level I to Levels II and III. Section VI is a guide to the design of epidemiologic studies, emphasizing considerations that may lead to a choice of case control or cohort approaches, the selection of controls, and the control of bias. Section VII gives a brief review of long-term follow-up studies (Level III) that might be conducted.

In Section VIII, nine end points were selected for further attention to provide an outline of current methods of study. Section IX is a critical review of publications dealing with environmental exposures and reproductive failure.

In this introductory section, five general themes will be introduced:

1. The distinction between investigating a *cluster* of adverse effects, as opposed to a supposedly hazardous *exposure*.
2. The level of the investigation, in terms of cost, complexity, and the time required for completion.
3. Some special problems of specifying exposure, whether by measures based on individuals or by measures based on populations.
4. The basis for decision making, and the considerations, scientific and otherwise, that enter into the process.
5. The interrelationships between findings based on reproductive failure, somatic cell changes, and in vitro tests of mutagenicity.

1) A Cluster of Effects Compared to a Hazardous Exposure

Residents of Alsea, Oregon, and later of Cape May, New Jersey, reported to journalists, public health officials and others, that spontaneous abortion (miscarriage) was occurring "too often," among pregnancies in their localities. Cape May residents also mentioned an excess of births with hydrocephalus. Although in both these examples, the residents themselves linked the apparent cluster of effects to an environmental exposure (pesticide), still the stimulus for concern was a suspected excess of reproductive problems. We will call this type of situation a "Cluster."

By contrast, residents of the Three Mile Island area, and those in the neighborhood of toxic wastes, provide an example of an exposure-provoked situation, an "Exposure."

Thus in Cluster situations, the investigation is provoked by some real or apparent excess in the occurrence of an adverse reproductive end point; in Exposure situations, an episode involving known or suspected toxic substances has occurred, and adverse effects are anticipated. Cluster type situations and Exposure type situations present unique problems to the investigator, and each may call for a different public health response.

With Cluster situations, the relevant outcome is (presumably) known or suspected. The first task is to determine the population at risk and to decide whether the *observed* concentration in space and time of the outcome (which constitutes the "cluster") represents a meaningful departure from the *expected* distribution in space and time. The second task, especially when there is no clue as to the cause, is to know which questions to ask in search of the cause.

With Exposure situations, it may not be obvious what outcomes are salient for evaluating whether effects have occurred. Health studies, lacking a targeted end point, become expensive and inefficient. Although in general, the methodologic problems are similar in the two situations, some arise particularly in Cluster situations, and in some Exposure situations, as will be discussed later.

2) The Level of the Investigation

We define three levels of investigation: *Level I* is based on easily accessible, routine records. The investigation will usually be carried out with speed and economy; it will seldom involve case examinations or special questionnaires (although clinical confirmation of supposed 'cases' is not excluded). By definition, end points that are not routinely examined (for instance, sperm studies) or recorded (spontaneous abortions) will not be studied.

Level II includes short-term purposeful epidemiologic studies, either case control or cohort. Investigations of a wide range of end points (including somatic cells) can be entertained.

Level III. This category is reserved for investigations the results of which cannot become known until months, or years have elapsed, following the initial stimulus that provoked the investigation.

The level does not describe the statistics or the design. Thus, in Level I, the investigator will usually be content to compare rates of incidence or prevalence in the suspicious population with expected rates generated from some other population, but the use of case control or cohort design is not excluded. Level II studies may be either cohort studies (in the sense that an exposed group is followed) or case-control studies (in the sense that an affected group is explored in terms of previous experience), but in either case, the end points of interest have already been recognized. In Level III studies, there can be no immediate answer, but the flagging of certain individuals or groups may in time enable the inquiry to be completed. This type of activity may have particular importance in discovering associations between somatic cell findings and subsequent morbidity and mortality. Models for computerizing and storing data for such prospective studies are described later.

Moving an investigation from one level to the next involves making decisions; some of the considerations to be weighed are outlined under (4) below, and discussed more fully in Section V.

3) Defining Exposure

Whereas many useful end points of reproductive failure are reasonably well defined, the same is seldom true of the exposure. As discussed later, the exposure is often poorly defined.

a) There may be multiple exposures. Typically in both the occupational environment (e.g., the chemical industry), and in the vicinity of toxic waste (Love Canal), the population "at risk" has been exposed to a range of chemicals and a range of levels of exposure for each. We do not know if one, or several of these, or even an interaction between two or more, is likely to be responsible for the effects.

b) Measurement of the environmental exposure: If the exposure happened some time prior to the investigation, it may be difficult (or impossible) to discover either the nature or dose of exposure. Ambient pollutants are often transient; pathways and doses of exposures in the soil or water may vary over time in the same location. Dosage levels may vary from place to place, even within one area or plant. Exposures which are not detectable by current measurement techniques may not be without effects. Breakdown products may be the responsible agent and may be difficult to measure.

c) Measurement of exposure to the individual: Ideally, one wants to measure levels of exposure to the individual; this might be obtained through badges worn during all periods of exposure. Often, noninvasive biochemical tests are not available. Other effects of exposure (e.g., chloracne on exposure to dioxin) might serve to indicate an exposed group; however, those health effects are unlikely to serve as surrogate measures of exposures for studies of reproduction.

d) Exposed populations: In many instances, there are no measurements of exposure to the individual, either because effects are being studied long after a transient exposure occurred, or because there are not techniques to measure exposure in the individual and/or the immediate (e.g., home) environment. The investigator resorts to announcing that a population (e.g., town, a factory) is "exposed." This approach may result in misclassification of the study population; some "exposed" individuals are not truly exposed. Misclassification will have the effect of reducing the chance that a study will detect an association between exposure and an end point.

Note that studies limited to comparisons between communities will also suffer if not only the exposures but also the outcomes are treated as group phenomena. This is because individuals within one community may differ considerably in characteristics known to effect the end point (for instance, smoking and birthweight). If these characteristics are not considered, they may lead to false inference in comparing the communities.

4) The Basis of Decision Making

The decision to initiate an investigation, to pursue it more deeply, to pronounce a relationship as present or absent, and to act accordingly, are matters that depend only partly on the scientific community. Nevertheless, these practical considerations must concern us. The statistical section has, therefore, stressed the limits of inference and the areas of uncertainty which will generally remain at the end of most investigations. We have preferred the perspective of an anxious community, employer or public health agency, rather than the perspective of an experimentalist. The experimentalist is often ultra-conservative. He requires the outcome of an experiment to provide strong reason to reject the null hypothesis (setting his chances usually at 1 in 20 that this will occur by chance), while he is less concerned at falsely accepting the null hypothesis (setting his chances typically at

1 in 5 that this will occur by chance). The community is not neutral: interest is focused on a possible excess of adverse outcomes and there is extreme concern that a relationship that is really present not be missed.

Bearing in mind these concerns, we have created tables in Section III that attempt to express the meaning, as well as the limits of inference, that can be derived from the statistical tests. We conclude that the most accessible statement to convey the limits of inference is often effect size. Thus, "the investigation is able to rule out as extremely unlikely, a sixfold rise in such and such outcome . . ." may be an intelligible and useful statement.

We envision that a decision-making body would include nonscientists, and would be concerned not only with probabilities and risks of adverse outcomes, but also with the nature of the outcome under discussion; its physical, emotional and financial cost to parent, offspring, and community (the "*burden*"). In the discussions that follow, the choice among end points for study is discussed mainly from the points of view of accessibility for Level I, frequency, reliability of diagnosis, and usefulness as a sensitive end point. At the point of decision making, burden takes on crucial importance. Thus, a modest effect size (say doubling) involving mortality or severe retardation, may outweigh a larger effect (fourfold) involving miscarriage. These issues are not considered further: they go beyond our present brief.

5) The Interrelationships Between Reproductive Failure, Evidence of Somatic Cell Effects, and In Vitro Tests of Mutagenicity

This topic is introduced here mainly to emphasize its importance, and the lack of knowledge in the area. Should decisions on the existence of effects, as judged by reproductive failure, consider at the same time undesirable effects of a substance on somatic cells of exposed individuals or in in vitro tests? What does evidence for the one indicate about the other? A preliminary approach would be to select a number of agents, and compare their known or suspected properties in terms of each of the three areas of this report. A table of this kind could serve as a basis for useful suggestions as to the need, in Level II and III investigations, for studying a spectrum of epidemiologic and biologic effects.

SECTION II

INDICES OF REPRODUCTIVE FAILURE

We set out below a list of potentially useful end points, that might be studied in connection with reproductive failure or dysfunction. Although this list indicates the wide range of outcomes which could be used, it is not all-inclusive. It is by no means clear how these end points relate one to another, nor whether changes in one will always be accompanied by changes in another.

The list more or less follows a biologic chronology. More detailed discussions on nine selected end points (shown with an * on the table) are given in Section VIII. In Section III, information relating to expected frequencies and sample sizes are shown for some end points. A dagger (†) indicates the suitability of the end point for Level I studies.

List of End Points:

1. Sexual dysfunction: libido; potency
2. *Sperm abnormalities: number; motility; shape
3. *Sub-fecundity: abnormal gonads, ducts and external genitalia; abnormal pubertal development; infertility of male or female origin; amenorrhea; anovulatory cycles; delay in conception
4. Illness during pregnancy and parturition: toxemia; hemorrhage
5. *Early fetal loss (to 28 weeks): morphology; karyotypes
6. †Late fetal loss (after 28 weeks) }
7. Intrapartum death } perinatal death*
8. †Death in first week }
9. *†Decreased birthweight
10. †Change in gestational age at delivery: prematurity; postmaturity
11. *†Altered sex ratio: in fetal deaths; at birth
12. †Multiple births
13. *Birth defects: major; minor; mutations
14. *†Chromosomal abnormalities: in fetal deaths; at amniocentesis; in perinatal deaths; in livebirths
15. †Infant death
16. †Childhood morbidity
17. *†Childhood malignancies
18. Age at menopause

Interrelations Between End Points

It is difficult to anticipate the spectrum of outcomes associated with putative environmental hazards for which there are no previous observations to provide at least tentative working hypotheses. Agents are known which induce *specific effects* upon developmental abnormality only in one organic system, but without marked effect upon other adverse outcomes. Yet such agents may also have effects that would be difficult to predict on the basis of known physiologic effect (e.g., the reported association of congenital heart defects in infants born to women exposed to maternal female sex hormone during pregnancy). Many embryotoxins and other reproductive hazards may be expected to result in multiple adverse reproductive outcomes. Agents resulting directly in disordered embryogenesis sufficient to impair fetal viability (and thus increase embryonic and fetal deaths) are also likely to be associated with increased frequency of structural defects in the fetuses which do survive gestation. Variations in outcome within an exposed population exposed to such known teratogens as rubella, thalidomide, etc., may result from differences in individual maternal or fetal susceptibility to the agent, to variation in dose of exposure, and to differences in timing of exposure during gestation.

Similarly, agents or biologic factors with putative effects upon the karyotype are likely to result also in increased fetal death and in increased rates of malformations associated with various cytogenetic syndromes in those that survive. But again, most agents identified to date which result in fetal deaths and birth defects do so without any observed effect upon the chromosomes, at least in terms of trisomies, monosomies, etc. While all generalizations in this area are subject to some qualifications, for the most part a suspect agent to which there is preconceptional exposure is likely to manifest through *mutagenic* mechanisms and no *consistent* pattern of *structural defects* is likely to be observed. Thus, an observed cluster of specific congenital abnormality, if attributable to an environmental insult, is likely to have resulted from one acting during gestation, not from preconceptional exposure.

There is also the theoretic possibility that an environmental agent associated with embryonic and fetal death or congenital malformation may act by postponing the time of embryonic or fetal death (or by inhibiting it altogether); in such a case, it would not be increasing the actual incidence of such events, but rather enhancing the likelihood of their recognition.

SECTION III

STATISTICAL ISSUES

Some methodologic problems are commonly encountered in investigations of exposures and reproductive outcomes because:

1) exposed populations tend to be small in number
2) the exposure is often poorly defined
3) one is often interested in rare events such as specific malformations
4) publicity relating to the episode under study may produce or accentuate reporting bias.

In this section we discuss these problems and how they affect statistical aspects of a well-designed reproductive study: statistical power, misclassification and bias. While we concentrate on power and strategies that may assist the investigator in increasing power, many of our recommendations could be motivated from other perspectives such as the maximization of the validity and utility of the study results. Our statistical discussion is purposefully kept at an intuitive level; it certainly is no substitute for an active statistical consultant or more formal statistical texts, such as Fleiss [1]. In the discussion that follows we assume (unless otherwise stated) that the data can be represented by a fourfold table, with dichotomous end point and dichotomous exposure, in which the measure of association is usually the odds ratio.

Statistical Power

Power, defined broadly, is the ability to detect and accurately estimate changes in frequency (or in mean value). An investigator should always consider power at the onset of the study, because it may influence the strategy, and it provides him with a realistic assessment of the meaning of the results.

Power depends on:

1. The number of people likely to have been exposed, and the number of people available who are unexposed.
2. The variability of the reproductive event. For discrete events, this will depend on the usual (expected) rate of the reproductive event in the unexposed population.
3. The significance level that will be accepted as confirmation of an association, e.g., the probability that the observed change has occurred by chance is not more than 1 in 20, $p < 0.05$, or 1 in 100, $p < 0.01$. This assumes a specific probability model, and there may be more than one feasible model.
4. The magnitude of the expected effect. It is immediately obvious that if exposure multiplies the frequency of occurrence of an outcome by 10, the exposure will be easier to detect than if it multiplies it only by a factor of 2 or 3.
5. The design of the study, and the statistical techniques used for analysis.

Statistical Considerations Influencing Design

1. Selecting the end point:

The higher the frequency of an outcome in the unexposed population, the greater the power. Frequencies of selected end points are given in Table 1. The effects of variations in frequency on statistical power are discussed later, and are illustrated in Table 2.

The observed frequency of an outcome can be increased by the researcher in various ways. For instance, he can intensify case finding. Thus, if he is comparing spontaneous abortion in two communities, the more complete the ascertainment, the higher the frequency, and the greater the power (of course, the case finding must be intensified equally, in both communities).

The frequency of an outcome depends, in part, on the definition of the sample in which the outcome is measured. For instance, chromosomal and morphologic abnormalities of the conceptus are 100 times more frequent among spontaneous abortions than among livebirths. Similarly, the frequency of azoospermia is likely to be higher among couples attending an infertility clinic than among the general population.

Table 1
Frequency of Selected Reproductive End Points

Event	Frequency per 100	Unit
Azoospermia	1	Men
Birthweight*<2500 g	7	Livebirths
Failure to conceive after one year of unprotected intercourse	10-15	Couples
Spontaneous abortion 8-28 weeks of gestation	10-20	Pregnancies or Women
Chromosomal anomaly among spontaneously aborted conceptions 8-28 weeks	30-40	Spontaneous abortions
Chromosomal anomalies among amniocentesis specimens to unselected women over 35 years	2	Amniocentesis specimens
Stillbirth	2-4	Stillbirths + livebirths
Birth defects	2-3	Livebirths
Chromosomal anomalies in livebirths	0.2	Livebirths
Neural tube defects	0.01-1.00	Livebirths + stillbirths
Severe mental retardation	0.4	Children to age 15 years

* More usefully analyzed as a continuous variable, as discussed in the text of Section III.

Table 2

Frequency per 100	Sample Size of Comparison Groups	Increase in Frequency Detectable with 95% Power*
3	50	7.7-fold increase
	100	5.3-fold increase
	250	3.3-fold increase
	300	3.2-fold increase
7	50	4.6-fold increase
	100	3.4-fold increase
	250	2.3-fold increase
	300	2.3-fold increase
15	50	3.0-fold increase
	100	2.4-fold increase
	250	1.8-fold increase
	300	1.7-fold increase

* a = .10, two-tailed, or a = .05, one-tailed.

2. Sample sizes:

The number of "cases" of the "exposed" is often absolutely limited by circumstances beyond the investigator. However, sample size can be somewhat increased, even in these situations, by doubling or trebling the numbers of "controls" or "unexposed." In practice, there is seldom much benefit to increasing the comparison individuals more than threefold.

3. Significance level:

Significance levels of .01 and .05 are commonly used in experimental situations since such levels indicate that the observed results would occur rarely because of sampling fluctuations alone. In some situations, however, one might consider a false rejection of the null hypothesis in 1 out of 10 tries to be sufficiently rare than a result obtained with a = .10 would be acceptable, or at least warrant further investigation. This is not necessarily a compromise in rigor; it should be a result of comparing the undesirability of falsely rejecting the null hypothesis with that of missing an effect that is really there.

Besides the choice of the significance level for a specific test, the probability of falsely concluding that an association is present at least once is affected by the number of associations that is tested within a single analysis. Consider the situation in which the exposure is poorly understood, and anticipated effects are vaguely described as "adverse reproductive outcomes." Then the investigator, undertaking what is called "data exploration," searches for effects of an exposure on a) birthweight, b) neural tube defects, and c) congenital heart defects, and sets a 1 in 20 chance in every case of finding an effect caused by sampling fluctuations alone. Since there is a 5% chance of making this so-called "type 1 error" on every test, the cumulative probability of making at least one type 1 error can be substantially larger than 5% when multiple hypotheses are tested. For a set of k multiple hypotheses that fall together as a "family," it is possible to insure that the family-wise type 1 error rate is no larger than "a" by conducting the individual hypothesis tests at a significance level of (a/k) [2].

From the point of view of power, it is clear that adjusting the significance level for multiple hypotheses within a "family" makes it more likely that a true effect might be missed. To avoid this loss of power for specific a priori hypotheses these are usually considered to be one-element families, i.e., the significance level for that hypothesis is not adjusted for any other tests. Since it is fitting that specific a priori hypotheses be treated differently from nonspecific tests of association, the investigator should be sure to specify carefully all of the outcomes that can be anticipated at the onset of the study.

4. Magnitude of the effect:

One strategy to maximize the observed strength of an association is to control as far as possible all sources of variation in exposure or end point that are irrelevant to their association. We consider this approach: a) for the exposure; and b) for the reproductive end point.

a) Refining the history of exposure.

For reasons already alluded to, a major problem in investigations of reproduction and exposure is how to specify correctly the exposure status or exposure dose of an individual. We assume here that misclassification, or error in assigning exposure status, affects cases and controls equally. Misclassification can make a "real" association appear less, because it introduces noise, or error, into the analysis.

Statistical power will therefore be enhanced by precise data on individual exposure to reduce misclassification. When dealing with reproductive end points, precision may involve ascertaining whether exposure is maternal or paternal, the timing with regard to conception, and the route of exposure. In some situations (e.g., for workplace exposure at sites where industrial hygiene information exists) such data may be readily available. In others, at hazardous waste sites, for example, in which the composition of the fill, and the routes and timing of migration are unknown, data will need to be carefully and comprehensively developed. Thus, there is already evidence from earlier studies (Seveso, Love Canal) that the application of hypothetic exposure models, albeit sensible, has led to extensive misclassification of exposure at these sites, thus generally reducing the power of analyses to detect health effects.

Biochemical measures of exposed and unexposed individuals are to be preferred, but sound environmental and interview data regarding the residents' locations and habits relating to exposure may produce dose groupings that permit a sensitive test for effects (and may also serve as a check on response bias). It is unusual for such data to be available for Level I analysis.

In cluster situations, a plausible preliminary hypothesis as to the nature of the exposure must exist before there is any hope of invoking this strategy for increasing the power of the analysis.

Consideration of exposure in relation to the relevant parent and timing vis à vis conception may also allow analysis of a more specific hypothesis, although it should be recognized that such tests are necessarily based on smaller numbers (e.g., exposure to the mother only; exposure prior to conception only).

b) Refining the end point.

The more the reproductive event can be refined to conform with an anticipated, biologically coherent outcome, the greater the power. End points should, therefore, be limited to the items relevant to the exposure and should exclude irrelevant items. For example, if "birth defects" is the supposed untoward effect of an exposure, one may decide to exclude from the count affected offspring with single locus gene disorders born to carrier parents.

On the other hand, there may be reasons to pool malformations, thereby increasing the rate. If it is thought that the exposure acts upon more than one end point, power can be increased with this strategy. If it is thought that the exposure acts upon only one end point, power can be reduced with this strategy.

5. Statistical design and analysis:

Four examples are given below of special design and analytic techniques that may enhance power:

In these examples we are no longer restricting discussion to the fourfold table and the odds ratio.

a) Use of continuous distribution rather than discrete categories.

When the end point can be studied as a continuous distribution (like birthweight, for instance), it is usually better to analyze it in that form. Selecting one or two cutoff points and producing two or three categories is wasteful of data, and may reduce power.

b) Use of conceptions rather than women as the unit of study.

One way to enhance power when dealing with reproductive events is to use conceptions rather than women as the unit of study, since each woman may have several conceptions. However, precautions must be taken to ensure a valid statistical analysis in the presence of correlated observations. The conceptions of the same woman will have common characteristics special to that woman. Therefore, undue weight may be given to the characteristics of women with several conceptions. A spurious finding may occur if women have numerous adverse outcomes (e.g., repeat spontaneous abortion) for reasons unrelated to exposure, and these women are more numerous in the exposed population.

We may assess the extent of within-woman correlation, much as in the analysis of repeated measures, and if the extent of correlation is only slight, the investigator stands to gain much in the way of power by using conceptions as the unit in multiple pregnancy populations, with suitable control for the within-subject correlation. Such control can be obtained by statistical models allowing for sequential dependence of pregnancy outcomes.

c) Stratification, and controlling of confounding variables.

For some end points, like birthweight, several characteristics of women are known to have an important effect, e.g., prepregnant weight and cigarette smoking. These characteristics may or may not be independent of the exposure. If they are not independent, then they will need to be controlled to avoid confounding, which can lead to false inferences about the existence and magnitude of the effect. Even if they are independent, then there are situations (for instance, cohort studies) in which by controlling one may usefully remove unwanted sources of variation.

Therefore, it is prudent design and analysis strategy to subdivide the entire population into strata relevant for the purpose of controlling for confounding variables. There are, then, several methods available for combining the evidence in each stratum regarding the existence, strength, and consistency of an association across strata (e.g., the Mantel-Haenszel procedure for several fourfold tables, or Fisher's method for combining evidence from independent experiments).

While controlling for confounding variables reduces bias in the estimates, and may increase power in some cases as a result of reduction of bias, it is often the case that inclusion of additional control variables in a statistical model reduces power by decreasing the degrees of freedom available in the data. This is especially likely to be the case when the sample sizes are small (N <30) and fixed. In such situations, stratification should be limited to variables for which there are strong a priori grounds for concern with respect to confounding. This consideration is negligible with large sample sizes.

d) Clustering techniques.

On occasion clusters of events arise strictly by chance. Determining when these aggregations are meaningful or merely random is particularly difficult when the apparent excess involves a rare condition. Analysis of clusters of uncommon events requires the use of specially developed statistical methods known as clustering techniques. These methods are sensitive to low-level epidemicity, whether it occurs in time or in place, or in both time and place, and their use will permit sound conclusions as to whether or not the observed excess is random.

Comparing End Points with Respect to Power

Table 2 shows, for various sample sizes, the magnitude of the effect that can be detected at the 0.1% significance level with 95% power [3]. Three levels of frequency are considered: events occurring at rates of 3%, 7%, and 15%, respectively. (Table 1 gives examples of reproductive end points which arise at these frequencies.)

Because the size of the sample studied also contributes to the power of the test, four different sample sizes are examined in relation to each of the three levels of frequency. These numbers (50, 100, 250, 300) have been chosen as reproductive studies in a small-to-moderate sized community. There might, for instance, be about 100 couples of reproductive age in the study area who, over the period of study, collectively contribute 300 pregnancies of which about 85%, or 250, may end in livebirths. Should one wish to conduct sperm studies, one might estimate, based on typical rates of participation, that perhaps 50 males among the 100 eligible will volunteer semen for analysis.

For each of these parameters (sample size and frequencies), Table 2 indicates the variation in statistical power by citing the increase in frequency that is detectable with a high level of certainty. Thus, for example, for an event whose usual rate of occurrence is 7%, one could be 95% sure with a sample of 100 of not overlooking a three-to fourfold rise in frequency; effects smaller than this — a doubling, say — may not, however, be ruled out with the same level of assurance.

A review of Table 2 shows that the power to discern modest effects increases with increasing frequency of the event under study. For a given sample size, events with a baseline frequency of 15% provide a more sensitive test than do events with a baseline rate of 3%, or even 7%.

Further, for a given level of frequency the effect size detectable increases with increasing size of the sample studied. Thus, if one considers events with a frequency of about 3% — for example, stillbirth or birth defects — one also needs to consider the definition (hence the size) of the sample in determining the statistical power of studies of these events.

It should be noted that the computations in this Table assume that the groups being compared are equal in size — that is, 50 each of 100 each, etc. However, the use of multiple comparisons, or unequal-sized groups, can improve the statistical power of a study in which the size of one of the groups is fixed, although the increase in power is not linear with each additional subject. For purposes of estimating power, the effective sample size with unequal groups is based on the harmonic mean of the groups being compared:

$$n = \frac{2n_1n_2}{n_1 + n_2}$$

Bias

It is clear that an aware and exposed population presents difficulties to the investigator well beyond the statistical issues. In Section VI we will discuss some of the strategies which may be used to control for the problem of reporting bias. However, we consider here one issue that is statistically not easily solvable. When community reports of adverse events are the spur to subsequent health studies, statistical concerns arise that the hypothesis has been suggested by the data. Usual tests of significance may be inappropriate because one has chosen to test for an effect in an area in which an effect has already been noted. Ideally, one would test the observation in another area. However, frequently the exposure circumstances may be unique or it may be necessary for other reasons to confine investigation to the area from which the original report emanated. One needs then a satisfactory way to compensate for the positive bias without introducing bias in the other direction. If the community is small, the events noted rare, and communication such that most of the current cases have already been found, then excluding the reported cluster constitutes too severe a penalty, biasing the test to a negative finding. If exposure is continuing, one could consider a prospective investigation of what would then truly be an independent sample. But with rare events, prospective study is usually not sensible. There may be no truly satisfactory solution and one may be forced to include the original cluster in the analysis, relying on scrupulous definition of the denominator and unambiguous results to estimate the probability that the increase has occurred by chance.

SECTION IV

GUIDELINES FOR LEVEL I ACTIVITIES: ACCESSIBLE SOURCES

Introduction

We define a Level I investigation as one that avails itself of easily accessible, routine records. The purpose is to be speedy, and parsimonious. A firm positive result can lead to quick preventive action, as has happened many times in the past. An inconclusive result requires careful evaluation. Since an absolute limit on Level I activity is set by the presence of accessible data, one task fufilled by this Committee was to set out the data that do exist, and to explain their strengths and weaknesses. A later task may be to formulate recommendations for extending and improving the basis of Level I type studies. In highly developed societies, these may be our most important resource.

The availability of baseline data, and its analysis, increases the chances that clusters will be discovered. Baseline data can also provide comparative data that otherwise may not be available. Birth and death records can provide baseline information for evaluation of malformation clusters, and can also provide a current survey of malformations. Unfortunately, the congenital defect items on the birth and death record are tabulated in only a few states, and appropriate analysis is seldom performed in a timely manner.

The basic methods for Level I data collection and analysis are similar for Cluster and Exposure situations. However, end points that are examined in Cluster situations may be limited to those on which suspicion has fallen (such as birth defects or miscarriages). By contrast, in Exposure situations such as Love Canal, all adverse reproductive outcomes or as many as feasible will have to be addressed.

1. Select the end points:

A first task is to select those adverse reproductive outcomes with which we can concern ourselves in Level I. These include:

1. Spontaneous abortion (if accessible through record systems)
2. Late fetal death
3. Birth rate
4. Low birthweight and/or premature gestation
5. Congenital malformation
6. Newborn screening, especially for hypothyroidism*
7. Outcome of an alpha-fetoprotein screening program, especially for neural tube defects.

Among the adverse outcomes we monitor, low birthweight and congenital malformations are favored by public health workers as indices of adversity. Valid birthweight data are routinely available on vital records; birthweight has been shown to vary with environmental exposures, as described later. Congenital malformations, if obvious, can be monitored and are perceived as carrying heavy burdens. Once the reproductive outcomes to be evaluated have been selected, the verification and data collection can begin. During this process, change due to artifacts and biased ascertainment can be identified.

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* The stored blood spot may also provide a valuable resource for additional analysis.

2. Define the population at risk:

The geographic area and population that is at risk must be well defined. This is essential to develop the denominator, and to determine if the reported events are unusual in frequency and/or severity. The area and population could be within a defined distance from an environmental hazard.

3. Verify the reports:

A) In certain situations in which increases are reported by hospitals, individual physicians or concerned citizens, one must be sure that there is not a "numerator epidemic." It is possible for high-risk pregnancies to be referred to one hospital or physician, and this may create a false increase.

B) Verify the diagnosis. In some situations there is overreporting of minor defects because of sudden awareness of a problem, or the introduction of a new technique, or a new physician moves into a hospital or community. Certain minor birth defects are particularly susceptible to overreporting.

C) Possible problems with the data collection or reporting system, such as birth and death certificates. There can be simple coding errors or changes in coding indices that create false epidemics.

4. Identify records:

During the data collection phase, there are several existing record systems to examine at the local, state, national, and international levels. When pursuing these investigations, identification of the available data sources is very important for case finding and for establishing a reliable data base to use for comparative purposes. The extent to which one pursues the case finding depends on the situation at hand. The following list reviews some of the available data sources, and their advantages and disadvantages.

A) Local or Community
  1) Hospital — Medical indexing system
        Obstetric logbook
        Nursery logbook
        Autopsy reports
        Discharge summaries
  2) Physicians
  3) Clinics — maternal and infant clinics
  4) Cytogenetic labs
  5) Occupational records

These data will be the most reliable and accurate but will require the most effort to collect.

B) State
  1) Vital records — Livebirth certificates
        Fetal death certificates
        Infant death certificates

Vital records are a good source for evaluating outcome such as low birthweight, infant mortality and birth rate. When looking at birth defects, one must be aware of serious underreporting of malformations on birth certificates. It is also possible that deaths may be underreported.

2) State birth defects monitoring programs. New York, Nebraska, Florida and Washington have such systems already. Several other states are planning them. Some of these are birth certificate systems only, while others combine hospital records and birth certificates.

3) Other state resources — Maternal and child health units in the health department
State Epidemiologist
Crippled Children's Service
Mental retardation centers
Various voluntary health associations (i.e., March of Dimes, Spina Bifida Association)

C) National

1) The National Center for Health Statistics (NCHS) publishes vital statistic data collected through states on numerous topics. This is an excellent source for determination of state, metropolitan, and national birth rates, infant mortality, and low birthweight. Further, the NCHS is a good source from which to obtain national rates of congenital malformations for comparison with state vital records. There are serious underreporting problems, so comparisons should be made only from vital records (birth certificates).

2) Center for Disease Control, Birth Defects Branch — Operates two birth defects surveillance systems — one national, the BDMP; and one local, the MACDP.

The national Birth Defects Monitoring Program (BDMP) monitors the incidence (about one-third of all births) of congenital malformations among 1 million newborn discharge summaries annually. This data source is not population-based and it is not a random sample of U.S. births; nevertheless, it represents the largest single source of uniformly collected and coded discharge data on birth defects in newborn infants. Although the BDMP functions primarily as an early warning system, it can be useful also for correlating incidence patterns, with such trends as temporal and geographic distribution of drugs, chemicals and other possible human teratogens. The BDMP is an excellent reference for regional and U.S. birth defect incidence rates among newborns from hospitals. The problem with hospital records on newborns is some underreporting of defects, since many are not diagnosed until later in life.

The Metropolitan Atlanta Congenital Defects Program (MACDP) is an intensive surveillance system using several methods to identify infants born with birth defects in the Atlanta area. Cases included in the system are all infants with abnormalities diagnosed before 1 year of age, whose mothers resided in the 5-county

Table 3
Characteristics of Two Congenital Malformation Surveillance Systems

Characteristic	MACDP	BDMP
Population	Five-county metropolitan Atlanta area. Population based on 24,000 births/year.	1200 hospitals throughout U.S. 1 million births/year.
Rapidity of reporting	Depends on defect — usually 2 months delay.	3-6 months.
Nosology	ICD-8 modified.	H-ICDA and ICD-9-CM.
Methods of ascertainment	Chart review at obstetric and pediatric referral hospitals. Cytogenetic laboratories. Vital records.	Cover sheet of neonatal discharge summary.
Identification of syndromes of associated malformations	Good. Published in surveillance report.	Poor.
Follow-up studies	Yes. Regular interviews.	Difficult. No regular interview.
Linkage of maternal and infant records	Yes.	No.
Data analysis	Baseline period: 1968 to 16 months before current period. 169 defect categories. Separate analysis for whites, blacks, and all races combined. Data analyzed each month.	Baseline period: 1970-1973. 161 defect categories. Races grouped for routine analysis. Data analyzed every 3 months.

metro-Atlanta area. The MACDP has been in operation since 1968 and is an excellent source of birth defect incidence data for comparative purposes.

Data from the two systems are published quarterly in the Congenital Malformations Surveillance Report. The characteristics of the two CDC congenital malformation surveillance programs are outlined in Table 3.

D) International

Several countries have established malformation surveillance systems. Informally, 10 of these countries share birth defect data through the International Clearinghouse for Birth Defects Monitoring.

SUMMARY

Having selected the end points, identified the population at risk, confirmed the reports of adverse outcome, and compared the rates established thereby with expectations based on existing records, the investigator may assess whether the residents in the study areas have experienced excessive adverse outcomes. He then must decide whether to proceed to Level II activities. As noted above, given a significant excess, it may be useful to pursue the investigation further. Given an inconclusive result, the decision as to whether or not to proceed will be influenced by many considerations, not all of them scientific.

SECTION V.

DECISION MAKING: MOVING FROM LEVEL I TO LEVEL II

Level I studies are limited by definition to easily accessible end points. They will lack power because they will usually be limited to poorly defined measures of exposure (for instance, to global measures for communities, as opposed to individual measures). They may also be deficient by being unable to take into account other known factors for reproductive outcomes, thereby risking confounding.

For a wide range of end points known to be sensitive to environmental exposure (spontaneous abortion; sperm abnormalities; somatic cell damage), Level I studies cannot be carried out. These end points require more time, more funds and more community involvement. We must consider the basis on which a decision might be made to include such end points and to pursue more in-depth investigations.

General Considerations

There are several steps to be considered within Level I evaluations, whether the situation is a Cluster, an Exposure, or, as is often the case, a combination of the two. The first tier of concern for evaluation of a Cluster is limited to consideration of the original observation(s). This might be followed by additional case findings through review of one or more readily accessible existing record systems. The period of observation for adverse outcomes might be expanded backward or forward in time to gain greater confidence in the legitimacy of the observations. Additional related outcomes might be considered in tandem. At this point it may be appropriate to obtain by interview additional descriptive information from members of the clusters to learn about possible exposures, firm up the time frame in which the events occurred, and generate etiologic hypotheses.

Initial observations on exposure situations may be limited to the knowledge that someone lives near a presumed hazardous site. Generally, more detailed data are necessary before proceeding, such as ambient environmental data or, better yet, measurements of specific compounds in homes or industrial settings. Samples of body fluids or tissues indicative of significant human exposure may be compelling evidence to continue or expand an evaluation.

In the end, the decision to proceed from the Level I descriptive evaluation to Level II analytic studies involves several factors. The first and perhaps most important of these considerations is, or should be, the *biologic meaning* of the descriptive data thus acquired. Is an association *plausible?* Is the level of human exposure high? Is the exposure in question to a highly toxic compound or agent? Second, statistical considerations are also important. Level I data for clusters should at least suggest an *increase of observed over expected* events. The number of cases or potential cases and/or the number of exposed or potentially exposed individuals should be large enough to be evaluated. Finally, the level of public concern has to be added to the equation. It should be noted that the final decision to proceed with additional studies is generally not a unilateral action but rather a joint move of local, state, and/or federal officials and ideally should proceed in a *timely fashion.* Level II studies will often require contact with individual subjects. In addition to satisfying the right of a community to be informed of the *possible* presence of an Exposure or Cluster, explaining the need for Level II studies to the community in advance may well alleviate the anxiety that might be generated when the Level II study actually begins, and individual participation is solicited.

Prior notification may also facilitate obtaining consent from both "cases" and "controls." Standard procedures should be elaborated for obtaining permission from both of these groups for release of their names to the investigators conducting Level II studies, and for assuring that participants will be informed of the results.

Summary of Criteria for Moving From Level I to Level II

1. A statistically significant association is observed at Level I, but there is a need to explore a broader spectrum of outcomes, and/or to specify more precisely the relationship of the outcomes to the exposure, e.g., threshold and dose-response components of the exposure.
2. The end point that stimulated the investigation, or that seems most biologically coherent with the exposure, is not accessible to Level I studies.
3. The results in Level I are inconclusive: the trend is in the expected direction, but the investigation lacks power (e.g., a sixfold effect can be shown to be unlikely, but a threefold effect is compatible with the findings). This is the most difficult situation, but perhaps the most likely.

SECTION VI

GUIDELINES FOR LEVEL II INVESTIGATIONS: EPIDEMIOLOGIC STRATEGIES

Failing a clear-cut result (either negative or positive) between exposure and Level I outcomes, the investigator may move on to further epidemiologic studies to examine an association studied in Level I in more detail, or to examine other end points. The statistical considerations of power and bias discussed in Section III now find application in the choice of study design, enabling the researcher to make maximal use of small numbers, rare events, and uncertain information sources. This will be true, whether the end point of interest is one of those listed in Section II or one described under somatic cell changes. Case-control and cohort designs each have advantages and disadvantages depending on the circumstances; in both, errors in planning and oversight in execution may reduce the power of the test or introduce bias. Experience is the best master. In this Section, principles of design are illustrated by examples from the field of reproduction, drawing attention to any special strengths or difficulties that have faced investigators.

Whether the provocation to an inquiry is based on a Cluster situation (originating in a reported excess of adverse outcomes) or an Exposure situation (originating in some supposedly hazardous exposure) does not usually influence the strategy chosen to study the relation between an exposure and reproductive functioning. One distinction (discussed in Section III) is the reported observation of an apparent increase in the frequency of a particular outcome, in the Cluster situation. A report of this kind cannot be ignored and the particular outcome noted must reasonably be included among the effects studied. One way to avoid the bias inevitably introduced is to extend the search to include other outcomes not yet noted, but reasonably expected to be associated with the reported outcome; i.e., if anencephaly is reported, investigate spina bifida; if prematurity is reported, investigate late fetal deaths. Also, since there are several examples of exposures which relate to more than one reproductive outcome (e.g., the associations of smoking with both decreased birthweight and increased fetal death, and of alcohol drinking with fetal death, decreased birthweight and congenital malformation), a comprehensive evaluation of the reproductive effects of an exposure will usually include study of a range of reproductive end points.

Two approaches may be taken in Level II (or III) studies:

In the *case-control study,* the frequency of exposure among persons who experience the disease (cases) is compared with the frequency of exposure among persons who do not experience the disease (controls).

In the *cohort study,* the frequency of disease among exposed persons is compared with the frequency of disease among unexposed persons. In a retrospective cohort study both exposure and disease have occurred before the study begins; in a prospective cohort study, the investigator follows the exposed and unexposed cohorts in order to ascertain outcomes. These approaches differ in the starting point for the definition of the sample. In the case-control study, the sample is defined by the outcome under study; whereas in the cohort study, the sample is defined by exposure status.

In the first two segments below, we consider some of the issues in design, analysis, and interpretation that are special to each of these research strategies, emphasizing those relevant to reproduction. In the third segment, we consider issues that are generally applicable to the study of the relation between an exposure and adverse reproductive functioning, regardless of the research strategy employed to examine the question.

1. Definition of the Control or Unexposed Group

a) The case-control study: In the case-control study the frequency of exposure among persons experiencing the disease (cases) is compared with that among persons not experiencing the disease (controls). By definition the control group is free of the outcome under study. Controls and cases should ideally be comparable in the following characteristics: 1) the a priori probability of exposure; that is, they should be drawn from the same population; 2) in the method of ascertainment; 3) in the method of data collection on exposure status and potentially confounding variables, and the reliability and validity of these data; and 4) in all characteristics (other than those being studied) which relate both to the outcome under study and to the exposure variable.

In practice, it is not always known at the outset of a study whether cases and controls are likely to differ in characteristics which relate both to the outcome and the exposure. Thus, in design of the study, efforts are made to ensure that cases and controls are comparable in all known risk factors for the disease. The ascertainment of cases and controls from the same population may be sufficient to ensure such comparability; in other instances, cases and controls may be matched for one or several characteristics which relate strongly to the outcome under study, and which may be associated also with exposure status. Table 4 lists, for several reproductive outcomes, the most strongly associated risk factors.

Table 4

Outcome	Significant Risk Factors
Spontaneous abortion	Maternal age Maternal smoking Maternal alcohol drinking Prior spontaneous abortions
Stillbirth	Maternal age Maternal smoking
Birthweight	Maternal age (young) Maternal parity Maternal ethnicity Maternal smoking Social class
Neural tube defects	Parental ethnicity (Irish vs non-Irish) Maternal age Social class (in some geographic areas) Prior neural tube birth
Down syndrome	Maternal age
Cleft lip and/or palate	Prior birth with cleft lip and/or palate
Trisomy (among abortions or term births)	Maternal age

More than one type of control may satisfy the criteria listed above. For example, if cases are the parents of infants with neural tube defects, the following control groups might be considered: parents of stillbirths; parents of livebirths with another type of congenital defect; and parents of normal liveborn children. The first two control groups in this example have the advantage that all parents have suffered an adverse reproductive outcome. Thus, if interview data are to serve as the primary source of information on exposure status, all parents may be equally accurate or inaccurate in their reports. They pose the disadvantage, however, that if exposure is related to more than one outcome, an association may be missed. The parents of normal children present converse advantages and disadvantages; that is, they allow the possibility of different accuracy in recall, but the certainty that the group is indeed unaffected without any adverse outcome. Often the solution to this dilemma in choice of controls is to select two control groups; one defined by an adverse outcome other than that being studied, and the other unaffected by any adverse outcome.

b) The cohort study: In the cohort study, the frequency of adverse outcome is compared between exposed and unexposed individuals. The study may be conducted retrospectively or prospectively with respect to outcome. Two approaches may be taken to define the unexposed group.

First, the unexposed group may have no exposure to the agent under study (e.g., comparison of individuals in an exposed community with individuals in an unexposed community).

Alternatively, the unexposed group may be exposed at lower doses than the exposed group (e.g., workers with varying levels of exposure to the same agent; individuals living at varying distances from the site of environmental contamination). In this instance, an effect may be missed if both low and higher doses of exposure are associated with the same effect.

The exposed and unexposed cohorts should be identical insofar as possible in:

1. The method of identification of the cohorts;
2. The method of follow-up;
3. The method of data collection on the outcome variables and control variables; and
4. Known risk factors for the outcomes under study.

The third criterion listed above puts some restrictions on the use of population data on the frequency of adverse outcomes as data of the 'unexposed' group. This approach can only be entertained if the methods of follow-up and data collection in the unexposed population were identical to those used for the exposed group, and if the unexposed population is comparable in all risk factors known to influence the outcome.

2. Case-Control and Cohort Studies Compared

a) Number of cases required: For a given effect size, level of significance and power, the case-control design will require fewer individuals than the cohort study, when the outcome being studied is rarer than the exposure.

When the exposure is rarer than the outcome, the cohort design for a given effect size, level of significance and power will require fewer individuals.

b) Number of controls and number of exposed: It is possible to increase power (for a given significance level and effect size) if two or three controls are chosen in a case-control design. Similarly, power can be enhanced by doubling or trebling the size of the unexposed in a cohort study.

c) Bias in recall: In case-control studies, this refers to the tendency for persons who have experienced an adverse outcome, or who have been made aware of a likely relationship, to recall an exposure selectively. In a retrospective cohort study, this refers to the possibility that the exposure status influences the ascertainment of outcome. For instance, this could occur if the source of data on adverse outcomes were through interviews with exposed and unexposed cohorts, rather than through prospective follow-up and ascertainment by the investigator. In retrospective cohort studies in which data on outcomes are obtained from medical records, consideration must be given to the possibility that exposure status may influence medical service use.

d) Estimate of risk: The measure of an association in cohort studies is the relative risk, while from case-control studies it is the odds ratio.

e) Time and cost: Prospective cohort studies require following a group over a period of time, hence they risk systematic attrition. To some extent this can be overcome with intensive efforts at maintaining cohort size, which may prove costly in the end. Some attrition may be inevitable, however. Case-control and retrospective cohort studies may involve about the same amount of time and expense.

f) Clinical description: Case-control studies may permit a fuller clinical investigation of the "case" (hence, more precise clinical classification). Prospective cohort studies may permit more precise measure of the exposure.

g) Spectrum of outcomes: Case-control studies typically explore one outcome. Therefore, if another outcome is the more usual effect of the exposure, it will be missed. Of course, several case-control studies, each of a different end point, may be carried out simultaneously. Cohort studies often permit a fuller appreciation of a spectrum of clinical outcomes. By contrast, case-control studies permit study of a fuller spectrum of exposures. However, this may be less of an advantage in the situations under consideration here.

3. Issues Specific to the Study of Reproductive Effects

In this section, we consider issues specific to the study of reproductive effects, regardless of whether a case-control or cohort study is undertaken.

a) The exposed parent and the time of exposure: The study of the relationship between exposure and human reproductive outcome is unique, since exposures to the mother, father, or both may influence reproductive success. Furthermore, these exposures may have occurred long in the past, immediately prior to conception, or during gestation. Even in this latter instance, exposures to the father may be relevant since some agents are carried in the semen and may be transmitted to the developing fetus via intercourse; and other agents may be carried home on work clothes and thus result in maternal (and fetal) exposure.

For some adverse outcomes, it is possible to limit the relevant period of exposure. Thus, nearly all chromosomal anomalies (excluding mosaicism and tetraploidy) must arise in a germ cell of either parent prior to conception or at fertilization. Exposures that occur during pregnancy are not relevant to the genesis of chromosomal anomalies. Similarly, decreased birthweight, unassociated with any other malformation or disability, is likely to result from exposures that occurred during gestation. For congenital malformations, the embryology of the defect will determine a time period after which an exposure could not have been causal.

b) Recall bias in assessment of exposure: This is the most serious disadvantage of a case-control study. Recall bias, which implies that the data on exposure may be influenced by the occurrence of the adverse outcome, must be suspected whenever interview data from affected and unaffected individuals serve as the only or primary source of information on exposure status. Recall bias may produce either spuriously positive findings (i.e., cases overreport exposure, controls underreport exposure, or both) or spuriously negative findings (i.e., cases underreport exposure). There are several ways in which the effects of recall bias may be assessed: 1) by comparison of self-reports of exposure with records of exposure collected prior to, or independently of, the outcome; 2) by biochemical measurement of exposure to the individual (e.g., plutonium, lead); and 3) by comparison of the frequency of self-reported exposure among several "worried" groups thought to have etiologically distinct conditions (e.g., chromosomally normal and chromosomally abnormal spontaneous abortions, neural tube defects and Down syndrome) with that among controls.

c) Source of data on outcome: Earlier we discussed the value of vital certificate data and medical records in identification of adverse reproductive outcomes. Another source of data in reproductive studies is interview data obtained from the parents. For outcomes such as birthweight and sex of the child, vital certificate data provide a source for validation. For outcomes such as congenital malformations, perinatal deaths and spontaneous abortions, hospital and physician records may be used for validation. However, since a large proportion of women may not seek medical attention for care of a spontaneous abortion, interview data will usually include spontaneous abortion data that cannot be validated from another data source. Since reporting of outcomes may vary with exposure status, the most prudent analytic approach to data of this type is to analyze the information on validated and unvalidated events separately. These analyses should be carried out controlling for gestation at abortion, since the majority of abortions for which care was not sought will have occurred in the 1st trimester of pregnancy. Insofar as these analyses within gestational groups (e.g., 8-12 weeks, 13-16 weeks, 17-28 weeks) show the same association, these data could then be combined.

If the identification of adverse outcomes is to depend on hospital records, physician charts, and vital certificates, then care must be taken to understand the medical facility use patterns of the population being studied. Information on birth certificates will provide some sense of these patterns; interviews with physicians, hospital administrators, and members of the community or workplace will provide additional information.

d) Can the unexposed sample be drawn from published data? In general, published series describing the frequency of reproductive outcomes among populations other than that under study will provide only "ballpark" estimates for the expected frequencies of events. Since, for the most part, these studies will not derive from the same population as the exposed sample, it is unlikely that they will substitute for the "unexposed" group in this second stage of an examination between exposure and reproductive outcome.

e) Control of potentially confounding variables: Nearly all of the known risk factors for pregnancy outcomes involve maternal exposures and characteristics (Table 4). In studies in which the effects of male exposure are being examined, the data on maternal characteristics and exposures will still need to be collected. Similarly, when the dependent variable is a characteristic of the sperm and semen, male characteristics and exposures are relevant.

SECTION VII

LEVEL III: LONG-TERM FOLLOW-UP STUDIES

With few exceptions, this type of design has been considered mainly for the purpose of discovering environmental carcinogens. The prospective studies of Hiroshima and Nagasaki, which are an important exception, examined reproductive outcome in the offspring of survivors.

In theory, one could create a list of individuals supposedly exposed to some adverse substance, and follow them till death. Such an approach could also be used to determine whether those individuals with a raised incidence of somatic cell changes are at increased risk for reproductive problems or cancer.

Such studies would be greatly facilitated by the existence of centralized and accessible registries of births, deaths and diseases, such as cancer, or of chromosomal anomalies, and by the development of automated procedures for record linkage and follow-up. Such systems are already being developed in

Canada and the United Kingdom, and include occupational information. Such automated follow-up procedures must have built-in safeguards so that confidentiality of health records and personal privacy are not compromised. In the U.S., the National Death Index is scheduled to begin functioning in 1981 [4]. The Index, to be created by the National Center for Health Statistics (NCHS), will enable those engaged in prospective studies to discover if an individual has died; and if he has, within which state. The next step, to discover cause of death for instance, would still be made at the state level but the Index is nevertheless an important advance on current possibilities for prospective studies. The NCHS is also planning a new data base on occupational mortality, and to this end a sample of states will begin the coding of the deceased's occupation, information which has long been part of the standard U.S. certificate, but usually not coded by state vital registrars. Census Forms which ask for detailed information on life-style and occupation are also proposed.

In order to permit follow-up studies using such records, sufficient information on exposed or control individuals must be kept to allow unambiguous identification of relevant vital and health records, including death registrations, and registries of cancer and genetic disease. Such information might include name, date of birth, names of parents, hospital record number, and other identifying information.

SECTION VIII

DATA ON SELECTED END POINTS

A. STUDIES OF SPERM*

1. Background

Studies of chemically exposed domestic animals, rodents, and humans have shown that sperm anomalies can be used as indicators and, in certain cases, dosimeters of induced antispermatogenic effects (for review see [5]). Since sperm are easy cells to obtain in large numbers and represent spermatotoxic as well as possible genetic damage to the gonads, sperm assays have received much attention in the assessment of reproductive damage and mutagenicity.

A recent survey of the literature [6,7] showed that human sperm assays have been more widely used than was generally suspected — more than 100 papers were found on the use of semen assays in assessing spermatogenic function in chemically exposed men. Studies cover 43 single agents, 11 complex mixtures, and 17 sets of multiple agents. About 85% of the exposures were to experimental or therapeutic drugs, about 10% were to occupational or environmental exposures, and about 5% involved personal drug use. Of the 71 chemical exposures studied, 48 were found to have some effect on human sperm production. See Table 5 for examples of the single agents studied. Studies of complex mixtures (e.g., occupational exposure to DBCP, personal use of tobacco, etc.) and combinations of 2 or more single agents were not included in this table.

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The mechanisms underlying induced human sperm anomalies are poorly understood. An evaluation of the results with sperm morphology assay in mice suggests that sperm morphology may have some value in identifying carcinogens and germ cell mutagens [6]. Data from human sperm studies are very limited, and at present, the main information that can be gained from the use of human assays is whether human spermatogenesis is affected by exposure to a chemical agent or mixture.

Table 5

Single Agents Studied with Human Sperm Assays[*]

Positive Effects	Suggestive but Inconclusive Effects
Acridinyl anisidide	Centchroman
Adriamycin	Colchicine
Aspartic acid	Diethylstilbestrol
Chlorambucil	Fluoxymesterone
Chlomiphene	Methadone
Cyclophosphamide	Metronidazol
Cyproterone acetate	Nitrofurantoin
Enovid	Trimeprimine
Gossypol	
6 Medroxyprogesterone	
Metandienone	
Methotrexate	
Nilevar	
Norethandrolone	
Norethindrone	
Prednisone	
Progesterone	
Salicylazosulfapyridine	
Testosterone	
Testosterone cyclopentylpropionate	
Testosterone enanthate	
Testosterone propionate	
WIN 13099[**]	
WIN 17416	
WIN 18446	

[*] Includes studies on sperm counts, motility, morphology, and YFF. The assignment of individual agents into columns is based on the data provided in the papers reviewed by the Gene-Tox Panel and may change as more data become available. Does not include studies of single agents showing no effect.

[**] See reference [7] for details of the "WIN" compounds.

2. Parameters to be Studied

Four human sperm parameters can be studied — count (density), motility, morphology (cytology), and YFF (a new test thought to represent Y-chromosomal nondisjunction during human spermatogenesis).

Sperm count is reported as the number of sperm per ml of ejaculate (or total sperm ejaculated) and is usually determined by hemocytometer. The measurement is technically easy and automated methods are also available. However, interpretation of results may be confounded by a number of factors such as variable continence time before obtaining specimen, and collection of incomplete ejaculate. For normal ranges see review by James [8].

Sperm motility is the swimming ability of the sperm, and is expressed as a percentage of motile sperm, on a graduated scale of 1 to 4, or as a combination of both. Although motility may be the best true performance evaluation of testicular function in relation to fertility, it is also the most subjective parameter of the four, and is very sensitive to time and temperature after collection. Thus, semen motility is a very difficult parameter to measure in a large-scale cross-sectional study, especially when samples are collected at home. Normal ranges of sperm motility have been difficult to define and are usually laboratory- and scorer-specific [9].

Sperm morphology (also referred to as seminal cytology) is the visual assessment of the shapes of sperm in an ejaculate. Although sperm-head shape is usually emphasized, some assessments also incorporate midpiece and tail abnormalities. In general, there has been little agreement in the definition of normal shapes or in the categories of abnormal shapes. This has resulted in much interlaboratory and interscorer variability [10,11]. Several studies [12-14] have shown that quantitative approaches to the visual assessment of morphology can be used with considerable success. The morphology assay is usually performed by using air-dried smears, fixed, and stained with a modified Papanicolaou method [15]. Classification of sperm into shape categories can be done systematically. The criteria for morphologic analyses must be carefully controlled and slides of concurrent controls should be analyzed together with slides of exposed men in a blind-study design. One approach to assuring a uniform scoring criteria for morphology is to make repeated comparisons to a coded set of standard slides at regular intervals throughout the analyses [7]. Using this latter method, normal ranges have been established for several unexposed populations.

The *YFF* test is based on scoring the frequency of fluorescent spots in human sperm stained with quinacrine dye. Based on studies in somatic cells, it is thought that these spots represent Y chromosomes. Although one expects approximately 50% of the sperm to have one spot, in practice frequencies are in the 30-40% range. The YFF test scores the frequency of sperm with 2 spots, which are thought to represent sperm with 2 Y chromosomes due to meiotic nondisjunction. Unlike the other sperm assays (counts, motility, and morphology), YFF has no counterpart in common laboratory or domestic animals. The Y-chromosomal fluorescence seems to be unique to man and the gorilla.

For the analyses of sperm with double fluorescent bodies, air-dried smears can be fixed and stained with quinacrine dihydrochloride as described by Kapp and Jacobson [16]. It is recommended that 2,000 sperm be scored under a fluorescent microscope as OF (sperm containing no fluorescent body and presumably those with no Y chromosome), IF (those sperm presumably with one Y chromosome) and 2F (sperm presumably containing 2 Y chromosomes). Smears should be of the quality required to easily visualize approximately 50% of the sperm with a single fluorescent body.

The YFF test is very new and only a few populations of exposed men have been analyzed. Normal ranges have been tentatively described [16,17].

3. When are Sperm Studies Indicated?

The following data are very important in making any decision on whether the use of human sperm studies is warranted:

a) What are the demographics of the exposure (size of the exposed population, geographic location, etc.)?
b) Who was exposed (predominantly men, predominantly women, or both); what were the age groups predominantly exposed?
c) What are the details of the exposure (route, duration, and when did it occur in relation to the proposed time of semen collection, etc.)?
d) What are the estimates on the exposure dose (can the exposed population be divided into dose groups)?

Sperm studies in man may be warranted if there are:

a) Sufficient animal data available indicating an effect of the exposure on spermatogenesis.
b) Data that suggest there may be a problem with human infertility or pregnancy outcome that could be linked to the male.

A screening questionnaire may be used to determine if the fertility of the exposed couples is affected. This may be followed by a more detailed questionnaire to confirm this observation and to determine to what extent the infertility may be male or female related. If indicated, sperm studies may then be done to determine whether human spermatogenesis is affected and what the cause might be. This could be done as a cohort or case-control study. A systematic, statistically rigorous approach for selection of participants would have to be developed.

4. Problems in the Application of Sperm Studies

The four human sperm assays described above are technically straightforward methods. The assays for counts, morphology, and YFF are parameters that do not appear to be affected by postejaculation technical factors. Motility, however, is highly sensitive to time and temperature factors. In studies where home collection is used the motility assay is not practical.

In Table 6, the variation of the sperm assays for counts, morphology, and YFF bodies are compared. Of the three assays, the morphology assay requires the smallest sample size to detect with confidence a 25% change in the mean compared to control values. However, it is important to recognize that an exposure may affect one of these parameters and not the others. Data are still insufficient to predict a) which assay would be most sensitive to an agent, and b) the interdependence of these end points. The conservative approach for assessing changes in human spermatogenic function should include assays for sperm count, morphology, YFF, and, whenever practical, motility.

The number of men exposed to the agent as well as the likelihood of high exposures are important for study design. Since between-male variability in semen characteristics is high even among fertile and presumably healthy men, rather large numbers of cooperative subjects are required to establish differences between control and exposed groups in cross-sectional studies in which each individual is usually sampled only once (see Table 6).

Longitudinal study designs may be more appropriate when fewer men are available for sampling. In this study design, repeated semen samples from a man are collected at different times in relation to the time of exposure to assess chemically induced sperm defects. Since variation of sperm morphology within an individual is considerably less than variation among individuals [12], in principle, fewer people are required to detect induced changes. These studies, however, have several major constraints: a) repeated samplings during a period of months and perhaps years are required; b) samples before exposure are needed, (or within days of an acute exposure, which is probably insufficient time for any induced effects on morphology likely to be seen); and c) the number of men needed for an effective study is unknown.

The effects of age, smoking, illness, medication, and other possibly confounding factors must be considered in the analysis of all human sperm data.

Table 6

The Relative Statistical Sensitivities of Three Assays of Human Sperm [17]

	Counts	**Morphology**	**2 F Bodies**
Assumed Distribution	Log-normal	Normal	Normal[a]
Mean[b]	132×10^6/ml	41.9%	0.8%
Std. Dev.	160×10^6/ml	12.4%	0.7%
25% change in mean (Std. Dev. Units)	0.2	0.8	0.3
Sample size for 5% level test with 80% power	214	26	41

[a] With square-root transformation.

[b] Based on 35 unexposed, new hirees in a pesticide plant.

B. FERTILITY

There is evidence of environmental exposures that cause short-term or long-term infertility, affecting both men and women.

The number of livebirths to supposedly exposed individuals is a relatively crude way to discover the relevant process. Nevertheless, in the case of DBCP-exposed men, the dysfunction was apparent in time to the workers and their families, and it was their own reports that initiated investigations.

Several attempts have been made to use measures of fertility in surveillance:

1. A life-table technique for workers in different industries keeping information on all pregnancies as they occur.

2. A standardized fertility index, that derives an expected number of births for each worker from the National Fertility Survey. Each worker fills out a three-minute form, which covers limited demographic and reproductive information; and from that source, the number of live children that are expected to have been fathered by him are calculated. If this were done for men and women in a whole factory or industry, then a deficit, if large enough, could be demonstrated.

3. Another measure of fertility is to compare the duration of time that elapses between the initiation of 'unprotected' intercourse and livebirths. This type of measure requires more detailed interviews (Level II studies) compared to the above, but it does provide useful information. Case-control studies of miscarriages and births can certainly be used to study this end point further.

To our knowledge, infertility clinics have not yet been used for epidemiologic purposes.

We have no equivalent for women of the sperm studies now being used in men. Ovulation and menstrual cycles might be studied, in this context, but so far have received rather little attention.

C. SPONTANEOUS ABORTION

A full evaluation of the relation of a particular agent to reproduction must consider the consequences of exposure to either parent, the timing of exposure, the dose and duration of exposure, and a broad range of possible outcomes. It is unlikely that any one study can be designed to evaluate all of these hypothetic pathways, and all of these effects. However, studies of spontaneous abortions offer the possibility of examining a range of outcomes including: frequency of fetal loss overall, chromosomal abnormality, morphologic malformations, and fetal death for apparently normal conceptions.

In this section we discuss:

1. The frequency and nature of spontaneous abortion.
2. The utility of studies of the aborted conceptus.
3. Advantages in studying chromosomal and morphologic anomalies among abortions, rather than livebirths.
4. Special considerations in studies of spontaneous abortions.

1. The Frequency and Nature of Spontaneous Abortion

Approximately 15% of recognized pregnancies end in spontaneous abortion by 28 weeks gestation. Estimates from interview studies and hospital-based studies range from 10-20%; estimates from prospective studies are on the order of 20-25%. The probability of spontaneous abortion is highest in early gestation, gradually decreasing with increasing gestation. The frequency of spontaneous abortion is increased among older women (aged 30 years or more), among women with previous spontaneous abortions, and among women who smoke or drink alcohol during pregnancy. Thus, comparison of rates of spontaneous abortion for different populations should consider whether variations in these known risk factors are likely to confound the analysis and interpretation.

Approximately one-third of spontaneous abortions occurring between 8-28 weeks gestation are chromosomally abnormal. Many of the anomalies found among spontaneous abortions rarely or never survive to term; others (trisomy 13, 18 and 21) occur both among abortions and term births, but in far greater frequency among abortuses. Chromosomal anomalies are at least 60-fold more common among spontaneous abortions than term births (Table 7).

Table 7

Incidence (per 1000) of Selected Chromosomal Anomalies in Induced Abortions, Spontaneous Abortions, and Livebirths

Anomaly	Induced Abortions (from ref. [20])	Spontaneous Abortions (ref. [21])	Livebirths (ref. [22])
Sex Chromosome			
45X	8.00	43.42	0.05
47XXX	6.00	0.00	0.52
47XXY	4.00	4.20	0.46
47XYY	2.00	0.00	0.46
All Trisomies	16.00	156.85	1.27
Trisomy 13	not banded	9.80	0.05
Trisomy 18	2.00	7.00	0.12
Trisomy 21	not banded	7.00	1.25
Trisomy 16	2.00	61.62	0.00
Triploid	2.00	50.42	0
Tetraploid	2.00	14.01	0
Rearrangements	0.00	12.61	2.37
Other	24.00	40.62	.30
All abnormal	68.00	322.13	6.20
Number studied	500	714	56,952

About two-thirds of spontaneously aborted conceptions are chromosomally normal. About half of these chromosomally normal abortions are morphologically abnormal; in the majority of such cases, the dysmorphology suggests that embryonic development has been disrupted early in gestation. In the remainder of dysmorphic cases, an embryo or fetus with an anatomic malformation is found.

Some of these anomalies (e.g., neural tube defects, cleft lip and/or palate, ventricular septal defect) are similar to those found among term births; others have been described only among spontaneously aborted conceptuses.

In the remaining one-quarter of spontaneous abortions, an apparently normal embryo or fetus is identified.

An increase in the frequency of spontaneous abortion might reflect an increase in one or several of these different outcomes. Studies of the abortus, when feasible, may elucidate which outcome has been affected. In the absence of these studies, gestation may serve as a crude indicator of the likely effect. The frequency of chromosomal anomalies among spontaneous abortions decreases with increasing gestation. Among abortions less than 16 weeks gestation, at least 50% of aborted conceptions are chromosomally abnormal; among abortions of 16 weeks gestation or later, the frequency of chromosomal anomaly is approximately 25%. Thus, an exposure leading to an increase in late abortions only is most likely to have led to an increased risk of fetal death for chromosomally normal abortions. The converse statement cannot be made so easily for early abortions, since an excess might represent either increased aneuploidy or an increased rate of fetal death for euploid conceptions.

2. The Utility of Studies of the Conceptus

Cytogenetic and morphologic studies of the conceptus will, of course, help to elucidate the mechanism which accounts for an association of an exposure with spontaneous abortion. These studies can aid also in distinguishing the relevant definition of exposure, with respect both to time of exposure and parent exposed. For nearly all chromosomal anomalies the error must occur either prior to conception, in the germ cells of either parent, or at the time of fertilization. Thus, knowledge that the conceptus is abnormal chromosomally will usually limit the search for causal factors to those exposures which occurred in either parent prior to conception. For some anomalies (i.e., tetraploidy, mosaicism), the search is directed to exposures occurring at the time of fertilization or shortly thereafter. In some trisomies and most triploidies, it is possible to distinguish the parent in whom the cytogenetic error originated; in such cases, the search for antecedent factors can be narrowed down to that one parent.

The processes underlying the abortion of chromosomally normal conceptions can be illuminated by consideration of the morphology of the abortus. For instance, in about half of these cases, the aborted specimen consists of only fragments of membrane, yielding little information on the stage of intrauterine life when development stopped. A similar proportion of specimens consists of an empty sac, sometimes with a small portion of cord. The early cessation of development which is evinced here may be due either to factors operating prior to or at the time of conception (associated possibly with genetic mutations which are, as yet, not able to be measured) or to factors operating early in gestation.

The epidemiology of this type of abortion is virtually unexplored.

In the remaining 50% of abortuses, an embryo can be identified. These embryos are of at least three types: disorganized embryos, apparently normal embryos, and embryos with focal malformations. In the first type of embryo, development has been disrupted early in gestation; causal factors most likely lie in exposure(s) occurring either prior to conception or early in gestation. In the second type, the embryos are apparently normal, although they vary in size, sex, gestation, and in degree of maceration. Each of these dimensions may provide clues as to the process underlying the abortion. The abortion of such an embryo or fetus raises the possibility that a maternal factor, including the wide range of common or unusual substances to which the mother is exposed and which cross the placental barrier, has interfered with the maintenance of pregnancy. In the third type, that is, an embryo or fetus with a focal malformation but normal karyotype, teratogenesis may again have its source in the fetal environment provided by the mother.

3. Advantages in Studying Chromosomal and Morphologic Anomalies Among Abortions Rather than Livebirths

a) Statistical power

The majority of chromosomally abnormal conceptions (at least 92%) are lost through spontaneous abortion rather than surviving to term. It appears also that a large proportion of fetuses with focal malformation are lost through spontaneous abortion, although this frequency is not as well documented as that for chromosomal anomalies. Since the frequency of these anomalies is considerably higher among abortions than among livebirths, changes in either the type or frequency of abnormality may be missed in studies of defects at term, unless the exposed population is very large, or the defect unique and rare (e.g., thalidomide and phocomelia). There is statistical power to detect smaller effects when the relationship of exposure to defect is studied in abortions rather than births. Furthermore, there is economy in the numbers of pregnancies which must be examined. For example, in order to identify an exposure which doubles the incidence of chromosomal defect at conception, the records of 22,843 exposed newborns would have to be examined to insure an 80% chance of detecting the effect. In contrast, to detect the rise in the incidence of spontaneous abortion which would follow on this same increase in the frequency of chromosomally abnormal conceptions, only 901 exposed pregnant women need to be identified. An even more powerful test would be to search for an increase in chromosomal anomaly within a population of spontaneous abortions; this approach would require a sample of only 180 abortuses in order to detect the same doubling.

b) Increased lead time

Another important advantage to the use of studies of spontaneous abortions is the lead time gained for the discovery of an agent which induces abnormality in the conceptus. Since spontaneous abortions generally occur 6 months earlier than term births, they provide an opportunity for earlier detection of hazards.

c) Potential for precise examination of the conceptus

Spontaneously aborted conceptions, unlike liveborn infants, can be examined fully: cytogenetic and pathologic study of the abortus permits specification of the outcomes with rare precision, and in some instances may permit measurement of exposure to the hypothesized hazard.

4. Special Considerations in Studies of Spontaneous Abortions

a) Ascertainment

Ascertainment of spontaneous abortions by definition is limited to spontaneous abortions among recognized pregnancies. Estimates of the frequency of unrecognized spontaneous abortions (that is, spontaneous abortion in which the woman was not aware she was pregnant and in which pregnancy cannot be confirmed by pathologic examination of the specimen) vary considerably. In a recent study [18] where urinary hcG levels were measured on the 3rd week after the last menstrual period, the frequency of unrecognized abortion (raised hcG level, but no other sign of pregnancy) was estimated to be about the same as that of recognized abortion. As understanding of the meaning of these raised hcG levels grows, there is the exciting possibility that an outcome, previously unstudied, can be added to the list of end points set out at the beginning of this report. As yet, however, the determinants of these raised hcG levels are unstudied, and it is not clear whether the causes of recognized and unrecognized abortions are similar.

Several sources of data can be used to identify recognized spontaneous abortions: vital certificate data, hospital records, physician records, interview data from either the woman or her mate. For the most part, vital certificate data will provide an incomplete source of information on abortions occurring before 20-34 weeks gestation, since reporting of these fetal deaths is not required in most states. The completeness of ascertainment from either hospital records or physician records will depend, of course, on the frequency with which medical attention was sought for care of spontaneous abortion, and patterns of service use. Both frequency and type of medical service use are likely to vary between communities, and possibly with socioeconomic variables as well. In a series of women identified for spontaneous abortions in New York City, the frequency with which women sought care for spontaneous abortions varied with gestation. In about 40% of 1st trimester abortions, no medical care was sought. Interview data from the women are likely to provide the most complete ascertainment; however, it has the disadvantage that in some instances episodes of amenorrhea may be reported as spontaneous abortions.

b) Analysis of retrospective data on spontaneous abortions

One approach to enumerating all spontaneous abortions that occurred in a defined population over a defined time period is to obtain information from three sources; the woman, physicians in the area, and hospital facilities. This series of spontaneous abortions will comprise four types of events: spontaneous abortions reported by women with no medical attention having been sought; spontaneous abortions reported by the woman and validated through medical records; spontaneous abortions in which the women report seeking care but no record of this care is found; and, lastly, spontaneous abortions identified in medical records but unreported by the women.

The first two categories (abortions where no medical attention was sought and abortions validated in medical records) are likely to be the largest.

One approach to the analysis is to consider these two (or four, depending on the size of the sample) categories of spontaneous abortions separately; that is, to examine the relation of spontaneous abortion to exposure among women who did not seek medical attention, and women who did. Before this analysis is undertaken, the gestational distribution of abortions in these two groups should be compared, since it is likely that the proportion of abortions under 13 weeks of gestation is higher among women who did not seek medical care than among those who did. Since exposure may lead to abortion at only one period of gestation, gestation should be controlled (possibly in intervals: less than 13 weeks, 13-16 weeks, and 17-28 weeks) in examining the relation of exposure and spontaneous abortion among women who sought medical care and those who did not. Insofar as the association of exposure and spontaneous abortion is similar within gestational groupings for each of these groups of women, the data could be combined to consider the relation of exposure to spontaneous abortion in general, regardless of whether or not medical confirmation was obtained. Insofar as the findings are not similar, the possibility of bias, either in reporting or in care-seeking behavior, must be entertained.

c) Reliability and validity of reproductive histories

In view of the limits set out regarding the use of medical records for spontaneous abortion research, investigators increasingly are utilizing an interview approach, taking maternal and/or paternal reports of reproductive history. There is some evidence to suggest that maternal reports of reproductive events are reliable: that is, interview information is generally compatible with reproductive history data in the woman's obstetric record [19]. It has been hypothesized that paternal recall will be less reliable and precise as to frequency and timing of events than maternal recall. However, there is virtually no evidence regarding the validity (as opposed to reliability) of reproductive history data obtained by interview.

d) Equalizing the period at risk

By definition, spontaneous abortions occur at an earlier gestation than do livebirths. This may present a problem in case-control studies of spontaneous abortion, where controls are term births. In defining exposure status during pregnancy, the period of gestation examined in the control should be confined to that of the referrent case. Failure to do so could result in the spurious finding of an association between exposure and the favorable outcome (term birth), since controls are at risk of exposure for a longer period of time than are index cases.

e) Defining the unit for analysis

The occurrence of one spontaneous abortion increases the risk of abortion in subsequent pregnancies. Thus, conceptions occurring to the same couple are not strictly independent, nor are characteristics of the couple (e.g., age, smoking and alcohol use) which need to be controlled in the analysis of fetal loss. At issue is whether in the study of spontaneous abortions (and some other reproductive end points), analyses should be based solely on the number of couples, or on the number of pregnancies, or some combination of the two.

In the past, some reproductive research has based sample size on the number of pregnancies, ignoring the independence assumption required by the analytic method applied. The practical reason for wishing to use pregnancies as the unit is that frequently (especially when investigating a localized phenomenon) one is able to cull only a small sample of exposed individuals, though each may have had several conceptions. Resolution of this methodologic problem rests on utilizing an analytic technique which controls for the degree of dependence between different conceptions to the same couple. The use of such analytic techniques permits pregnancies, rather than women, to be considered the unit of analyses. Increases in statistical power will usually follow on this increase in the sample size.

D. CHROMOSOMAL ABNORMALITIES

Information on chromosome abnormalities diagnosed at amniocentesis or at birth is sometimes routinely available through special chromosome registries. Examples of this are the New York State-Northeastern Chromosome Registry [23], and the Interregional Cytogenetics Registry System [24].

Participating cytogenetic laboratories in these registries report (anonymously) all information on all amniotic fluid culture results, both normal and abnormal, as well as the parental ages, reason for study, and county of residence of parents. Only abnormal results from diagnostic chromosome studies done on peripheral blood or other tissues are reported to the New York-Northeastern Regional Registry, while all cases, both abnormal and normal, are reported to the Interregional Registry, including detailed information on chromosomal polymorphisms.

New York-Northeastern Regional Registry members include all licensed cytogenetics laboratories in New York State as well as a few laboratories from New England. Reporting of chromosome abnormalities is mandated by the New York State Department of Health, and the Registry is run by the Birth Defects

Institute. The Interregional Registry is supported by NIH funds, and consists of five laboratories across the USA located in New Haven, Connecticut; Albany, New York; Indianapolis, Indiana; Memphis, Tennessee; and Portland, Oregon. (Participation of these laboratories is subject to review by human subject review boards of each institution.)

Since all cases of amniocentesis for chromosome analysis are reported in these registries, they will provide reliable data on the incidence of chromosome abnormalities at 16-20 weeks of gestation for women of a given age who have amniocentesis. The overall incidence of chromosome abnormality in cases coming to amniocentesis is usually about 2%, and is close to twice the incidence seen in livebirths to women of the same age. The difference is partly due to loss of many of these chromosomally abnormal fetuses as late spontaneous abortions or late fetal deaths. The rate of amniocentesis use in women over 35 years varies widely throughout the country, but in the regions served by these registries it is used by about 15-30% of all eligible women on the average. Although women undergoing amniocentesis differ in such things as socioeconomic status from women not undergoing amniocentesis, this is unlikely to influence the frequency of chromosome abnormalities.

While data on the incidence of chromosome abnormalities in livebirths can also be obtained from registries, this is less satisfactory because of uncertainty about the size of the population from which the cases were drawn. Since the New York State Registry includes all registered cytogenetics laboratories in the State, it should include all cases diagnosed out of the total of livebirths in the State, with the exception of the small number of patients who obtained cytogenetic studies across the State line. Since it is rare these days for a diagnosis of Down syndrome to be made without cytogenetic confirmation, and the other autosomal trisomies are also confirmed this way, the ascertainment of cases of autosomal trisomy from cytogenetic laboratories should be almost complete. The incidence of sex chromosome anomalies cannot be obtained this way, since these conditions usually are undiagnosed for many years after birth. The Interregional Registry has an uncertain denominator, since it is unclear what population is served by the large referral hospitals in the registry. If it is assumed that the size of the population at least remains relatively constant, then temporal comparisons can be made in any one region.

Hook has suggested that one can use an intraregistry comparison, the proportion of all livebirths with an autosomal trisomy syndrome which are the result of a sporadic (nonfamilial) chromosome rearrangement, as an index of the rate of chromosomal damage and thus, of mutation, in the population [25]. Using this approach, Hook was able to demonstrate an increase in sporadic Robertsonian translocations occurring over a particular period in the New York Registry.

At the stage of a Level II investigation, the regional incidence of chromosome abnormalities at amniocentesis and at birth can be obtained by canvassing cytogenetics laboratories which serve the populations of interest. The fact that these laboratory services are usually centralized in large medical centers makes this task relatively simple.

E. BIRTHWEIGHT

There is little question that birthweight is the single most accessible and general end point for reproductive failure. Its advantages in many parts of the world are:

It is recorded routinely and with a high level of precision for livebirths. The information is, therefore, available for Level I studies, as well as for more detailed analyses.

It is certainly a sensitive index of several known environmental exposures, for instance, smoking.

It correlates with other outcomes (in particular, curtailed gestation; congenital malformations; late fetal death; neonatal death; sudden infant death syndrome; chromosomal syndromes). Since it is often more uniformly recorded than some of these, it serves as a screen for other outcomes.

It provides a continuously distributed variable with attendant statistical advantages.

Some limitations of birthweight as an end point are:

It obviously omits infertility, very early loss, and often late fetal death from consideration.

It is affected by known personal attributes and habits, like prepregnancy weight, cigarette smoking, and social class, by major differences in dietary intake, and by ethnicity. Therefore, in Level I studies, unless some of these are controlled in making comparisons, results that rely on community comparisons will often be unsatisfactory. On the other hand, Level II studies have a real advantage when studying birthweight, because so much is already known and can be taken into consideration in the design.

As survival rates for very low birthweight infants are by no means uniform, if birthweight studies are restricted to livebirths, distortions could occur. On the other hand, birthweight is less completely recorded for fetal deaths (even late fetal deaths).

Birthweight can often be combined with duration of gestation, and read as "intrauterine growth retardation." Duration of gestation is less precisely recorded for many reasons, one being that real doubt may exist regarding the time of conception. Methods have been developed to overcome this problem. One criticism of these methods is that the women for whom the datum of gestation is less precise differ somewhat sociodemographically from the general population, and applying a correction to the data glosses over these differences.

F. SEX RATIO

Sex of infant among livebirths is as regularly recorded as birthweight, so that sex ratio (no. of males/no. of females) is a widely accessible end point. The at-birth sex ratio is always close to 1, with slight excess of males: in Britain it has fluctuated between 1.03 and 1.06 since 1870. Less data are available at earlier stages of gestation, but data on karyotyped spontaneous and induced abortions indicate that the sex ratio in recognized pregnancies is also close to 1. There are problems with the latter data because of the possibility of maternal rather than fetal cells being examined, but there can be little doubt that the large excess of males once thought to be present in early gestation is not there even as early as 6-8 weeks.

Genetic theory predicts that an induced increase in mutation rates will lead to an excessive loss of males when the female is exposed, and an excessive loss of females when the male is exposed, though these trends will be disturbed by changes in rates of nondisjunction affecting the sex chromosomes. The sex ratio was intensively studied in the atomic bomb survivors, finally leading to the conclusion that differences in the expected direction were not demonstrated.

Differences in sex ratio have been suggested to result from the time of fertilization relative to ovulation. Changes have been found in wartime conceptions, and one study suggested social class differences. Whether these changes related to time of fertilization, or to environmental factors affecting postconception survival is not clear.

G. CONGENITAL MALFORMATIONS

Classification

Congenital malformations are usually classified according to the WHO International Classification of Disease System. This is the system used, for example, in the two monitoring systems described in Section IV above for Level I investigations. For small populations, the rarity of most individual malformations (see Table 8) makes it necessary to attempt to group them in some way; often, the overall rate of major and minor malformations may be the only way to use the data, in spite of the obvious problems with such a heterogeneous grouping (see Section III). A common approach to grouping malformations is by organ systems — thus, one may group CNS defects, facial clefts, limb defects, heart defects, etc. Such a classification may make little sense in a study whose focus is etiologic, since it will inevitably create groups of heterogeneous etiology. It would be better to attempt to identify cases with similar types of cause, e.g., known single gene defects, chromosomal defects, known environmental causes, multifactorial situations, etc. This is difficult in practice without extensive history taking and examination of the patients. However, it may sometimes be achieved using hospital records, and could be attempted at a Level II investigation.

Table 8
COMMON MAJOR MALFORMATIONS AMONG NEWBORN INFANTS

ICD #	Malformation	Rate per 1000
740.0	Anencephaly	1.04
741.9	Myelomeningocele	.33
742.3	Congenital hydrocephalus	.36
745.1	Transposition of the great arteries	.26
745.4	Ventricular septal defect	1.17
746.7	Hypoplastic left heart	.23
747.0	Patent ductus arteriosus	.72
749.0	Cleft palate	.33
749.1	Cleft lip and cleft lip with cleft palate	.78
752.5	Undescended testes	1.21
752.6	Hypospadias — first degree	2.12
752.6	Hypospadias — second or third degree	.52
	Clubfoot — all types	2.05
754.51	Talipes equinovarus	1.01
754.53	Metatarsus adductus	.49
754.6	Calcaneovalgus	.55
754.3	Congenital dislocation of the hip	.78
755.0	Polydactyly — Type B	2.15
758.0	Down syndrome	1.53

Prevalence of all major malformation is 26/1,000. These data are derived from surveillance of 30,681 infants of at least 20 weeks gestational age born at Boston Hospital for Women.

Other classification schemes based on the embryologic process involved in the formation of the defect may also be considered as our knowledge of these relationships increases.

The advantage of a meaningful classification scheme is to permit refinement of the malformation variable. For example, if 1st trimester exposures are being considered, inherited single gene defects, chromosomal anomalies and defects due to postural abnormalities in the 3rd trimester may be ignored.

It has been suggested that certain dominantly inherited traits could be used as "sentinel phenotypes," so that monitoring the incidence of new noninherited cases could reveal a change in the mutation rate. Such a condition would have to be readily diagnosable at birth, be relatively free of heterogeneity, and have a high rate of penetrance. Classic achondroplasia, polydactyly, and the acrocephalosyndactyly syndromes have been suggested as possible candidates, although all are unsatisfactory for one reason or another, in our view.

Methods of Study

The observed incidence and variety of congenital malformations will vary depending upon the age, race, and sex of the patient examined, and the methods used for examination. A trained, motivated, systematic examiner will find a much higher rate of defects, and an autopsy will always reveal more malformations. An appropriate control group must take all of these factors into account. If both exposed and unexposed children are to be examined, the observer should be unaware of their exposure status.

In Level I or Level III studies, it may be possible for children at risk to be examined by special study physicians. These should be trained in the identification of major and minor congenital anomalies, and in syndrome recognition. The examination should be conducted using a checklist of features (for example, see Table 9). A decision must be made beforehand whether to include minor anomalies in the study, and whether to include examination for internal anomalies, such as heart defects or abnormalities found only at autopsy. Chromosome analysis should be carried out if possible on all children with any two of growth retardation, multiple anomalies, or mental retardation.

The identification of a consistent pattern of malformations as the result of a particular exposure is convincing evidence of a real effect. The recognition of such a "syndrome" should then be one of the goals of a Level II investigation of malformations. However, it is also possible for the effects of a particular exposure to vary, ranging from spontaneous abortion to congenital defect, to growth retardation, and to behavioral abnormalities. The effects may vary with dose and time of exposure, as well as with other fetal and maternal factors.

H. PERINATAL AND INFANT DEATHS

In the United States, late fetal deaths (stillbirths), neonatal and postneonatal deaths are well recorded, although gaps have been demonstrated. Also, several states link death certificates for infants dying in the first year of life with their birth certificates, making an invaluable data source available to Level I investigations.

Perinatal deaths are strongly influenced by socioeconomic status (which may be related to role of exposure); they also reflect the incidence of malformations, of low birthweight (<2500g), and increasingly, of very low birthweight (<1500g).

Therefore, the more useful end point seems to be birthweight. Although the rate of survivors among very low birthweight infants shows considerable temporal and geographic variability, this variation today is mostly considered to relate to level of medical care.

One study suggested that a pollutant of water was related to perinatal deaths for low birthweight infants. And some studies show that maternal smoking not only increases the rate of low birthweight babies but also adds to the mortality experienced by them.

The risk of sudden infant death is increased by low birthweight, and by maternal smoking.

Table 9

NEWBORN EXAMINATION

Head

- Frontal bossing
- Keel-shaped brow
- Flat brow
- Prominent occiput
- Flat occiput
- Metopic suture palpable
- Metopic suture open to glabella
- Metopic fontanel
- Third sagittal fontanel
- Scalp defect
- Double whorl of hair
- Triple whorl of hair
- Absent whorl

Face

- Supraorbital ridge palpation
  - Left
  - Right
  - Both
- Eyebrows confluent
- Flat bridge of nose
- Prominent bridge of nose
- Nostrils anteverted
- Long nasal septum
- Epicanthal fold
  - Left
  - Right
  - Both

Eyes

- Ptosis
  - Left
  - Right
  - Both
- Brushfield spots
  - Left
  - Right
  - Both
- Iris stroma visible
- Iris freckles
  - Left
  - Right
  - Both

Mouth

- Cleft lip microform
- Cleft gum
- Cleft uvula
- Prominent gum
- Broad alveolar ridge
- Small chin

Ear

- Preauricular skin tag
  - Left
  - Right
  - Both
- Skin tag on ear lobe
  - Left
  - Right
  - Both
- Skin tag elsewhere
- Preauricular sinus
  - Left
  - Right
  - Both
- Darwinian tubercle
  - Left
  - Right
  - Both
- Darwinian point
  - Left
  - Right
  - Both
- Helix overfolded
  - Left
  - Right
  - Both
- Elf ear
  - Left
  - Right
  - Both
- Lack of helical fold
  - Left
  - Right
  - Both
- Lack of helical fold on top only
  - Left
  - Right
  - Both
- Interrupted helical fold
  - Left
  - Right
  - Both
- Lack of helical fold opposite tragus
  - Left
  - Right
  - Both
- Bridged concha
  - Left
  - Right
  - Both
- Tragus bifid
  - Left
  - Right
  - Both
- Tragus absent
- Ear lobe crease
  - Left
  - Right
  - Both
- Ear lobe notched
  - Left
  - Right
  - Both
- Ear lobe bifid
- Sinus on ear lobe
- Sinus on back of helix
- Lop ear
  - Left
  - Right
  - Both
- Cup-shaped
  - Left
  - Right
  - Both
- Excess helix tissue
- Helix attached to scalp

Neck

- Webbed
- Excess neck folds
- Branchial sinus

Chest

- Extra nipples
  - Left
  - Right
  - Both

TABLE 9 (Cont'd)

Areolar skin tag
  Left
  Right
  Both
Xiphoid bifid
Xiphoid not palpable

Abdomen
Diastasis recti
Single umbilical artery
Umbilical hernia

Genitals-Male
Testis in scrotum
  Left
  Right
  Both
Testis in groin
  Left
  Right
  Both
Hypospadias (1°)
Chordee
Epispadias
Scrotum-up-on-penis

Genitals-Female
Labia majora divided
Labia minora prominent
Vaginal tag

Back
Sacral dimple
Pilonidal sinus
Dimple over bone
  On shoulder
  Over sacrum
  Other

Arm
Cubitus valgus

Hand
Clinodactyly 5th finger
  Left
  Right
  Both
Thumb retroflexible
  Left
  Right
  Both
Tapered fingers
Overlapping fingers
  Left
  Right
  Both
Fingernails
  Hyperconvex

Simian crease
  Left
  Right
  Both
Bridged simian
  Left
  Right
  Both
Simian-trans. nml
  Left
  Right
  Both
Sydney line
  Left
  Right
  Both
5th finger
  Single crease
  Left
  Right
  Both
5th finger
  Extra crease
  Left
  Right
  Both

Legs
Genu valgum

Feet
Increased space
  1-2 Toes
  Left
  Right
  Both
Syndactyly 2-3 to 1 IP
  Left
  Right
  Both
Syndactyly 2-3 to tip
  Left
  Right
  Both
Overlapping toes,
5th over 4th
  Left
  Right
  Both

Overlapping toes,
  Other
  Left
  Right
  Both
Recessed 4th toe
  Left
  Right
  Both
Toenails, hypoplastic
Toenails, hypoconvex
Heel prominent
  Left
  Right
  Both
Sole crease
  Left
  Right
  Both

Other Features (Describe)

I. TRANSPLACENTAL CARCINOGENESIS

Introduction

To our knowledge, environmental agents seldom cause cancer during childhood. The main reason may be that latent periods are usually longer than the 15 year pediatric span.

The shortest latent period known for a human cancer is 2 months — for lymphoma after immunosuppression for renal transplantation. In theory, infants might be born with lymphoma because their mothers received immunosuppressive therapy during pregnancy. A substantial number of women have been so treated, but we know of no lymphomas in their children.

Cancer can, of course, be present at birth. The incidence in the U.S. during the first month of life is about 37 per million livebirths [26]. We do not know environmental causes of such tumors, with one exception: neuroblastoma at birth in a child with the fetal hydantoin syndrome [27]. Four children are known with this combination of disorders in the U.S., 1976-1980. The experience illustrates the possibility of identifying environmental carcinogens by either concurrence of cancer with syndromes known to be due to specific environmental agents, or through the maternal history when congenital tumors are observed. The possibility of an environmental cause would be enhanced if time-space clusters of specific congenital tumors were observed.

Diphenylhydantoin is apparently the second transplacental human carcinogen identified, the first being diethylstilbestrol (DES). Here, a cluster of a rare tumor of the cervix or vagina in young women in Boston led to recognition of the contributing role of DES. As usual, the environmental pathogens were identified by simple retrospective studies. Some prospective studies of DES have been made, but the frequency of cancer in daughters is too low to be determined precisely. But of those taking the drug during pregnancy, only about one in a thousand of their daughters developed cervical or vaginal cancer [28]. Instead of seeking details of the exposure and natural history through prospective studies, a special national disease registry for patients with DES-type tumors was established. Information about the interval of susceptibility during pregnancy, and about pathogenesis was obtained in this way.

Methods of Study

Teratogens and carcinogens are almost always first detected by alert clinicians. A causal relationship suggested by a series of clinical observations can sometimes be tested by study of data in tumor registries or in prospective studies conducted for a general health surveillance. Thus, DES was confirmed as a transplacental carcinogen by data from the New York State Tumor Registry. And knowledge of the fetal alcohol syndrome was extended by study of data already collected in the collaborative Perinatal Study of the NICDS. Thus, these resources can be used more to amplify relationships than to detect them in the first place.

When an exposure is suspected to be carcinogenic and a substantial population of children have been exposed, one can match identifying information of the population at possible risk against tumor registry data or against the National Death Index [29]. In this way, follow-up will be simplified. A unique number for each person would greatly aid in matching. Adults usually have Social Security numbers, but children do not. Attention should be given to providing such numbers at birth, a suggestion made for the U.S. at least as long ago as 1968. Recording the mother's maiden name and her Social Security number can be of great assistance in follow-up of children.

Regional excesses in mortality from specific cancers can be informative in identifying environmental causes of adult neoplasia, e.g., asbestos exposure of shipyard workers during World War II as a cause of increased lung cancer in cigarette smokers in coastal Georgia. Childhood cancers are probably insufficiently frequent for detection in this way of transplacental carcinogens.

Problems in Studying Transplacental Carcinogens

We can reason that any agent that can cause cancer in adults and is known to cross the placenta may cause cancer in the offspring, including constituents of cigarette smoke, immunosuppressive drugs, and benzene. None are (yet) known to be transplacental carcinogens. Perhaps the cancers they induce are among the more frequent types and long delayed in their onset. Both of these circumstances would mask their detection. Diphenylhydantoin occasionally causes lymphoma in persons using the drug for epilepsy. One would expect children with the fetal hydantoin syndrome — due to the same drug, to be prone to lymphoma. Instead, unaccountably, they develop neuroblastoma.

Finally, it should be pointed out that clusters of specific cancers in children may lead to the detection of an environmental carcinogen, such as DES, but many excellent studies of leukemia clusters in childhood have failed to reveal an environmental cause. Leukemia is frequent enough to cluster often by chance in a country as populous as the United States. Vaginal cancer in young women is too rare for such clusters to occur, thus explaining the difference in the two experiences.

The detection of cancers from transplacental chemical pollutants will perhaps best be recognized in the future as such occurrences have in the past; namely, by alert clinicians practicing in the area where a new environmental agent has been introduced.

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SECTION IX

SUMMARY OF PUBLISHED DATA AND AN ANNOTATED BIBLIOGRAPHY ON EXPOSURES AND REPRODUCTIVE FUNCTION

The following tables are an updated version of those originally appearing in an article by Strobino, Kline and Stein (J Early Hum. Devel 1:371-399, 1978). The papers summarized in the tables represent a selected bibliography. Special attention was given to original and early reports, and also to recent reports. An attempt was made to include all population studies: case reports were included if they were numerous, or where the effects seemed highly specific in relation to a particular agent.

THE TABLES

Criteria for selection

We have tried to locate all reports in the scientific literature in 1976 and the previous 10—15 years that related detrimental effects in humans to parental exposures to chemical and physical substances. A bibliography was compiled from our own reading, from consultation with colleagues (see Acknowledgements), from annotated reviews of special areas, Medline searches and the Index Medicus.

From this bibliography we selected the papers summarized in the tables. Special attention has been given to early and original reports, and also to those published in the current period. We have tried to include all population studies, whether the associations reported were present or absent or inconclusive. With regard to case reports, we have included those where the case reports were many, or where the effects seemed highly specific in relation to a particular agent.

Key to the tables

The effects of each supposedly hazardous agent are summarized in Tables I—III.

+ indicates that the investigation was based on population groups and that it demonstrated a statistically significant association. + may also refer to a study based on multiple case reports of a highly specific outcome (e.g. fetal alcohol syndrome).

0 indicates that no association was demonstrated.

+/0 indicates that the results of a study were inconclusive because the data were conflicting or statistical tests were of borderline significance.

+? indicates that an association was demonstrated but only on the basis of single case reports, or of population studies in which the study design was seriously flawed. 'Serious' flaws include such features as lack of a control group or no consideration of statistical significance.

The problem of power (0)

In considering studies marked 0 in the tables, we found that little or no attention had been given to the statistical power of a study. Power is the probability that an effect, if present, will be detected. The power of a study is determined by the frequency of the outcome variable in the unexposed population, the size of the effect expected, the acceptable level of significance, and the sample size [1,2]. The frequency of developmental disability is always low in the unexposed population. Also, the size of the effect to be expected from exposure to a mutagen or teratogen is usually small. By a small effect we mean, for example, that there is a relative risk of

1.3 when exposed individuals are contrasted with unexposed individuals. Very large sample sizes are therefore required before an investigator can, with confidence, accept the null hypothesis that there is not even a small effect.

Insufficient numbers and a failure to consider power undoubtedly explain some of the many inconsistencies between studies that are noted in the tables. Taking the investigations of maternal irradiation and leukemia as an example, several investigators [108–109,111–113] have shown that children exposed to diagnostic irradiation in utero have an increased risk of developing leukemia or other malignancies in later life. Kato [107] was unable to confirm these findings in survivors exposed in utero to the A-bomb even though the dosage was extremely high. One explanation is that the exposed population, 1300, is of insufficient size to detect a relative risk of 1.4 — 1.6. The power is, in fact, well below 75%.

If the expected incidence of an outcome in an unexposed population is 0.001, and the risk of the outcome is doubled in exposed individuals, then a population of 10,100 exposed individuals must be studied in order to have a 75% probability of demonstrating a difference significant at the 5% level (using a two-tailed test). In this calculation we have assumed that the expected incidence is constant and that data on an unexposed sample will not be needed. If this assumption cannot be made and two samples (exposed and unexposed) are required, then one would need 20,195 individuals in each sample to detect a relative risk of 2.0.

Of course, if exposure is always or almost always associated with detrimental effects, these considerations become unimportant. They do weigh heavily, however, if a particular exposure is announced to be 'not a hazard', on the basis of an inadequate study.

TABLE I

Maternal exposure at conception and/or during pregnancy (N.B.: numbers refer to references)

Agent	Outcomes								
	Spontaneous abortion		Sex ratio	Late fetal deaths	Neonatal deaths	Low birth weight/ prem.	Developmental disabilities	Cancer	Childhood mortality
	Rate	Fetal defects							
I. Drugs A. Sex hormones									
oral contraceptives				0 18			0 16 0 17 0 18 0 177		
ovulation stimulators	+? 22 (ectopic pregnancy and hydatidiform mole)						+? 19 +? 20 +? 21		
hormonal pregnancy tests							+ 23 0 24		
androgens and certain synthetic progestogens							+ 25 masculinization + 26 of female fetus		
progesterons and/or any other sex hormone use (some studies combine all hormone administration together e.g. pregnancy tests, medication to prevent abortion, etc.)							+ 27 + 28 + 29 + 30 +? 31 0 32 (27–32: congenital heart defects most often)		

TABLE I (continued)

diethylstilbestrol											+	178		
											+	179		
											+	33 adenosis	+	33
											+	37	+	34
											+	38	+	35
											+	39	+/0	36
											0	37 (urinary tract anomalies)	+	37
													0	40 (males)
											+	40 (males)		
B. Anticonvulsants	0	41			0	41	+	41	0	41	+	41 mostly cleft		
	0	49			+	47	0	47	0	47	+	42 lip with or		
(in several of these studies it is difficult to separate the effects of the drug from those of the disease for which it is given, i.e. epilepsy)	0	52			0	48	+	48			+	43 without		
					0	51	+	51			+	44 cleft palate and		
											+	45 congenital		
											+	46 heart defects		
											+	47		
											+	48		
											+?	50		
											+	51		
											+	52		
											+	53		
C. Antimetabolites														
folate antagonists (methotrexate, aminopterin)	+	54	+	54							+	54		
											+	55		
alkylating agents											+?	55		

TABLE I (continued)

Agent	Outcomes								
	Spontaneous abortion		Sex ratio	Late fetal deaths	Neonatal deaths	Low birth weight/ prem.	Developmental disabilities	Cancer	Childhood mortality
	Rate	Fetal defects							
D. Tranquilizers									
thalidomide							+ 56		
							+ 57		
							+ 58		
diazepam							+ 59		
							+ 60		
							+ 61		
others (meprobamate, chlorodiazepoxide, etc.)	0	62		0 62	0 64	0 62	0 62		0 64
	+/0	63		0 64			+ 63		
	+	66		0 66			0 64		
							+? 65		
							0 66		
E. Salicylates				+ 67	+ 67	+ 67	0 67		
				0 69	0 69	0 69	0 68		
F. Oral anticoagulants (warfarin)				+ 70			+? 70		
							+? 71		
							+? 72		
							+? 73		
							+? 74		
G. Heroin	+	75							

TABLE I (continued)

Agent	Outcomes								
	Spontaneous abortion		Sex ratio	Late fetal deaths	Neonatal deaths	Low birth weight/ prem.	Developmental disabilities	Cancer	Childhood mortality
	Rate	Fetal defects							
II. Alcohol			+? 78	+ 175	+ 76	+ 76	+ 76 fetal		
			(inc. in			+ 77	+ 77 alcohol		
			affected			+ 79	+ 79 syndrome		
			females			+ 80	+ 80		
			at birth)			+ 81	+ 81		
						+ 82	0 82		
	+ 180					+ 173	+ 173		
	+ 181					+/0 176	+ 174		
	+ 182	(chromosomally normal)					0 175		
III. Smoking	+ 87	+ 94*	0 84	0/+ 83	+/0 83	+ 83	0 84		
	+ 88		0 86	+ 84	+ 85	+ 84	0 91		
	0 89		0 87	+ 85	+/0 86	+ 85	0 92		
	+/0 91		0 92	+ 86	0 88[1]	+ 86	+ 90		
	0 95				0 89	+ 87	+ 171		
	+ 96			0 89		+ 88	+ 183		
	+ 171			0 91	0 92	+ 89			
	+? 172			0 92	0 93	+ 91			
	+ 182	(chromosomally		0 93		+ 92			
		normal)		0 97	[1] includes	+ 93			
					all	+ 95			
					peri-				
					natal				
					deaths				
IV. Irradiation high doses	+ 99		0 105	+ 99	+ 99	+ 106	+ 98 micro-	0 107	+ 106
			(F_2 gen-		+ 106		+ 99 cephaly		
			eration)				+ 100 and men-		
							+ 101 tal retardation		
							+ 102 predominantly		
							+ 103		
							+ 104		

TABLE I (continued)

low doses		+ 115 (F, generation)				0 114 0 116	+ 108 + 109 0 110 + 111 + 112 + 113 +/0 116	+ 112 (whites) 0 112 (blacks) 0 114 0 111
V. Environmental pollutants: occupation and residence								
anesthesia	+? 117 + 118** + 119*** + 121 + 122*** + 123*** 0 124 +? 125	+/0 117 0 121	0 121 0 122 + 124	0 121	+? 117 + 119 + 124	0 117 + 120 +/0 121 +/0 123 + 124	0 122	0 122
	+ 184							
vinyl chloride						+/0 126 0 185 0 186		
mercury						+ 127 + 128		
lead						+? 129 + 130		
(lead smelter)	+ 187				+ 187	+ 188		
polychlorinated biphenyls (PCB)			+? 131		+? 131	+? 131 (skin staining)		

TABLE I (continued)

dioxin			+? 132	
hydrocarbons				0 189
industrial chemicals	+ 190			
hospital laboratories	+/0 191			
laboratory work		0 192 perinatal	+ 192	
hospital work medical occ.)	+ 193 "threatened abortion"	0 193 perinatal	0 193	
		+/0 193 perinatal	+/0 193	
hexachlorophene		0 193 perinatal	+? 194	

*Association with chromosomally normal fetuses.
**Association also found with early abortion.
***No association found with gestation at abortion.

KEY
+ association demonstrated;
0 no association demonstrated;
+/0 evidence inconclusive;
+? association present but questionable.

TABLE II

Maternal exposure before conception only (N.B.: numbers refer to references)

Agent	Outcomes									
	Fertility	Spontaneous abortion		Sex ratio	Late fetal deaths	Neonatal deaths	Low birth-weight/ prem.	Developmental disabilities	Cancer	Childhood mortality
		Rate	Fetal defects							
I. Drugs										
sex hormones		+ 195 (dec.) + 196 (dec. chromosomally normal)								
oral contra-ceptives	+ 143 + 144	0 133 0 135 0 18 + 142 (dec.) + 169 (dec.)	0 134 + 136 + 138 0 140 + 141	+ 137 (inc ♀) +? 139 (inc ♀) + 27* 0 140 (spont. abort) +/0 18* 0 168	0 133 0 18 + 169 (dec.)	0 133	0 133 0 169	0 133 0 135 +/0 16 + 27 0 17 0 18 0 170		
DES (in utero)		+ 198 and "all poor pregnancy outcomes"					+ 197 and "all nonviable outcomes"			
ovulation stimulators								+ 145 0 146 0 147		
anticonvulsants					0 48	0 48		0 48		
antineoplastic agents	+ 55							0 148		

TABLE II (continued)

II. Alcohol							+ 176	+? 174	
III. Smoking							0 86		
							0 95		
IV. Irradiation	+ 149			+/0 150	0 150	0 150	0 150	0 150	0 150
high doses	0 152				0 151	0 151		0 151	
low doses		+ 158	+ 158		0 155	0 155		0 153 Down	+ 113
		0 161						+ 154 Down	
								+ 155 trisomies	
								+ 156 Down	
								0 157 Down	
								+ 159 Down	
								+ 161 Down	
								0 162 Down	
								0 163 Down	
								+/0 160	
V. Environmental pollutants: occupation and residence									
anesthesia	+ 121								

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*These studies have shown an excess of males in abnormal offspring from pregnancies where mother had been exposed to oral contraceptives in the past.

KEY

+ association demonstrated;

0 no association demonstrated;

+/0 evidence inconclusive;

+? association present but questionable.

TABLE III

Paternal exposure (N.B.: numbers refer to references)

Agent	Outcomes									
	Fertility	Spontaneous abortion		Sex ratio	Late fetal deaths	Neonatal deaths	Low birth-weight/ prem.	Developmental disabilities	Cancer	Childhood mortality
		Rate	Fetal defects							
I. Drugs										
anticonvulsants								+ 44 0 48 + 49		
antineoplastic agents	+ 55									
II. Alcohol	+ 199 (azoospermia, testicular atrophy and pathology)						0 82			
III. Smoking							0 97			
IV. Irradiation										
high doses	+ 149 0 150 + 200 (sperm count)			+/0 150	0 150 0 151	0 150 0 151	0 150	0 150 0 151	0 150	
low doses			+ 14					0 153 0 154 0 156 (overall X-ray exposure)	+/0 113	
microwave exposure	+ 201 (sperm count and motility)							0 160 (overall X-ray exposure) + 156 (radar exposure) 0 160 (radar exposure) 0 159 0 162		

TABLE III (continued)

V. Environmental pollutants: occupation and residence								
anesthesia	0 122	+? 117 0 122 0 123	+/0 117	0 122	+? 117	0 117 0 122 (major malf.) + 122 (minor malf.) + 123	0 122	0 122
vinyl chloride		+ 202 + 184 + 164 +/0 203				0 202 0 184 0 126 0 204 0 186		
hydrocarbons							+ 165 0 166 0 205	
chloroprene		+ 167						
kepone	+ 132							
lead	+ 207 (sperm morphology) + 207 (sperm count and motility)						+ 206 (Wilms)	
DBCP	+ 208 (azoospermia, oligospermia) + 209 (sperm count) 0 209 (sperm morphology) + 210 (sperm count)							
		+ 211						
Ethylene dibromide	+/0 212 (infertility)							
Epichlorohydrin	0 213 (sperm count and morphology)							
Carbaryl	+/0 214 (sperm)							

KEY
+ association demonstrated;
0 no association demonstrated;
+/0 evidence inconclusive;
+? association present but questionable.

TABLE IV

Exposure to either parent - community studies (N.B.: numbers refer to references)

Agent	Outcomes									
	Fertility	Spontaneous abortion		Sex ratio	Late fetal deaths	Neonatal deaths	Low birth-weight/ prem.	Developmental disabilities	Cancer	Childhood mortality
		Rate	Fetal defects							
Love Canal landfill		+ 215 + 216					+ 216			
Lead (lead smelter)		+/0 219					+ 217 + 218	0 188		
2,4,5-T		+? 224						0 220 (cleft palate) +? 221 (neural tube defects) 0 222 + 223		
Dioxin		+/0 225						+/0 225		

KEY
+ association demonstrated;
0 no association demonstrated;
+/C evidence inconclusive;
+? association present but questionable.

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REPORT OF PANEL III

MONITORING THE HUMAN POPULATION FOR MUTAGENIC EFFECTS:
Detection of Gene Mutations and DNA Damage

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I. BACKGROUND AND INTRODUCTION

Man is exposed to a rapidly increasing variety of synthetic chemicals in the environment. Among the agents represented in this proliferation of chemicals are some which have mutagenic effects. The list of chemicals for which there is experimental evidence of genetic activity has expanded rapidly during the last two decades. While mutation contributes to the genetic diversity of a species, there is reason to believe that spontaneous mutation rates are sufficient to maintain the genetic variability of the human population. Furthermore, substantial evidence indicates that induced increases in the mutation rate could produce a general decline in genetic health.

Since the consequences of exposure of human populations (or those of other species) to mutagenic agents occur long after a chemical is introduced into the environment, early identification of potential hazards is crucial to minimizing exposure and resultant risk. Consequently, a number of agencies of the federal government, as well as private industry and academic institutions, have exerted considerable effort in the development of efficient and effective methods for the detection of mutagenic agents. Preliminary efforts also have been made to evaluate quantitatively the relative potency of mutagens and the implications (risk) of mutagen exposure for public health. Such attempts at mutagenic risk assessment, however, are at an early stage of development.

The task of assessing risks associated with chemical mutagens is extraordinarily complex because of the great diversity of mutagenic agents, and the multiplicity of ways in which they can interact with biologic systems. Chemical agents are known to have genetic consequences both by inducing alterations at the chromosomal level and by inducing mutations at the single gene level. Chromosomal alterations, as described in Panel I Report, include changes in chromosome number (aneuploidy and polyploidy), and changes in chromosome structure (deficiencies, translocations, duplications, and inversions). Gene mutations can result from base-pair substitutions, frameshift mutations (additions and deletions of base pairs), or multilocus deletions. All of these genetic events may have serious implications for the genetic welfare of man.

Human genetic disorders attributable to alterations in chromosome number (aneuploidy) include Down syndrome (mongolism), Klinefelter syndrome, and Turner syndrome. In the majority of cases, these conditions are attributable to devise, develop, and use test approaches which detect and evaluate mutagenic potential, and there is a concomitant need for human population monitoring for potential mutagenic hazards.

The need to develop screening programs to detect environmental chemical mutagens is emphasized in the Environmental Mutagen Society position paper known as the Committee 17 Report [2]. These programs should address our needs to both identify approaches to the assessment of mutagenic potency as well as to provide a data base for the overall toxicologic safety evaluation process. Toward the latter objective, the U.S. Environmental Protection Agency has established the "Gene-Tox Program" for assessment of the current status of bioassays in genetic toxicology. The results of tests on more than 2,800 chemicals in approximately 39 genetic bioassays have been evaluated and encoded for computer analysis in the near future [3].

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A. SUSCEPTIBLE POPULATIONS

The conditions described in the previous section are multisystem disorders involving abnormalities in the skin, brain, immune system, skeleton, etc. With the disorders involving repair deficiency — ataxia telangiectasia (AT) and Fanconi anemia — molecular explanations for the cellular observations are rapidly evolving, but there is at present no obvious relationship between the different defects in DNA repair and most of the clinical signs of the disorders. It is important to determine whether these signs are the result of damage to DNA and if so, whether this damage is produced intrinsically by the human body or by extrinsic agents.

No defect in DNA repair has as yet been discovered for Bloom syndrome, but the similarity of many features to those of Fanconi anemia and AT makes it likely that such a defect eventually will be revealed. Assuming that this is the case, the occurrence of a DNA repair defect, chromosomal instability, frequent immune defects, and a high incidence of cancer in Bloom, Fanconi, and AT patients suggest that these disorders and signs are related. The study of the molecular basis of these diseases is only in its infancy, and further experimentation will no doubt provide further examples of repair-defective human conditions which will aid in elucidating the mechanism of repair and mutagenesis, and also hopefully of carcinogenesis.

The molecular and cellular studies described above have certain direct clinical applications. They can provide the possibility of early (even prenatal) diagnoses and of a *reliable* diagnosis before the onset of the full clinical symptoms; for example, before XP patients have developed tumors. In the case of AT, the cellular studies suggest that full doses of radiotherapy should be avoided. They also have the potential of ascertaining which, if any, forms of chemotherapy might be similarly contraindicated.

The diseases themselves, despite their rare occurrence, are of general importance. The epidemiologic studies of Swift have suggested a possible increase in the incidence of cancer in AT and Fanconi heterozygotes [1]. He has estimated that such heterozygotes comprise approximately 1% of the population. The high incidence of cancer in these individuals may be a significant proportion of the total cancer incidence in the general population. Identification of such heterozygotes, for example by increased cellular or chromosomal sensitivity to chemical mutagens, would allow monitoring for cancer in subjects at risk.

II. THE NEED FOR GENETIC MONITORING

Although human populations are not deliberately exposed to potential mutagens in order to measure mutagenicity, there nevertheless are circumstances in which observations can be made directly on humans. These most often occur because of occupational exposure or administration of therapeutic drugs. In these instances, the typical approach is to identify an exposed group and a suitable control group for comparison of some genetic effect. The experimental design is thus similar to that used with other organisms. The numbers of persons required may be small or very large, depending on the test used.

A different situation occurs when a population is to be monitored to detect an increase in mutations over time, usually with no suspect mutagen in mind. In this instance, the population serves as its own control, the rate at one time being compared with the rate at another. Again, the population required may be large or small, depending on the mutational parameter being assessed.

It is the general goal of population monitoring and testing to develop efficient systems that require fewer persons and resources; a goal that, at present, is far from achievement. In considering tests that might be applicable to humans, some limitations are obvious compared to experimental mammals. No test is acceptable that requires administration of hazardous materials, such as radioactive compounds or mutagens. Furthermore, biologic specimens are limited to body fluids the occurrence of meiotic nondisjunction in a parent of the affected individual. Such nondisjunction leads to the formation of germ cells having too many (Down syndrome and Klinefelter syndrome) or too few (Turner syndrome) chromosomes. Collectively, these and other clinically serious chromosomal abnormalities in man are estimated to occur with an incidence of 0.5% among total live births in the United States. In this context, it is noteworthy that Down syndrome is the most frequent, single definable cause of severe mental retardation. While the relationship between chemicals and the incidence of aneuploidy in man is uncertain, exposure to mutagens has been shown to increase the frequency of occurrence of aneuploids in several experimental organisms. Besides causing genetic abnormalities in children and adults, variations in chromosome number can lead to spontaneous abortion of affected human embryos, as mentioned previously in this book. It has been estimated that approximately 20% of all spontaneous abortions involve chromosomal defects, the etiology of which is unclear. Among such defects are both aneuploidy and polyploidy.

In addition to alterations in chromosome number, structural changes in the karyotype can have severe consequences. Among human disorders associated with such structural alterations are cri-du-chat syndrome (due to a deficiency), and a form of Down syndrome (involving a translocation), less common (about 5% of all cases) than the simple aneuploid type. A number of other rare but severe abnormalities are ascribable to structural aberrations in chromosomes. With recent major advances in cytogenetic techniques, there is now a much enhanced capability to detect chromosomal alterations in man. It is likely, therefore, that the proportion of disorders which can be associated with chromosomal damage may increase in the future. Significantly, it has been known for many years that mutagens can induce karyotypic abnormalities in a diversity of organisms.

Gene mutations can have a variety of effects depending upon the mechanism of gene expression. It should be noted, moreover, that the effects of gene mutations overlap with those of small deletions which may involve a chromosomal region larger than the confines of a single gene, and the two types of alterations may not always be readily distinguished. For simplicity, we can view gene mutations as changes which give rise to alterations in gene products or no product at all (null mutations). Depending upon the mode of genetic expression, the resultant mutant alleles may behave as dominants or as recessives. Also, several other types of dominance relationships are known to occur.

The relationship of the induction of gene mutations to human health is supported by the existence of a large number of genetic diseases which may be attributed to mutant alleles of a gene. Among the many dominant genes known in man are a number of alleles responsible for physical anomalies and malformations as well as other aberrant conditions. Included are some cancers referred to as hereditary neoplasms: the most thoroughly studied hereditary neoplasm is bilateral retinoblastoma, reported to occur with a frequency of 1/20,000 births.

The total disease burden attributable to dominant gene mutations has been estimated to be similar to that for chromosomal defects, approximately 0.5%.

Recessive mutant alleles are also of major importance with respect to human genetic disease. There are over 1,000 known genetic disorders which have the heritable properties of recessive gene mutations. Among these are such well-known diseases as sickle cell anemia, cystic fibrosis, and Tay-Sachs disease. Since an individual expresses a recessive characteristic only if he receives the recessive allele for that characteristic from both parents, many individuals undoubtedly carry deleterious recessive genes without expressing the corresponding disease state. In fact, carriers of deleterious recessive alleles typically far outnumber those with the disease, and most individuals probably carry at least one deleterious recessive [1]. Therefore, induced recessive mutations may remain unexpressed for a number of generations until a mating occurs in which both parents are carriers of a recessive allele for the gene in question. An offspring receiving the recessive allele from both parents would express the corresponding characteristic. There is concern among geneticists that exposures to new mutagenic agents in the environment could be increasing the genetic load of the human gene pool, and that the irreversible damage, which takes the form primarily of new unexpressed recessives, can lead to an increasing incidence of genetic diseases in future generations. Surveys of the pediatric wards of major metropolitan hospitals have revealed that at any given time 5-6% of the patients are suffering from diseases caused by single-gene defects — the result of homozygosity for recessive gene mutations. No estimates are available, however, concerning the fraction of these cases attributable, in whole or in part, to new mutations, as opposed to mutations derived through many generations of human evolution.

Taken in their totality, diseases of genetic origin are of major importance to human welfare. They can lead to debilitation and/or death and to the concomitant human suffering. It has been estimated that *at least* 10% of all human disease appears to exhibit a significant genetic component.

The contribution of chemical agents to the human genetic disease burden, either as it exists now or as it is likely to exist in the future, is highly debated. Concern over the possibility that certain chemicals may pose a threat to the integrity of the human genome and thus to future generations is predicated on sound theoretic reasoning. While definitive proof is lacking, there is little doubt that many chemicals have the potential to induce human mutations. Although man has evolved through thousands of generations with mutagenic agents present in the environment, the number and spectrum of such agents has increased markedly in our recent history due to the industrial revolution and recent technologic developments. Although we are still exposed to many naturally occurring mutagens, we are now also exposed to many man-made chemicals, some of which have mutagenic activity. This new type of exposure may be inflicting significant genetic damage in man. Because of this concern, there is a need to such as blood and urine or expendable cells such as blood cells, hair follicles, and sperm. These restrictions rule out some tests that are used in other organisms or in cell culture systems.

The development of systems to monitor human populations for genetic effects should be undertaken for several reasons: 1) there is a burden of human disease of genetic origin, and a consequent need to identify and eliminate causes of these disorders, where possible; 2) there is a likely role of genetic damage in the etiology of cancer; 3) the potential magnitude of environmental exposures to mutagenic substances and animal data suggests that such exposures may result in somatic and heritable mutations; 4) there is a need to diminish latency in detection of damage to man from these environmental exposures; 5) there is a need to compare findings in man directly with those derived from animal and short-term tests, thereby improving our ability to assess human risk based on non-human studies; and 6) there is a need to identify individuals unusually susceptible to genetic damage because of differences in repair capacity or metabolism. The background for the following discussion has been provided by others elsewhere [2-8].

Direct measurement of genetic damage in man by standard epidemiologic studies is difficult. Such studies typically employ the incidence of spontaneous abortion, congenital defects, or malignancy as an indicator of genetic damage: each of these end points has marked limitations. Thus, a feasible, properly validated group of assays which directly evaluated genetic damage in man would be of great value to us in our assessment of risk from mutagen exposures.

Laboratory-based monitoring techniques should make it possible to detect genetic hazards from environmental exposures sooner and after lower exposures than is currently required. The latency problem is well illustrated by the history of the detection of health effects from smoking. Cigarettes began to be consumed in large numbers around 1900; but it was not until the mid 1920s that death rates for lung cancer increased, and another 20 years before the association of these events was made. If similar delays occur in disease onset and detection of causal associations resulting from the exponentially increasing production of chemicals in the past two decades, the public health impact may be severe, though unknown for many years. A program of laboratory monitoring to detect these events prior to their clinical manifestation is obviously highly desirable.

Finally, although recently developed efficient and reliable batteries of mutagenesis tests have demonstrated that many chemicals are capable of causing mutation, extrapolation of these test results to human health effects is difficult. Development of parallel tests for genetic damage that can be applied both to man and to mammalian or submammalian systems might allow a direct comparison of human, and non-human findings. Where they can be undertaken (for example, among patients receiving chemotherapeutic drugs), such comparisons may provide important information for extrapolating risk to situations where direct comparisons are not feasible.

The need for monitoring goes beyond techniques to monitor cytogenetic abnormalities and reproductive events described in earlier sections of this book. As discussed in those sections, cytogenetic tests probably will not detect the full spectrum of genetic damage (e.g., may not detect gene mutations), and are costly. Reproductive effects frequently are not specific indicators of genetic damage and typically require monitoring large reproductively active populations. Because effective evaluation of human populations exposed to genetic and reproductive hazards probably starts with consideration of these effects, we have included cytogenetic and reproductive end points for comparison in our tables and briefly in the discussions that follow.

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III. CRITERIA FOR DEFINING A HUMAN POPULATION AT RISK

Exposure of human populations to hazardous chemicals produced by modern technology has been well documented. Based on the exposure and probable health effects, these populations can be divided into three groups: 1) low-risk; 2) likely-at-risk; and 3) at-risk. Low-risk populations are those who have no known exposure to hazardous agents.

Two types of populations likely-at-risk are those who have been exposed to 1) a known mutagen and/or carcinogen, or 2) mutagenic complexes or mixtures.

The mutagenic activity of known compounds can be determined by submammalian and mammalian assay systems. Mutagenic complexes and mixtures can be detected by mutagenic monitoring of the environment. Populations groups, such as industrial and chemical workers, hospital personnel, patients, etc., might be exposed to known mutagenic and/or carcinogenic compounds. Accidental exposure of a population to a known mutagen can also occur. Many workers, as well as residents in heavily industralized areas or in the vicinity of toxic waste dumps, may well be exposed to mutagenic compounds or mixtures of such compounds. The degree of the potential risk depends both upon the nature of the mutagenic compound or complex and the level of exposure.

Human mutagenic-monitoring studies should be performed for the population groups which are at-risk or likely-at-risk. Low-risk populations should be used for control studies as they establish baseline data for the given assay system. Mutagenic monitoring of the at-risk population may determine the association between genetic damage and particular health hazards in a human population. Establishment of the relationship between findings from mutagenic monitoring and from epidemiologic studies can eventually lead to quicker and better identification of potential risk and thereby provide greater protection of the human population from health hazards caused by mutagenic agents.

Likely-at-risk populations are the most diverse groups because of the different levels and types of exposures. The priority for the selection of these population groups for the mutagenic monitoring study is ranked as follows:

1. Exposed to high dose(s) of potent mutagens and/or carcinogens
2. Exposed to highly mutagenic complexes or mixtures, at unknown dose(s)
3. Exposed to low dose or to weak mutagens and/or carcinogens
4. Exposed to low dose or weak mutagenic complexes or mixtures.

Mutagenic monitoring of the population likely-at-risk could provide direct evidence as to mutational damage in exposed populations; could identify population groups for epidemiologic studies; and could provide biologic dosimetry for adverse health effects.

IV. METHODS POTENTIALLY USEFUL FOR EVALUATING HUMAN POPULATIONS EXPOSED TO MUTAGENIC HAZARDS

A. Body Fluids

A summary of methods potentially available to monitor human populations for genetic effects is provided below and in Tables 1 and 2. A description of specific assays follows in Appendix I.

The first method listed is for the detection of human exposure to mutagenic substances by the analysis of body fluids. (This technique might be used in conjunction with environmental sampling and chemical analysis, which was deemed beyond the scope of this report.) The most widely used bioassay technique (described in the Appendix) involves concentration of the sample by resin extraction and testing the sample for its ability to mutate bacteria.

The bioassay procedure provides a moderately sensitive test for the presence of mutagenic activity in body fluids. It is generally not as sensitive as standard analytic methods for chemical detection, but has the following advantages: 1) multiple substances and metabolites will be active in a single assay, enabling the detection of mutagenic activity from unanticipated sources; 2) the biologic activity is demonstrated, rather than simply the presence of substances; and 3) the assay may also tend to "integrate" the effect of multiple substances, although this capability has not been well characterized. The chief difficulties with this test are: 1) lack of sensitivity for certain specific substances compared with chemical techniques, 2) the decomposition of mutagens during storage or loss during extractions, and 3) limitations on our ability to concentrate a specimen. Also, solutions must be found to technical problems such as materials in the mixture tested causing toxicity or increased growth of tester organisms.

Alternately, chemical analysis of body fluids for specific mutagens may be undertaken. A technical description of these procedures is not included here. Chemical assays would be most appropriate for demonstrating exposure with established analytic techniques when specific substances are known to be in the environment, but the extent of human exposure is unclear. Biologic assays are most useful when exposures are suspected but the specific suspect chemicals (or possible interactions between chemicals) are uncertain, or when chemical analytic techniques are not available. It must be recognized that both the biologic and chemical approaches require that the mutagens enter the body fluid being tested. In most instances, exposure of other body compartments cannot be confirmed.

B. Expendable Tissues

Laboratory tests to detect genetic damage in specimens of human blood, sperm, or other tissues constitute a second means of evaluating humans exposed to mutagenic hazards. Potential tests are listed in Table 1. These techniques differ by: 1) the genetic specificity of the damage, which ranges from specific effects on chromosomes or DNA to indirect measures (such as alkylation of hemoglobin or altered sperm morphology); 2) the directness with which damage is assessed, from direct physical evidence of damage (DNA adducts, sequence changes, or chromosomal aneuploidy), to evidence of response to that damage (DNA repair or chromatid exchanges); and 3) the size of the genetic unit studied, from single nucleotides of DNA, to single genes, to entire chromosomes. These assays also differ in their statistical power to detect damage, and in the likelihood of their being affected by artifact, but little information is available to suggest the magnitude of these factors.

Table 2 describes the application of these tests to the measurement of damage in somatic tissue, germinal tissue, and offspring of exposed individuals.

Somatic tissue analyses generally utilize readily available blood products (typically peripheral lymphocytes). Sperm is the only readily available germinal tissue which excludes the sampling of women and possibly introduces sizable participant bias. Damage in germinal tissue, however, more clearly implicates risk of heritable effects — at least to the extent of demonstrating access of the active agents to germ cells.

Techniques for detecting damage in somatic or germinal tissues allow analysis of much smaller populations than techniques examining offspring. Monitoring offspring, however, provides the only direct test for inherited effects.

Further specifics of these assays are discussed in the Appendix. As noted above, cytogenetic methods and population monitoring techniques primarily described elsewhere in this report were included in these tables for completeness, but will not be discussed in the Appendix.

V. CURRENT STATUS OF THE USE OF MUTATION ASSAYS TO EVALUATE HUMAN POPULATIONS*

Given our limited understanding of, and experience with, direct laboratory tests for mutation in man, current use of these tests should be limited largely to studies aimed at evaluating the tests rather than evaluating exposed human populations.

The current practical uses of these tests in evaluating populations are probably best limited to the following:

1. Use of tests for mutagens in body fluids to seek evidence of exposures. Situations in which this appears to be of most value are when human mutagen exposure is suspected but not documented, and exposures are mixed, unknown, or when analytic chemical techniques are not available for the suspected compounds. From the onset it must be recognized that evidence of an exposure, not a demonstrated health effect, is being sought. Possible confounding exposures (e.g., smoking) require consideration, and test results must be statistically sound before the conclusion is reached that exposure to an environmental mutagen has occurred.
2. Testing small-to-moderate sized populations for unscheduled DNA synthesis, hypoxanthine phosphoribosyltransferase (HPRT) activity in peripheral lymphocytes, sperm morphology, and double Y bodies in sperm is currently technically feasible. However, these tests are presently of limited utility in assessing genetic risk. This is because the techniques for these assays are a) not sufficiently established to preclude technical problems or confounding factors in the populations studied which may affect results; b) the biologic validity of these tests as measures of specific genetic damage is unclear; and c) the health implications of findings in these assays are unknown.

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* This discussion excludes cytogenetic and reproductive studies which are considered elsewhere.

3. Large populations can be tested for electrophoretic variants in the blood of offspring. Of the currently feasible assays, this approach alone indicates an inherited gene mutation (although transmissibility has not been tested). The test is limited by the need for sampling large populations. The size of these populations may preclude testing of groups with specific exposures, limits ability to control for confounding exposures, and makes the technique logistically difficult.

TABLE 1. Possible Methods for Evaluating Human Populations Exposed to Mutagenic Hazards by Effect Monitored

Description of Test	Intended to Monitor
EXPOSURE	
Mutagens in body fluids	Direct exposure
Analytical chemistry of body fluids	↓
LABORATORY EVIDENCE OF GENETIC DAMAGE	
Hemoglobin alkylation	Protein damage
Damaged DNA bases in urine	DNA repair
DNA adducts*	DNA damage
DNA strand breaks*	
Unscheduled DNA synthesis*	
Induced (by N-OH-AAF)	DNA repair
Uninduced	↓
Sister chromatid exchanges†	Chromatid exchanges
HPRT activity in peripheral lymphocytes	Gene mutations
Sperm with variant LDH-X*	↓
RBCs with variant hemoglobins	
Chromosomal aberrations*†	Heritable or somatic chromosomal structural changes
Micronucleus test*†	↓
Chromosomal aneuploidy*†	Heritable or somatic chromosomal numerical abnormalities
Sperm morphology†	Germ cell mutations
Double Y bodies in sperm†	Chromosomal numerical abnormalities
Induced electrophoretic variants in blood	Heritable mutations
CLINICAL EVIDENCE OF DAMAGE	
Cancer†	Somatic cell mutations
Sex ratio change†	Mutation in offspring
Proportion of translocations in Down syndrome	Questionable mutation
Spontaneous abortions†	↓
Congenital defects†	
Sentinel phenotypes†	

* Techniques that may be applied to more than one tissue type.

† Techniques primarily discussed elsewhere in this volume.

TABLE 2. Possible Methods for Monitoring Human Populations for Mutagen Exposures: Somatic Cell Damage, Germ Cell Damage (Sperm) and Genetic Damage in Offspring

Description of test	Estimates Of Population size required	Latency	Persistence	Feasibility	Relative cost
MUTAGENIC EXPOSURES					
Mutagens in body fluids	tens	minutes	hours	+ +	+ +
Analytic chemistry of body fluids	↓	↓	↓	+ +	+ +
SOMATIC CELL DAMAGE					
Hemoglobin alkylation	tens	minutes	3 months (NTE 3)	+	+ +
Damaged DNA bases in urine			days to weeks	0	+ + +
DNA adducts			days to	0	+ +
DNA strand breaks			years	0	+ +
DNA sequences				0	+ + +
Unscheduled DNA synthesis			↓		
Induced			(constant)	+ +	+ +
Uninduced			hours	+	+ +
Sister chromatid exchanges^{a}		↓	days to months	+ +	+ +
HPRT activity in peripheral lymphocytes		days	weeks	+ +	+ +
Variant hemoglobins		months	↓	+	+ + +
Chromosomal aberrations^{a}		days	years	+ + +	+ + +
Micronucleus test^{a}			↓	+	+
(reticulocytes)			days	+	+
Chromosomal aneuploidy^{a}	↓	↓	years	+ + +	+ + +
Cancer^{ab}	thousands	years	lifetime	+ + +	+ +
GERM CELL DAMAGE (SPERM)					
Sperm morphology	tens	20 days	weeks	+ +	+
DNA adducts in sperm		weeks	to year	0	+ +
DNA strand breaks in sperm				0	+ +
DNA sequences in sperm				0	+ +
Unscheduled DNA synthesis in sperm		↓		0	+ +
LDH-X variants in sperm		months		0	+ +
Double Y bodies in sperm				+	+
DNA quantitative distributions in sperm	↓	↓	↓	0	+
Sex ratio changes^{ab}	tens of thousands	year	lifetime	+ + +	+

	Estimates of				
Description of test	Population size required	Latency	Persistence	Feasibility	Relative cost
GENETIC DAMAGE IN OFFSPRING					
Electrophoretic variants in blood	tens of thousands	generation	lifetime	+ +	+ + +
DNA sequences	↓	↓	↓	0	+ + +
Chromosomal aberrations[a]	thousands	↓	↓	+ + +	+ + +
Chromosomal aneuploidy[a]	↓	↓	↓	+ + +	+ +
Proportion of translocations in Down syndrome[a]	hundreds of thousands	↓	↓	+ + +	+ +
Spontaneous abortions[ab]	thousands	↓	↓	+ + +	+ +
Congenital defects[ab]	thousands	↓	↓	+ + +	+ +
Sentinel phenotypes[ab]	millions	↓	↓	+ + +	+ + +

(Feasibility Scale: 0 untested, + pilot studies, + + small studies, + + + currently available for monitoring)

a Methods discussed elsewhere in this report.

b Methods not requiring laboratory tests.

APPENDIX I

DESCRIPTION OF ASSAY SYSTEMS FOR HUMAN POPULATION MONITORING TO DETECT GENE MUTATIONS AND DNA DAMAGE

A. Measurement of Exposure
B. Measurement of Damage
C. Measurement of Somatic Cell Mutations
D. Measurement of Germ Cell Mutations
E. Measurement of Germ Cell Mutations in F_1 Progeny

A. MEASUREMENT OF EXPOSURE

Mutagenicity Assay of Body Fluids

A presumption of genetic hazard can be made if it can be shown that mutagens are circulating in the blood or are present in other body fluids. This is most readily done by testing the mutagenic activity of blood or urine in microbial systems or in in vitro cell culture systems [1,2].

Although it is not possible to equate mutagenicity of body fluids with actual mutation in the host, it is reasonable to assume that such persons are at increased risk of mutation. An increase in mutagenicity can be detected in single individuals, making the test especially useful in assessing exposure of small groups.

Potential confounding sources of genotoxic activity in body fluid samples include drugs, foods, beverages, and life-style factors such as stimulants, skin lotions, cosmetics, and suntan oil. Hence, studies in children may be particularly useful [2a]. In some cases, mutagenic derivatives of chemicals are excreted in biochemically conjugated forms, so that it is necessary to liberate the active genotoxic with enzymes. Certain chemical classes of genotoxins, such as those with short half-lives (i.e., nitrosamines), may not be detected by body fluid tests. Developmental efforts will be required to determine which classes of occupational hazards can be detected with body fluid tests.

Since blood and urine samples may be routinely collected during medical examinations, body fluid testing may be routinely integrated into a medical monitoring system. A battery of different genetic assays might be employed in order to detect the spectrum of potential genotoxic damage. The optimal methodology for a newly discovered hazard could be explored in laboratory animals when initial results are suggestive. The range, sensitivity, and specificity of these methods remain to be established. The costs of body fluid tests should be relatively modest. After a body fluid test achieves recognition as a useful surveillance tool for exposure to a chemical or a class of chemicals, it could substantively contribute to the prevention of occupational disease.

The urine analysis method is readily applicable to human monitoring situations at the present time for the following reasons:

1. Preliminary studies in humans have demonstrated that mutagens can be detected in the urine of humans exposed to various therapeutic drugs, industrial chemicals, and cigarette smoke under typical exposure regimens (Table 1). Thus, the application of this technology has been documented and knowledge of the normal background noise can be estimated;
2. Sample collection for urine is noninvasive and can be readily obtained from both sexes on a repetitive schedule;
3. Biologic analysis can be coupled to qualitative and quantitative chemical analysis;
4. Dose-response studies can be performed on target "indicator" cells of several phylogenetic levels including human cells, if desired;
5. Cost and performance time associated with this approach are amenable to large-scale sampling studies.

TABLE 1. Examples of Human Monitoring Using Urine Analysis

Population	Exposure	Response*	Reference
Patients	Cytoxan	+	Siebert and Simon, 1973 [3]
Patients	Metronidazole therapy	+	Legator et al., 1975 [4]
Patients	Niridazole	+	Legator et al., 1975 [4]
General	Cigarette smoking	+	Yamasaki and Ames, 1977 [5]
Nurses	Cytostatic drugs	+	Falck et al., 1979 [6]
Workmen	Styrene production	–	Cerna and Dobias, 1980 [2]
Workmen	Rubber manufacturing	+	Falck et al., 1980 [7]
Workmen	Coke production	–	Moller and Dybing, 1980 [8]
Workmen	Epichlorohydrin	+	Legator et al., 1978 [1]

* Microbial target cells employed in all studies.

One of the advantages of the urine analysis approach is the ability to process the urine samples before testing. Organic compounds can be concentrated in urine by passing the urine over sorbent resin columns such as XAD-2. Enzyme treatment of urine samples with sulfatases and glucuronidases will result in the release of conjugated chemical species. Thus, the potential for enhancing the responses of whole urine samples is very good with this method. Similar types of sample processing can be applied to fecal extracts. It should be noted, however, that sample processing techniques may introduce artifacts, and results must be interpreted with care. Blood has been studied less often but can be studied with appropriate modification.

A drawback to these types of assays is the inability to measure cumulative exposure. Of equal importance is the inability to translate the results into a quantitative risk, either for individuals or populations. Nevertheless, this test shows promise of being important in evaluating human exposure to many mutagens.

Detection of Chemically Damaged DNA Bases in Urine

Chemically damaged DNA bases may be solubilized by excision repair or by generalized nucleic acid breakdown and may be excreted via the urine in relatively undegraded states. Löfroth et al [9] noted the excretion of methylated purines following exposure to dimethyl sulfate and proposed that these compounds originated from methylated nucleic acids. Currently available chemical separation and identification techniques can detect such bases in experimental animals following the administration of large doses of alkylating agents. Such large doses are expected to be rare and extreme in human populations, and far more sensitive methods are desirable for human monitoring. Developmental work is underway at the University of California at Berkeley (B.N. Ames, personal communication) to adapt and combine sophisticated chromatographic separations and ultrasensitive mass spectrographic methods to resolve and measure large numbers of urine fractions. (At present, the ribosidic but not the nonribosidic fractions can be analyzed. A large number of expected base alkylation adducts have been prepared for model studies.) The general method is likely to be sufficiently advanced for at least limited application within only a few years. It is complicated by the presence of some 50 unusual base fractions which derive primarily from the normal turnover of tRNA, by its reliance on expensive and highly specialized procedures, and by its probable requirement for prior knowledge of the nature of base damage induced by particular agents. The method is nevertheless likely to display great sensitivity and chemical diagnostic capabilities.

B. MEASUREMENT OF DAMAGE

Unscheduled DNA Synthesis (UDS) in Peripheral Lymphocytes

DNA that is damaged by radiation or chemicals can often be repaired by localized removal of the damage area and replacement with new nucleotides. If the new nucleotides are radioactive, the extent of repair can be measured [10, 11]. A number of authors have suggested the measurement of DNA repair, usually designated unscheduled DNA synthesis, as an indicator of exposure of chromosomes to mutagenic agents. As yet, this system has not been used to detect damage to persons exposed to mutagens. A report that the in vitro induction of unscheduled DNA synthesis in lymphocytes of normal persons is positively correlated with chromosomal aberrations in circulating lymphocytes suggests that unscheduled DNA synthesis may be associated with mutation, although it is difficult to predict the nature of the association [12].

In this procedure, venous blood is drawn and the lymphocytes are removed and cultured in the presence of hydroxyurea to inhibit normal (scheduled) DNA synthesis. Tritium-labeled thymidine is added to the culture: the only DNA synthesis that should occur results from the repair of single-strand gaps generated by the excision of damaged nucleotides. The extent of tritiated thymidine incorporated into these gaps provides a measure of unscheduled DNA repair synthesis (UDS). There have been few UDS studies on lymphocytes in vivo. Limited studies of normal human lymphocytes have indicated that sex, age, and blood pressure may affect both the background and chemically induced responses. There is no available information on individual variability (day-to-day, sampling time, diet, life-style, emotional attitude, etc.). Controlled studies are required to evaluate such factors for their impact on population monitoring. If they can be controlled, and if suitable control populations can be identified, UDS in peripheral lymphocytes could become a usable exposure monitoring technique within only a few years. Repair synthesis is comparable in human and animal tissues, as are the methods for its measurement, with the important proviso that for general human application the incorporation of radioactive precursors of DNA must be carried out in vitro after the tissues have been sampled. The method is necessarily indirect; uptake of radioactivity need not be specific for repair; and there can be false negatives due to toxic inhibition of repair. Following an acute exposure to a mutagen, repair synthesis is likely to be largely complete in a day or so.

Hemoglobin Alkylation

Measuring alkylated amino acids in hemoglobin has been proposed as a means of monitoring exposure to alkylating agents. This is far from a genetic event, but protein alkylation strongly implies the concurrent alkylation of nucleic acids, a potential first step in the production of mutations. In one method, hemoglobin from circulating red blood cells is extracted and analyzed for the presence of alkylated derivatives of histidine or cysteine. In another method, the loss of histidine or cysteine is measured as the ratio of these amino acids to nonalkylatable amino acids. These procedures have only been reported for mice injected with known mutagens, and on human red blood cells treated with mutagen in vitro (and in only two laboratories); there are no reports of their practical application to humans. All reported work has been on pooled hemoglobin samples, and the reported sensitivities of the methods are low. Before these techniques can be considered for routine monitoring, extensive validation studies would have to be performed to standardize protocols, measure reproducibility and the individual variability, and determine the sensitivity of the method in detecting alkylation by diverse mutagens. Hemoglobin alkylation is thus experimental in terms of its applicability to human monitoring.

The assessment of the degree of alkylation in man requires the development of nonradioactive methods using samples acquired with a minimum of risk to the individual.

The possibilities for using hemoglobin from red blood cells as a chemical dosimeter have been explored by Ehrenberg and coworkers in a series of experiments on mice using directly alkylating agents [13-15], as well as agents that need metabolic activation [14-16]. After treatment of the animals with the radiolabeled compounds, the degree of alkylation of *guanine-N*-7 of DNA and of cysteine-*S* and histidine-*N*-1 and -N-3 in proteins from different organs were determined parallel to the determination of alkylation of cysteine and histidine of hemoglobin. It was shown that for small, rapidly diffusing and sufficiently stable molecules, such as ethylene oxide (biologic mean-life, about 12 minutes [13-14]), and methyl methanesulfonate (biologic mean-life, about 40-50 minutes [15]), the erythrocyte dose is representative of doses of other organs, including the liver and the testes. For more unstable compounds, such as the reactive metabolites from vinyl chloride [17], and dimethylnitrosamine [14], the gonad doses were considerably lower than the erythrocyte doses.

The accumulation of alkylated groups in hemoglobin and the relatively large amounts of hemoglobin that may be isolated from one blood sample and analyzed, together with an application of sensitive analytic techniques, make it feasible to determine the small quantites of alkylated amino acids that may be formed as a consequence of exposure to genotoxic compounds in the environment.

Work in progress with the compound methyl bromide indicates that, due probably to the lipophilic character of this compound, the degree of alkylation of DNA is much smaller than expected from the reaction kinetic data, and that a "correction factor" should be introduced when estimating DNA-alkylation from hemoglobin data. Other factors that modify the alkylation pattern and factors that affect the biologic response include: steric factors, charge, bifunctionality, high reactivity, interactions with other compounds, and repair mechanisms.

Continued studies aimed at an evaluation of such factors and further development of the techniques to determine the degree of alkylation of hemoglobin should provide a basis for meaningful risk estimates for a larger number of mutagenic compounds [18-20].

Although this system is very agent-specific and requires additional development, it may ultimately prove useful where identity of the agent is known, such as in occupational exposures.

DNA Adducts

Many chemicals alter DNA bases, either degradatively or by adduction. DNA extracted from any readily obtained human tissue can be analyzed for the presence of chemically induced abnormal bases. Some products, such as 7-alkypurines involving small adducts (e.g., methyl or ethyl), are innocuous, but may serve as general indicators of DNA damage. Adducts at most other sites, and especially bulky adducts which interfere with subsequent DNA replication, are highly genotoxic unless efficiently repaired; and certain base alterations are directly mutagenic without a requirement for subsequent DNA replication. Except where noted, the following analytic methods are readily available in research laboratories, but will require several years to adapt to standardized screening with the concomitant development of a human data base.

Many altered bases trigger excision repair, which may be detected as a consequence of associated strand breakage or UDS (see above); however, repair mechanisms involving glycosylases and insertases have recently been discovered; these do not involve strand breakage and gap formation, and it is unclear what fraction of DNA damage is susceptible to this type of repair. Damages in uncleaved DNA may be detected either by the use of endonucleases which cleave at the sites of damage, generating breaks and gaps which can be detected as such, or by extensive (and preferably enzymatic) digestion of damaged DNA followed by high-resolution analyses for aberrant bases by fractionation techniques (e.g., paper, column, thin-layer and high-pressure liquid or gas chromatography) and identification techniques (e.g., mass spectrography) where amounts are exceptionally small. It should be noted that available DNA degradative methods may not always be suitable, and that radiolabeling to assist detection of rare products will generally be impossible in the case of human tissues.

DNA adducts have been measured in human cells and tissues exposed in culture, and are readily measured in tissues and sperm of laboratory animals [13]. Sensitivity, in such experiments, currently requires radioactive adducts, an asset that would not be available for general human use. Feasibility in the human is, therefore, uncertain but should improve, perhaps to the point of applicability as methods improve for chemical analysis of specific adducts [21]. Sperm would be prime test targets because of their genetic relevance, their availability, and the accessibility to adduct formation during their final preejaculatory weeks when repair is no longer present.

DNA Strand Breaks

Many chemical lesions in DNA can produce either single- or double-strand breaks, both directly or as a result of subsequent excision repair. Such damage can be measured in any readily sampled human tissue, including circulating white blood cells of all types, skin biopsies, and biopsies from more profoundly invasive procedures. A plethora of analytic methods is available, most of which are in many laboratories and are well standardized. Validation studies on animal models are in most cases reasonably well under way, but validation in humans and the development of a background data base will, again, require several more years. The viscosity of DNA is reduced by both double- and single-strand breaks, and can be measured by rotating cylinder viscometry with high sensitivity; however, that is a rather specialized technique. A much more widely available method is ultracentrifugation, which usually takes the form of sedimentation velocity (through gradients or not) or band sedimentation centrifugation. Appropriate manipulation of the method allows discrimination between single- and double-strand breaks, which may be useful because double-strand breaks more likely may be precursors of chromosomal mutations at low doses. In the special case of small circular DNA molecules, such as the genomes of mitochondria, equilibrium density gradient centrifugation in the presence of ethidium bromide is a particularly useful and sensitive assay for both types of breaks. Electron microscopy to characterize length distributions, particularly with the use of enzymes or other proteins which recognize single-strand breaks, is also useful but is rather specialized. A recent method, but one which is very sensitive and is already widely available, is gel electrophoretic determination of molecular weight distributions. Finally, several methods are available to differentiate DNA according to both strand length and presence of single-strand gaps in mainly double-stranded DNA (a consequence of single-strand breakage followed by enzymatic gap formation) by differential binding to membrane filters or columns of various types. Other methods which have been used in the past but are of insufficient sensitivity for human monitoring are ultraviolet spectrophotometry (including hyperchromic effects and decreases in melting temperature), light scattering, flow dichroism, and recognition of DNA termini by various proteins. A general limitation on the use of any of the above techniques is the ease with which breaks are introduced into DNA by just the sorts of manipulations which are required to isolate DNA from human cells.

C. MEASUREMENT OF SOMATIC CELL MUTATIONS

HPRT Specific Locus Test in Peripheral Lymphocytes

One of the most useful systems in the study of cell biology has been the hypoxanthine phosphoribosyltransferase (HPRT) locus, which is X-linked in mammals. Mutations to loss of activity of this enzyme cause resistance to the guanine analogs 8-azaguanine (AG) and 6-thioguanine (TG). Thus, wild-type HPRT$^+$ cells can be selected against by incorporation of one of these analogs. Conversely, HPRT$^+$ cells can be selected for (i.e., revertants from HPRT$^-$ cells) in the presence of HAT (hypoxanthine-aminopterin-thymidine) medium. It should be possible to select for HPRT$^-$ cells in normal HPRT$^+$ persons with one of the analogs; this was one of the first systems used to detect spontaneously occurring mutants in freshly cultured cells from humans [22, 23].

The frequency of TG-resistant cells has been reported to be on the order of 1×10^{-4} among peripheral blood lymphocytes of normal persons [24]. The frequency is generally higher among cancer patients under therapy with known mutagens. This system, therefore, offers the possibility of detecting somatic mutations through selection in lymphocyte cultures. Much has yet to be elucidated about the variables that influence the results. Nevertheless, the ability to measure mutation in somatic cells and to document the nature of the mutation makes this a promising system.

Hemoglobins

The red cells of an individual contain almost pure hemoglobin. In the human population there are many different heritable mutations that lead to individuals whose red blood cells contain variant hemoglobin. Some of these hemoglobins differ sufficiently from normal hemoglobin to produce monospecific antibodies. By attaching a fluorescent molecule to the monospecific antibodies against a certain variant hemoglobin, one should be able to localize the few red blood cells that contain this different hemoglobin.

The first successful variant hemoglobin system was developed with the sickle cell hemoglobin in humans [25]. In a normal human, the frequency of red blood cells that contain sickle cell hemoglobin is 10^{-7}. The mutation from hemoglobin A (HbA) to hemoglobin S (HbS) is a specific base pair substitution and may not occur with many mutagens [26]. It is, therefore, very important to expand the type of mutations that are scored. Monospecific antibodies have now been produced to hemoglobin-C and -E, both base pair substitutions; to hemoglobin Wayne and Cranston, both frameshift mutations, and to hemoglobin Constant Spring, which is a terminator mutation.

The frequencies of the mutant cells are so low that handscreening for the mutant cells is not feasible, even for a modest screening program. Development of automatic counting systems is in progress. The reaction of the monospecific antibody with the variant cell is not proof that this cell originated by mutation, since unknown epigenetic changes may mimic the mutant. Therefore, the genetic origin of the mutant has to be established, which can be done by isolating enough mutant cells to obtain biochemical proof of the mutant protein.

Theoretically, this technique is as genetically sound as any to be used as a general monitoring system for the human population; but in summary, several technical and theoretic developments remain to be accomplished.

1. Establish the mutational nature of variant cells;
2. Develop the automated counting systems;
3. Validate the response in humans who accidentally or therapeutically have been exposed to known mutagens;
4. Develop animal modeling system for interpretation of the data;
5. Produce monoclonal high affinity antibodies to the various variant hemoglobins.

D. MEASUREMENT OF GERM CELL MUTATIONS

LDH-X Variants in Human Sperm

Most systems for detecting heritable mutations in mammals require the study of offspring of the exposed individuals. Several thousand offspring must be screened to obtain any meaningful data. One alternative to monitoring progeny of exposed individuals is being developed. It is the direct screening of sperm from exposed individuals for point mutations through the use of an appropriate marker protein. The advantage of such a single cell screening system would be that each cell, rather than each individual, could be scored for a mutational event. Such a system could be used to a) monitor the human population for genetic damage caused by exposure, and b) screen chemicals for their mutagenicity.

An immunologic system, based upon the use of antibodies against lactate dehydrogenase-X (LDH-X), appears promising. An antibody population has been prepared that binds rat LDH-X but not mouse LDH-X [27-31]. In a sandwich fluorescent antibody technique, this antibody stains rat sperm, whereas mouse sperm remains unstained. If an LDH-X gene undergoes mutation such that the amino acid sequence of the enzyme is altered so that it will bind the anti-rat LDH-X antibody, sperm containing such mutant molecules can be identified by immunofluorescent stain. From DBA/2 control mice, 72 fluorescent sperm were found among 167×10^6 cells, giving a spontaneous frequency of presumptive mutations of 0.43×10^{-6}. The frequency of these events increases linearly upon treatment of the mice with increasing dosage of procarbazine [32]. The experiment with procarbazine was repeated and a similar dose vs response relationship was obtained. Experiments using mitomycin-C and ethyl nitrosourea (ENU) are in progress to obtain dose vs response relationships.

Amino acid sequence and antigenic site analyses of the LDH-X from mouse, rat, and human are required for better characterization of the presumptive mutational events. Until this information is available, it remains unclear just what types of changes are being detected and how they relate to modification of DNA.

Antibodies are being made that will detect similar mutational events in human sperm.

Although antibodies required for the mutation study have been produced, and very encouraging results are being obtained in terms of dose vs response relationship, it seems impractical to think that this system can be applied to human mutation monitoring purposes for perhaps another two years.

E. MEASUREMENT OF GENE MUTATIONS IN F_1 PROGENY

Reproductive Potential

A commonly used test in experimental animals is the dominant lethal, generally assumed to detect the induction of chromosomally abnormal sperm. There are reports of loss of fertility on exposure of human males to various chemicals. It is difficult to attribute such effects to a genetic event rather than a cellular effect. An increase in genetically defective sperm, detected by diminished fertility, would require a very large and carefully controlled study. The use of birth control and the changing social patterns of family size virtually rule out evidence on fertility as useful in monitoring mutagenesis.

Phenotypic Effects

Much thought has been directed to the possibility of selecting sentinel phenotypes that result, at least in part, from mutation and that can be used to measure increases in mutation rate. Only a small number of single gene traits have been identified that would be especially useful for this purpose. However, since the frequency of mutation is so low, a very large population is necessary to estimate the frequency of new mutants or to detect a change in that frequency. This could be solved in part if complete and readily accessible health records existed for large political units. Newcombe [33] has studied the problem from the point of view of computer linkage of public records.

One shortcoming of this approach is that the time between the mutation and the conception might be long, making it very difficult to relate an increase in inherited defect to the responsible agent. On the positive side is the fact that clinical defects are part of the concern for chemical mutagens, and the relevance to human risk and health load is direct.

Electrophoretic Variants in Blood Serum and Erythrocyte Proteins

In recent years, it has become possible to base the study of point mutation in man (and other animals) on changes in defined proteins. This has the advantage over the use of "sentinel phenotypes" in that the number of useful markers is potentially much larger, and numerous biochemical indicators can all be examined on the basis of a single blood sample; whereas a full evaluation of the relatively few (15-20) "sentinel phenotypes" requires repeated examinations of the cohort of children under consideration.

The technique of choice at the present is one-dimensional electrophoresis, using starch or polyacrylamide gel as the supporting medium. At least 35 different proteins of the blood serum and erythrocyte are suitable for accurate screening for variants by this technique (Table 2). Recently, this technique has been applied to the study of the genetic effects of ionizing radiation from atomic bombs [34]. Since only about 1 in 7 biochemical variants of these proteins is detectable by electrophoresis, only a minority of biochemical variants can be detected by this approach. Cost wise, the expenditure per locus screened is in the same range as the approach employing "sentinel phenotypes," although a precise comparison is

Table 2. List of proteins currently being screened by electrophoresis in the Department of Human Genetics in Ann Arbor for the occurrence of mutation.

Protein	Symbol	E. C. No.
Albumin	ALB	
Ceruloplasmin	CRPL	
Haptoglobin	HP	
Transferrin	TF	
Acid phosphatase	ACP1	3.1.3.2
Adenosine deaminase	ADA	3.5.4.4
Adenylate kinase-1	AK1	2.7.4.3
Carbonic anhydrase-1	CA1	4.2.1.1
Carbonic anhydrase-2	CA2	4.2.1.1
Esterase A	ESA	3.1.1.1
Esterase B	ESB	3.1.1.1
Esterase D	ESD	3.1.1.1
Galactose-1-phosphate uridylytransferase	GALT	2.7.7.10
Glucose phosphate isomerase	GPI	5.3.1.9
Hemoglobin α	HG α	
Hemoglobin β	HG β	
Hemoglobin δ	HG δ	
Isocitrate dehydrogenase	ICDS	1.1.1.42
Lactate dehydrogenase A	LDHA	1.1.1.42
Lactate dehydrogenase B	LDHB	1.1.1.42
Malate dehydrogenase	MDH	1.1.1.37
Nucleoside phosphorylase	NP	2.4.2.1
Peptidase A	PEPA	3.4.1.1
Peptidase B	PEPB	3.4.1.1
Peptidase C	PEPC	3.4.1.1
Peptidase D	PEPD	3.4.1.1
Phosphoglucomutase-1	PGM1	2.7.5.1
Phosphoglucomutase-2	PGM2	2.7.5.1
Phosphogluconate dehydrogenase	6PGD	1.1.1.44
Triosephosphate isomerase	TPI	5.3.1.1
Glucose-6-phosphate dehydrogenase	G6PD	1.1.1.49
Glutamic oxaloacetic transaminase, soluble	GOTS	2.6.1.1
Hexokinase-1	HK1	2.7.1.1
Hexokinase-2	HK2	2.7.1.1
Phosphoglycerate kinase	PGK	2.7.2.3
Uroporphyrinogen I synthase	UPS	4.3.1.8

impossible because it is neither clear how many loci are subject to mutation resulting in a specific "sentinel phenotype" nor what proportion of mutation at those loci associated with "sentinel phenotypes" is detected by the morphologic approach.

Techniques are becoming available for screening human enzymes for variants resulting in extreme loss of enzyme activity, and in alterations in enzymatic thermostability. The former, conveniently studied with the use of the centrifugal fast analyzer [35, 36], has been found in a study of a series of 10 enzymes to have a frequency about 3 times as great as electrophoretic variants [37]. The latter, whose detection is more time consuming than the detection of nulls, is also about 3 times as frequent as electrophoretic variants [37]. The "efficiency" of indicators of this type for the study of mutation, as contrasted with electrophoretic variants, will be determined by the relative frequency of newly arisen mutants among the variants, on which point there is as yet no information.

A technology just becoming available involves the use of 2-dimensional polyacrylamide gel electrophoresis, in which the first dimension is determined by isoelectric focusing of a disk gel and the second dimension, obtained by applying the disk gel to a polyacrylamide slab, results from standard electrophoresis [35, 38-40]. Application of this technique to blood serum, or an erythrocyte or lymphocyte lysate, results in the visualization of as many as 1000 protein moieties when a sensitive silver stain is employed. It is not yet clear how many of the proteins visualized will be sufficiently reproducible (and have the other necessary characteristics) to be useful in studies of mutagenesis, but the number should not be less than several hundred. Inasmuch as preliminary studies have suggested a high degree of constancy, it would appear that much of the reading of these gels can utilize computer programs specifically developed for the analysis of these images, with operator intervention into the analytic process whenever a protein candidate for mutation is encountered. (The logical problems involved in developing the necessary programs are much simpler than in automated karyotyping.) The actual verification of a suspect protein as a mutant will require a variety of supplementary techniques, such as the use of monospecific antibodies to isolate the protein and high-pressure liquid chromatography and protease digests to characterize the protein.

Finally, one must consider the study of induced mutation at the nucleotide level, using restriction-site endonucleases to scan for alterations in short nucleotide sequences or gains or losses of DNA sequences in excess of 100 nucleotides. While this approach is currently not as efficient as electrophoresis, especially of the 2-dimensional type, the technology is advancing so rapidly that it would be presumptive to foreclose this as an option. A disadvantage of this technology is that, in contrast to the study of proteins whose abnormalities can often be directly related to disease processes, the bulk of the mutations detected by this approach will not involve translated DNA, and so their biomedical significance will be unclear.

The entire biochemical approach to the study of germinal mutation is in a state of flux and rapid evolution. There is no doubt it is a viable technology, in both the human and experimental setting [41, 42]. How it will compare in cost to sentinel phenotypes is not clear. Much depends on the circumstances under which it is pursued. For instance, in West Germany the search for mutant hemoglobins using the blood stain submitted for the diagnosis of phenylketonuria is quite inexpensive [43]. A particular advantage of the 2-dimensional approach is that in theory, by combining information available from lymphocytes, erythrocytes, serum, hair follicles, and saliva, one could score as many as 1000 different proteins for mutants, a possibility which outstrips any other in the amount of information to be contributed by a single child born to parents exposed to a potential mutagen.

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The Implementation of These Guidelines

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My assigned responsibility is to attempt to summarize what has been a very intense two days, with particular reference to defining the interrelationships between the three sets of guidelines which have been developed. In the usual sequence, one would follow that sort of summary with some comments about the scientific and financial demands of these protocols. It occurred to me, however, that it would be worthwhile to reverse the usual order of events, and open my remarks with a discussion of the costs of *not* developing and then pursuing the implementation of a proper protocol to evaluate the impact of current exposures to potential mutagenic and reproductive hazards.

THE COST OF INADEQUATE EVALUATIONS OF POTENTIAL MUTAGENS AND TERATOGENS

For more than 50 years, students of genetics and teratogenesis have been writing voluminously concerning the effects of radiation and chemical agents in experimental systems, and alluding to the implications for humans. The public, especially during the last decade, has been attentive. The resulting public concerns have been manifested in two very concrete fashions. On the one hand, it has become mandatory to screen new pesticides, herbicides, pharmaceuticals, etc. for mutagenicity and teratogenicity while they are still under development, with the objective of intercepting those which appear particularly hazardous. On the other hand, on the basis primarily of observations on non-human model systems, an effort has been made to set permissible limits to exposures to a variety of man-made agents in the environment. Historically, both of these developments have their roots in an effort to screen the public from carcinogens, but as the extensive overlap between mutagens and carcinogens has emerged, increasingly the two types of screening have become synonymous, a point to be returned to later.

So far as I am aware, no responsible person wishes to quarrel with the thrust of either of these developments. Unfortunately, however, as scientists and administrators alike seem to agree, there is a wide band of uncertainty in our efforts to screen out mutagens and/or extrapolate from an experimental setting to effects on human populations. These uncertainties permit persons of good intent to develop quite different viewpoints; a situation abetted by conflicts in the interpretation of such sets of human data as are available. We are not talking about differences to the right of the decimal place, but about an order-of-magnitude difference in the positions responsible parties have adopted. The uncertainties are such that the National Science Foundation, in its first Congressionally mandated effort to provide the Congress with a 5-year projection of the nation's research needs, targeted improving the science and technology base for risk assessment as one of its 5 high priority items [1]. A more concrete manifestation of these same concerns is the recent Toxic Substances Control Act (15 USC 2601), requiring EPA to formulate standards for the development of test data relating to human genetic effects.

The humanitarian aspects of a proper appraisal of the risks in question are obvious. In this connection, I assume familiarity on the part of the audience with the voluminous committee and other reviews of the impact of spontaneous mutation on the health of a population and the consequences of an increased mutation rate [2-9].

Not quite so obvious as the humanitarian have been the financial aspects of the appraisal problems. Unjustifiably stringent regulations upon industry will place our nation at a competitive disadvantage among industrialized countries, at a time when it is clear that even at best we face some difficult years. The pronouncements of the new Administration suggest a concern that regulations concerning environmental pollution have become overly stringent, but every fresh revelation of some of the unnecessarily careless waste disposal practices of the past only further stiffens public concern. The longer the necessary resolution of the strong differences of opinion which exist concerning this matter is drawn out, the greater the expense to us all. I am sure I do not have to emphasize what failure to reach a clear position regarding nuclear energy has cost this country. Furthermore, continuing uncertainties will encourage further individual and class-action legal initiatives for damages which already cumulate to a very large sum, initiatives where the basis for a just settlement is very obscure. Although most of these actions now relate to carcinogens, I suspect that during the next decade the suspicion of mutagenic and teratogenic damage will come increasingly to the fore. There is ample legal precedent for awarding damages on the presumption of injury; in the absence of sound data — in this case, probabilities of genetic or teratogenic damage — the courts, which *must* reach a position, will be seriously handicapped in the discharge of their responsibilities. Understanding the possible carcinogenic, mutagenic, and teratogenic implications of the many environmental agents to which we are exposed is not an academic exercise but one of the two or three most important challenges to biomedicine in the next decade. Decisions entailing billions of dollars are involved. In this connection, I must once again chide many of my genetic colleagues for talking big but thinking small. We have spoken copiously of the possible risk to future generations of environmental contaminants, but, in my opinion, thus far failed either to formulate or request funds to pursue proper epidemiologic studies. I shall speak to the one exception to that statement later. Hopefully, this Conference will be seen in retrospect as the occasion when geneticists, stimulated by the ineptitude with which the issues created by the Love Canal episode were pursued, first laid out to Government what had to be done in the nation's interest.

COORDINATING OUR THREE SETS OF GUIDELINES

The three sets of guidelines with which we have been presented are each the product of much thought and experience, and it is certainly not my intention to critique them. Rather, I would like to speak to the issue of their coordination in any real-life situation. By and large, the guidelines of Panel I deal with studies of *somatic* effects which, if positive, create the *presumption* of transmitted genetic damage but by no means prove it, much less permitting an estimate of its magnitude. The guidelines of Panel II deal in part with the detection of both mutagenic and teratogenic influences, and, if the population is still exposed to the agent(s) in question at the time of study, a positive finding with respect to some indicators, such as congenital defects, would not necessarily permit a decision as to which kind of influence is involved. In addition, the guidelines of Panels II and also of III deal with a mixture of somatic and true genetic end points, using the word 'true' in the sense of that which can be transmitted across generations. Now, the costs of the studies of such somatic cell end points as chromosomal damage, SCEs, and frequency of mutant erythrocytes or leukocytes are far less than studies of such indices of transmitted genetic damage as certain types of congenital defects, chromosomal abnormalities, or biochemical traits. It will be administratively tempting to implement the former but not the latter. This would be a tragic mistake. *At present, we have no sound basis for extrapolating from somatic cell end points to genetic effects.* Perhaps the outstanding example of this stems from the studies in Hiroshima and Nagasaki of atomic bomb survivors and their children. The demonstration of chromosomal damage in survivors [10-12] as well as increased rates for a variety of malignancies (reviewed in reference [13]) has been clear and unequivocal. However, the demonstration of genetic damage in their children has been much more equivocal, as we shall see later. It is essential that as the monitoring of populations is initiated, we must build the intellectual bridges, the conversion factors, between the various kinds of studies envisioned in the three sets of guidelines, and it has to be done with human data.

In the face of the labor involved in building these bridges, there are sometimes suggestions that one use a mixture of human epidemiologic and non-human experimental data in setting risks to human populations. If there were any lingering doubts about the absolute requirement for human data all the way, I would suggest they should be dissipated by our recent analysis of the accumulated data concerning children born to survivors of the atomic bombings of Hiroshima and Nagasaki [14]. This analysis differs from its predecessors in the use it makes of individualized estimates of gonadal dosage, only available since 1978, and the incorporation of recently acquired sets of data. Although our colleagues and ourselves have at one time or another attempted to collect data on all the aspects of pregnancy termination mentioned by Panel II, for this new analysis we have pursued the more qualitative and, in our opinion, reliable of the available indicators, namely, 1) untoward pregnancy outcomes (stillbirth, congenital defect, and/or death during the first postnatal week), 2) death among live-born infants up to a mean age of 17, 3) sex chromosome aneuploidy, and 4) mutation resulting in electrophoretic variants of a battery of 28 proteins. For each indicator, the difference between the children of exposed and unexposed is of a nature to suggest genetic damage to the parents, but none of the differences begins to approach significance. On the thesis that the radiation exposure did in fact have a genetic effect, a thesis justified by a great body of experimental data, we have undertaken to estimate the genetic "doubling dose" for this type of radiation exposure, i.e., the amount of radiation producing the same frequency of mutation as occurs spontaneously. This estimate, of some 160 *rem,* is approximately 4 times greater than the estimate derived from the specific locus test system in the mouse, which latter estimate has until now largely guided our thinking (see reviews cited earlier). Incidentally, with reference to the guidelines of Panel II, we no longer employ changes in the sex ratio in the evaluation of a genetic effect, because of the difficulty in establishing *a priori* expectations in view of the possible induction of sex chromosome aneuploids and the potential role of the Lyonization phenomenon in obscuring the expression of sex-linked mutations in the human female.

I have heard a lot of comments the past 2 days, to the effect that we do not know how to monitor human populations for transmitted genetic damage. Those comments manage to overlook the fact that over the past 35 years, under the aegis of the NAS, financed by the (now) DoE, a protocol has been developed, finally yielding the data I have just summarized. It is not perfect, but neither is it pie-in-the-sky. Unfortunately, it is so difficult and laborious to implement that I sense a real reluctance to face up to what it takes. The sooner we get cracking on what we can do right now, the sooner we meet the moral responsibility we have brought upon ourselves by our frequent presentations to the public of the potential genetic dangers with which they are surrounded.

Should our analysis of the Japanese study stand — and you may be sure it is being subjected to very critical review at present — then it will forever document the difficulties in extrapolating with any great accuracy from contrived laboratory situations to effects in heterogeneous human populations. Lest there be misunderstanding, I have been and remain a strong proponent of the usefulness of animal experimentation in establishing the broad outlines of a situation, but believe we absolutely must have human data for the necessary fine tuning. Furthermore, should a positive finding emerge from a survey of human populations, it would and should be a strong stimulus to experimental programs designed to clarify the nature of the agents involved.

Having now stressed the need for establishing, insofar as possible, the interconversions between the various end points covered by the guidelines, let me emphasize that once the basis for extrapolating from somatic to genetic effects in humans has been established, then the more costly and elaborate studies of transmitted genetic damage may be no longer necessary, or necessary only under particular circumstances. But without the somatic-genetic linkage, we will never know the genetic significance of an increase in visible chromosome damage or SCEs in somatic cells, or of mutant erythrocytes, or of abnormally-shaped sperm. This salient truth is often lost sight of. For instance, I note that Brusick [15], in his recent generally excellent book entitled "Principles of Genetic Toxicology," in the chapter on Environmental and Human Monitoring, says nothing about the study of children born to individuals thought to be at risk of a genetic insult!

Incidentally, the one point on which I would be critical of Guidelines I and II is failure to emphasize sufficiently the need for establishing decay curves following a mutagenic insult, for such phenomena as gross chromosomal damage, SCE frequencies, or sperm abnormalities. The populations which will come under study will be at varying intervals from the genetic insult, and the ability to extrapolate from somatic genetic end points to germinal requires intimate knowledge of that decay curve.

COMMENTS ON THE DESIGN OF FIELD STUDIES

There are in principle just two designs for field studies. On the one hand, one can establish well-defined samples ("census tracts") of the general population which are then continuously monitored for changing mutation rates, most conveniently on the basis of appropriate examination of newborn infants and their parents. Pilot studies on how this best can be done, employing various biochemical markers of mutation, are being performed in Michigan by Neel et al [16], and in West Germany by Vogel and Altland [17]. A basic problem with the prospective census tract approach in any highly industrialized country such as the United States, Japan, or West Germany is that even if no upward trend in mutation rates is detected in the future, it can be maintained that an undesirable increase in the mutation rate has *already* occurred, which this approach might serve to legitimize. On the other hand, one can undertake a "worst cases" analysis, in which one identifies groups of individuals at presumed high risk to a mutagen and pursues appropriate studies on their children, plus the necessary control studies. The chief experience to date with this approach stems from the joint United States-Japan follow-up studies in Hiroshima and Nagasaki, to which I have already alluded. Dr. Miller has spoken in this conference primarily to the problems in identifying populations at high risk of chemical exposures within the United States. With respect to radiation exposures, a recently completed study commissioned by the U.S. Nuclear Regulatory Commission [18] suggests the most appropriate groups within the United States to be workers in health services, nuclear power plants, Department of Energy-supported facilities or nuclear shipyards, and people who received routine pre- and postnatal radiologic examinations. In addition, three groups receiving special environmental exposures were identified; namely, people exposed to fallout from the Nevada Test Site, plutonium from the Rocky Flats Plant in Colorado, or radon from phosphate-rich lands in Florida. I need not comment on how critical the identification of suitable control groups becomes.

The exact magnitude of the study which must be envisioned depends entirely on the magnitude of the anticipated effects. Here of course is where controversy intervenes. On the basis of our follow-up studies in Hiroshima and Nagaski, as well as the experimental data, I do not believe that current U.S. exposures to mutagens and teratogens, even in high-risk groups, have had a substantial impact on the end points we have been discussing. We are thus looking for small effects, usually in as large a sample as can be assembled. This requires the utmost sophistication in epidemiologic design, a sophistication which some feel has been lacking thus far in many of the federally funded research projects in this area (see, for example, Committee on DoE Research [19] or the voluminous recent exchanges re the Love Canal fiasco). Let us return briefly to this matter of determining the necessary sample size for a study, so well discussed by Panel II. Let us agree that any study should certainly not fail to detect an increase in mutation rates of 50% or greater. Elsewhere — as an illustrative calculation — I have suggested that, given the restriction of a Type I error of 0.05 and a Type II error of 0.20, and employing protein phenotypes, for which the assumed baseline average mutation rate was 10^{-5}/locus/generation, it would require 2 samples of some 7,000,000 observations each to detect a 50% increase in mutation rates [20, 21]. Vogel has presented similar estimates [17, 22]. Of course the number of children contributing to that sample depends on the number of bits of information per child. If both parents are exposed and there are 50 indicators per child, the sample size is 70,000 children. If with the new 2-dimensional electrophoretic technologies one can reach 500 observations per child, then only 7,000 children will result in 7,000,000 observations. If the indicator were a gross chromosomal defect, which for the calculation can be assumed to have a mutational origin of approximately 4 x 10^{-3} among conceptuses reaching the 20th week of gestation (E.

Hook, personal communication), then the 2 sample sizes are approximately 19,000. Now the numbers apply to people. In either case, the numbers decline rapidly as the magnitude of the expected differences increases.

These are formidable numbers. They can most conveniently be obtained in studies of "census tracts," if these are made large enough; but few geneticists feel such populations are at risk of 50% increases in mutation rates in the years ahead. For the "high-risk" populations, one might be satisfied to detect within the previously mentioned statistical limits, an increase of at least 100%. Now the necessary numbers will be less than half the preceding figures but even these numbers are difficult to assemble from high-risk populations, since the number of available children from any one group is, fortunately, relatively small from the genetic standpoint. One way to increase the numbers is to make the studies retrospective as well as prospective. Retrospective studies must be conducted with exquisite care that the indicators not carry a poorly defined increased risk of early death which always complicates documentation of an effect. Under these circumstances the most suitable, the "cleanest" indicators of *transmitted* genetic damage are such subclasses of chromosomal abnormalities as sex chromosome aneuploids and reciprocal translocations, and the biochemical traits, some of which (the "nulls") while usually associated with serious disease when homozygous, do not seem to be deleterious in the heterozygous state in which they would present as mutations.

While in principle such data can be collected (and are being collected) at the present time, I would suggest that the prospects for methodologic improvements are excellent, and funding designed to make the collection of cytologic and biochemical data more efficient would be well justified. I refer especially to the development of the 2-dimensional polyacrylamide gel electrophoretic techniques which permit us to visualize 500 to 1000 proteins from readily accessible biologic samples. Coupled with automated image analysis and the power of monoclonal antibodies, high-pressure liquid chromatography, and rapid protein sequencing techniques, this technology has already ushered in a new era in our ability to identify and characterize protein variants in man — and protein variants are what we are all about. In this connection we should not overlook the possibility that recent developments in the ability to characterize variation in DNA may soon permit us to study mutation at this level, but, to understand the impact of the phenomena being measured on the proteins through which genetic damage is manifested, such studies, at least at the outset, need to be correlated with appropriate studies of selected proteins. By now it should be clear I cannot subscribe to the generally negative tone of the BEIR III report (1980) concerning the prospects for human data, as reflected in the following statement:

> "Few human data are — or are ever likely to be — available in the variety and depth needed to give us direct and simple answers to our questions. Hence, to estimate human genetic risks, we must find ways to apply information from other sources." (p. 94)

What other sources? I believe that the Genetics Sub-committee was simply out of touch with current technical developments.

There is an important aspect of the design of field studies which must not go unnoticed. This is the issue of sponsorship and direction, which is really the issue of credibility. Very simply stated, many advocacy groups have lost confidence in the objectivity of any government-sponsored study. I strongly urge that the implementation of the guidelines we are discussing demands the creation of a "blue ribbon" advisory committee, whose membership will include government officials and investigators, university professors, and representatives of industry and public interest groups. The task of the chairman of such a heterogeneous group will be difficult, but only under these circumstances will there be public acceptance of any findings. I do not mean to suggest such a committee simply for public display; it must have the expertise to pass judgment on the final design of the necessary epidemiologic studies. It is unlikely that *any* study will ever encompass all the possibilities in these protocols; there are some difficult choices to be made in the implementation of these various suggestions.

COMMENTS ON THE COST OF PROPER STUDIES

Although budgeting for various aspects of these guidelines is not part of the charge of this group, I believe it is incumbent upon us to provide approximate estimates. The costs of any proper study fall under 3 categories: front-end (collection) costs, laboratory costs, and data management costs. The front-end costs of any "census tract" approach are small, if one can use obstetric services as the focal point of the study with the opportunity they present for an in-hospital evaluation of the child, and the collection of a blood sample from its parents and itself. The front-end costs of a "worst cases" approach, which involves tracing persons with a mutagenic/teratogenic exposure, are substantially higher but presumably would be shared with an effort to evaluate the somatic effects of some particular carcinogen-mutagen, and would be a minor aspect of that effort. The laboratory costs depend of course on the end points. The costs of studies yielding the necessary sample size with respect to SCEs, chromosome damage, or in vivo somatic cell mutations are well within the bounds of funded research projects. So are the data management aspects of the undertaking. Larger sums of money are involved in the search in children for chromosomal or biochemical mutations. On the basis of the experience in Japan plus developments in laboratory technology which I believe to be imminent, I would set the laboratory costs of chromosomal or biochemical studies yielding the numbers cited earlier at 15 to 25 million dollars. The cost of the chromosomal studies is highly influenced by the degree of resolution one seeks — it will cost twice as much to do the kind of chromosomal "banding" studies which will facilitate the detection of small chromosomal lesions as to do studies directed simply towards the detection of aneuploidy. These costs are a fraction of what is spent each year in testing chemicals for carcinogenicity. Incidentally, it would seem of the utmost importance that from the outset, a feature of the research design be an effort to evaluate the cost-effectiveness of the component parts of any project.

SOME STATISTICAL CONSIDERATIONS

Concern is often expressed that even a very large study will be negative, in the sense of not yielding "statistically significant" results, and that this will create the presumption of no genetic effect where in fact one exists. In my opinion this concern is based on failure to understand the strategy of monitoring. There is no such thing as a negative study. In the first place, given present concerns, any substantial contribution to the available data is worthwhile. While proving the absence of any effect whatsoever is never practical, surely under present circumstances it is better to have some data on United States populations to point to than no data. Furthermore, the findings of the monitoring programs, even if they fail to demonstrate statistically significant effects, can always be used to set upper limits at specified confidence levels on the magnitude of a true effect which may have gone undetected in the statistical sense. Finally, now, if one accepts the presumption that there was a teratogenic-genetic effect, as for example we did in the Japan study, then one can always, taking the findings at face value, determine what they suggest regarding the exposure(s) necessary to double the end point for the phenomena in question, even in the absence of statistical significance.

The greatest statistical problem in a large monitoring program will be false-positive findings. This issue is well treated in Guidelines II, but is important and I believe it deserves reiteration. Let us assume that funding is available to do detailed studies on some 4 populations which appear to have had an unusual exposure to a mutagenic/teratogenic agent. Consider that within each of the 4 relatively high-risk populations, the parents are examined for the frequency of chromosome damage and SCEs, and their children for congenital defects and chromosomal and biochemical mutations. In this 4 x 5 matrix, by chance alone there should be one "significant" finding. Any use of this apparent "finding" will result in a major overstatement of the magnitude of the effect (cf. [23]). It must be clearly agreed from the outset that in this example, or a comparable situation, opinions concerning damage will be based on the totality of the data. The statistical methodology for dealing with this situation has recently received considerable attention in the context of seeking associations between histocompatibility antigens and disease states. Here, too, one does many tests in many populations. A rough rule-of-thumb procedure for the evaluation of a single test is to multiply the probability of exceeding the test criterion by the number of separate tests of hypothesis executed [24,25]. For instance, if 5 indicators have been examined in 4 populations, and the result is 19 observations for which the probability is greater than 0.05 but one for which the probability is 0.005, the "corrected value" for this latter is 0.10, and one would not conclude that there was an effect. A more sophisticated, multivariate approach has been described by Smouse [26].

THE COORDINATION OF STUDIES ON CARCINOGENESIS AND MUTAGENESIS

Epidemiologic studies of malignancy rates and life span in workers exposed to radiation in DoE facilities, nuclear shipyard workers, and uranium miners are already in progress, and more such studies are to be anticipated. It would seem only logical — and a considerable economy — that the genetic guidelines we have been discussing should first be implemented in these groups. But logical though this development would seem to be, it may need a little encouragement. That encouragement should come from the realization that this is not just a marriage of convenience but that, given the mutagen-carcinogen axis, detection of an increased germinal mutation rate today might be an advance warning of an increase in malignancy rates 10-20 years hence. The correctness of the obverse is more debatable. In this connection, one of the prime suspect groups from the standpoint of chemical mutagenesis may be 2-pack-a-day cigarette smokers. The consequences in terms of malignancy are well known, as is the mutagenic potential of their urine in the Ames test [27]. However, in the present state of knowledge it can be argued that most of the mutagenic potential of smoking is absorbed by the pulmonary tissues, with little of the mutagen reaching the gonads. Surely the study of this group should have high priority in efforts to understand the mutagenesis-carcinogenesis axis.

THE INTERNATIONAL SCENE

The guidelines developed at this meeting offer a particularly appropriate agenda for international cooperation. The current international interest has already been recognized by the World Health Organization through three Expert Consultations, held in 1978, 1979, and 1980 (see also references [28, 29]). Special groups at relatively high risk of a mutagenic exposure are scattered around the world. The best known of these is, of course, the children born to the survivors of the atomic bombings, on whom collaborative U.S.-Japanese studies have been in progress some 34 years; but there are such other lesser-known groups as the children born to Japanese engaged in the wartime manufacture of sulphur mustard gas [30], children whose parents were exposed to relatively high levels of arsenic in consequence of smelting operations in northern Sweden [31], children born to persons exposed to dioxin in the Seveso accident in Italy [32], or children born to Hungarians who have unsuccessfuly attempted suicide with insecticides or pesticides [33].

Few countries in which such populations occur will be in a position to undertake a complete protocol. It will be important that any partial protocol be undertaken in such a fashion that the results of studies in different countries be comparable. Furthermore, since the smaller countries are more apt to undertake studies which yield presumptive evidence of genetic damage, such as of chromosomal breaks and increased SCE frequencies, it is all the more incumbent on a country with the resources of the United States to undertake the studies on transmitted damage which render the other studies intelligible.

NO EASY WAYS OUT

The guidelines we have assembled these past 2 days offer no quick solution, no cheap litmus paper which an anxious public and a harried government can dip into our gene pool and get an immediate answer. The evaluation of the genetic and teratogenic implications of environmental and occupational exposures is surely one of the most complex and intensely practical challenges of our times. I hope I have made it clear that the reasons for monitoring are as much sociopolitical as scientific. Fortunately, the knowledge and technologies are at hand for an order-of-magnitude improvement in the ability of geneticists to deal with this problem, as compared with only a few decades ago. The next move is up to society, which alone, *properly informed,* can set the priorities which will result in the application of these guidelines.

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Pollutants and Children: Lessons from Case Histories

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From case histories of environmental disasters we can draw lessons about what needs to be done clinically, epidemiologically and experimentally, (usually in that order). Lessons can also be learned from these case histories about public reaction and politics, but I will leave this subject to specialists.

CASE HISTORIES

Dibromochloropropane: Five workers at a factory in California had been unable to father children. Their physicians discounted the possibility that exposures at work might be responsible. The men took their own semen specimens to a clinical laboratory for study. The laboratory declined to give the results (sterility) to the men, but agreed to give them to a scientist who worked with both the industry and the unions involved [1]. A survey of workers in the U.S. and Israel revealed a substantial problem regarding spermatogenesis [2].

Kepone: Workers at an insecticide factory in Hopewell, Virginia, developed severe neurologic disability. Again, their physicians did not pursue the question of a work-related exposure. Finally, a Taiwanese-born cardiologist thought enough of the possibility to contact the USPHS Center for Disease Control directly, instead of through his state health department, as is usually required. In a fortuitous lapse of protocol Dr. Renate D. Kimbrough's laboratory agreed to study a blood specimen. It revealed chlordecone (the formal name for kepone) in high concentration. Study of the workers revealed that some were also sterile. Study of their wives and children revealed that 30 of 32 had the chemical in their blood, tracked home on the workers' clothing [3]. Study of the James River revealed contamination of fish that caused the river to be closed as a source of seafood.

Lead: In the early 1930s an epidemic of lead poisoning was observed among children from an economically depressed area in Baltimore. An intern, Dr. Miriam E. Brailey, went to the area to seek the source of the lead. She had no success until a bystander, Melrose Easter, whose "breath was strong with whisky" said he thought burning battery casings was to blame, for "the smell was bad, even made the food taste bad" [4]. In that household, the fumes were leaden enough to affect not only the child, but also the mother.

In 1974, a physician practicing near Kellogg, Idaho, wondered if two young sibs with abdominal pain, diarrhea, and otitis media might have lead poisoning because of the proximity of their home to a lead smelter. He obtained blood samples and found the lead levels were elevated. The CDC promptly screened other children in the neighborhood and found an epidemic of elevated levels [5]. Another episode at El Paso, Texas, was similarly studied [6]. A search was then made for heavy metal absorption among children living near other smelters throughout the nation. Lead was found to be an environmental problem near Herculaneum, Missouri; arsenic near copper smelters in Tacoma, Washington, and Anaconda, Montana; and cadmium near a zinc smelter in Bartlesville, Oklahoma [7].

Elsewhere, as in Raleigh, North Carolina [8], women who worked at a battery factory wore their clothing home, where the contaminated dust caused lead poisoning in their children. Lead poisoning is still with us in children with pica in old lead-painted houses, and even from burning battery casings [9].

Methylmercury: "Strange incidents occurred in villages along Minamata Bay in [the] Kyushu district in Japan, in which many cats went mad and died. These incidents were the prelude to [an] epidemic of a mysterious disease in human beings" [10]. Thus began a description of the neurologic disorder that swept the area in the 1950s. The disease was caused by fish contaminated with methylmercury derived from factory waste dumped into the Bay. The effect on the human conceptus was not noticed at first, but eventually 40 children with cerebral palsy were identified in a population where only 1 or 2 cases were expected during the interval before fishing in the Bay was forbidden [11].

The value of analyzing preserved specimens was dramatically demonstrated by Dr. M. Harada and his associates at Kumamoto University [12]. They knew that in Japan the dried umbilical stump is kept by the family for decades in a plain rice-paper wrapper. Study of the umbilical stumps from a sibship of 6 children born over the years spanning the epidemic in the 1950s showed a peak in methylmercury content in parallel with the factory's mounting productivity. Unexpectedly, analysis of the dried umbilical stumps from another family with children born 20 years earlier also showed a peak in methylmercury content which paralleled a smaller peak in factory production during the late 1930s. Then it was recalled that an unexplained small outbreak of a neurologic disorder had occurred at that time. Subsequently, methylmercury has polluted water, fish, and people in

Niigata, Japan, and outbreaks of neurologic disorders have occurred in various parts of the world where grain coated with the chemical as a fungicide and meant for planting, was mistakenly used for baking. The worst epidemic was in Iraq in 1971, where more than 6,000 people were affected [13].

Asbestos: Beginning in 1956, a clinician in South Africa observed a cluster of patients with a rare tumor, mesothelioma. A survey revealed 33 cases, 25 of which were traced not to work exposures, but to living near open-pit asbestos mines as children [14]. Subsequently, the marked excess of bronchogenic carcinoma found among asbestos workers was linked to an interaction with cigarette smoking [15]. This finding raises a question about such an interaction in U.S. children exposed to fraying asbestos-sprayed schoolroom ceilings [16], and who later take up smoking.

PCBs: Polychlorinated biphenyls, contaminated with dibenzofurans, used as a heat-transfer agent in Kyushu, Japan, leaked into cooking oil during its manufacture. About 1,200 people who ate food prepared in the oil developed chloracne. Nine infants, in utero when exposed, were small for dates at birth, had transiently darkened skin, and 2 were born with teeth [17]. An effect from PCB-contaminated breast milk has been described by the same Dr. Harada [18] who studied the dried umbilical cords in relation to Minamata disease. PCBs have since been found to pollute chicken feed, rivers, and fish in the U.S. and elsewhere [19].

PBBs: Polybrominated biphenyls, mistakenly added to cattle feed in Michigan, caused debilitation and death. The cause was unknown until a farmer whose herd was devastated and who persisted in believing the feed was responsible, finally had his suspicion confirmed, in part by accident. A gas-chromatograph was mistakenly left on too long, and showed a late-occurring peak that proved to be due to PBBs [20]. PBBs, PCBs, and dioxin (TCDD) are deposited in fat and best excreted in the fat of breast milk. A study in Michigan revealed that about 96% of women in the lower peninsula of the state had PBBs in their breast milk [21]. This chemical, which killed cattle, has not been shown conclusively to cause illness in the most heavily exposed people.

Dioxin: In 1976, a runaway reaction at a factory that made trichlorophenol in Seveso, Italy, burst from its enclosure and drifted downwind as a toxic cloud. Wildlife and domestic rabbits died. About 40 children developed chloracne in about 2 weeks [22]. As yet, a massive study has shown no other well-substantiated effects from the areawide contamination.

In Moscow Mills, Missouri, a waste oil dealer agreed to dispose of chemical byproducts from a bankrupt factory where hexachlorophene had been made. He simply mixed the chemical with the waste oil in his tank trucks, and then sprayed it on the dirt floor of horse arenas. Sixty-three horses died, along with 11 cats, 4 dogs and hundreds of birds [23]. The cause was traced ingeniously to

2,3,7,8-tetrachlorobenzo-p-dioxin (TCDD). The children of the horse arena owner played on the contaminated floor, but suffered only transient symptoms — and one child developed a hemorrhagic cystitis which cleared in a few months [24].

The Love Canal: The story of the Love Canal is too well known to be retold here, except to note that 82 chemicals intermixing in a waste dump have as yet caused no illness in children substantially exposed at home, school and play. The potential problem of areawide contamination by mixtures of chemicals in dumps, only recently recognized, is formidable.

The atomic bomb: The most extensive and longest running study to date of a massive environmental exposure involves the atomic bomb survivors of Hiroshima and Nagasaki. Clinical, epidemiologic and laboratory surveillances have revealed teratogenesis, carcinogenesis, cytogenetic abnormalities, and cataracts. Increased mortality from causes other than cancer has not been found. Seven measures of heritable genetic damage have revealed that the effects are not frequent enough to be detected in the population studied. Psychologic effects probably are experienced by all of the survivors, but objective evidence is less than we would like to have.

It is important to note that after the qualitative adverse effects were defined, emphasis shifted to dosimetry and the consequences of low-level exposures [25].

ANIMAL STUDIES

Wild and domestic animals may be affected along with human beings (PCBs, dioxin, methylmercury) or without detectable human disease (PBBs). Studies of experimental animals usually come after human or veterinary observations suggest a "new" environmental pollutant. A notable exception: hexachlorophene was found to cause brain damage in experimental animals. The use of the chemical in a protective rinse against skin infections of newborn infants was controversial until 1972 when 600 kg of baby powder in France was accidentally mixed with 36 kg of hexachlorophene and killed 36 babies (data unpublished due to legal restrictions while the case was in litigation).

CLINICAL OBSERVATIONS

Almost all known human teratogens and carcinogens have been first recognized by alert patients or their physicians. The hypotheses thus generated are then tested epidemiologically and in the experimental laboratory.

Clusters have thus led to recognition of environmental causes of human disease. Some "rare" illnesses, however, are frequent enough to cluster by chance. Leukemia is an example. Despite valiant efforts over 20 years, study of time-space leukemia clusters has not identified any new leukemogens [26].

SOME STEPS IN THE EPIDEMIOLOGIC STUDY OF AREAWIDE CONTAMINATION

Take a census: In 1968, the military sought to learn through a series of workshops what sorts of action should be taken, should a nuclear disaster occur. A psychiatrist who specialized in disaster studies suggested that an effective way to diminish the population's "loss of orientation" is to take a census [27]. A census not only enumerates people (including children), but also gives them a sense of belonging to society again. In registering the population it is important to obtain a unique identifying number, such as a Social Security number, if possible, to aid in follow-up studies.

Obtain personal history of exposure: As part of the census one should obtain some measure of exposure dose: how far the person was from the source, for how long, etc. For an instantaneous exposure, e.g., the atomic bomb, the information needed is readily recalled and concerns only a moment in time. At the Love Canal the duration of exposure must vary with the places of residence, work, play, and school, as well as with sources of food and drink.

Identify the exposure: Uncertainty about the nature of an exposure increases public alarm. As soon as possible the agent and its impurities should be identified. In Seveso analysis of the dioxin-contaminated soil was handicapped by lack of appropriate laboratory procedures. Exposures from the atomic bomb were unknown until they could be estimated as part of a nuclear weapons test in Nevada. In this test samples of Japanese housing were constructed for estimating radiation exposure within [28]. At the Love Canal the great mixture of chemicals has leaked unequally in various directions through underground streams that change course over time.

Clearly, identifying pollutants and establishing doses can be difficult. It is important to obtain specimens of soil, vegetation and animals — domestic and wild, for evaluating exposures of people. In Seveso, caged domestic rabbits, in whose livers dioxin was especially concentrated, were a valuable guide to human exposures [29].

Preserve human specimens: Human tissues may provide some indication of exposures or body burden many years after exposure — and in fact long after death; e.g., lead in the bones of ancient Peruvians [30]. Study of mummified umbilical cords for methylmercury content at Minamata Bay deserves mention here as well as above. Teeth, hair and dried blood spots are among inadvertently preserved specimens that have been informative about environmental exposures long since past. One should seek to preserve not only specimens that can be measured for chemical content, but also tissues in which subclinical effects may serve as a biologic dosimeter; e.g., cytogenetic abnormalities as revealed in preserved lymphocytes. Also, the urine or feces might be studied in short-term assays for mutagens/carcinogens.

MEASURING EFFECTS

Review the literature: To speed searches of the literature a "crisis response team" has been established at Oak Ridge National Laboratory that can provide a printout within 24 hours concerning the effects of a chemical. The information provided is from human observations and laboratory studies, reports of which are often too widely scattered in the literature or too recent to be readily available from published indexes in local libraries. The information available may help determine the components of the study to be made.

Use existing data sources: Surveillance of some health effects can be made by using data from vital certificates, hospital charts, insurance records or special disease registries. In the United States, the birth certificate gives the birthweight, may provide some information about congenital malformations, and contains the mother's maiden name, of use in follow-up studies. The death certificate diagnoses, unavoidably imperfect, are nonetheless a good indication of causes of death. Outpatient and inpatient surveillance of hospitals can provide information about acute effects. In the area of Mt. St. Helens, for example, a surveillance of hospitals revealed no serious acute diseases from the volcanic ash [31]. For long-term follow-up, the National Death Index [32] should soon simplify determination of deaths in cohorts of people under study. The Index, located at the National Center for Health Statistics, allows identifying information for exposed persons and control groups to be matched against a list of persons who have died in the United States year by year. When matches are found, the state is identified in which the death occurred, and a copy of the death certificate is requested in the usual way. The simplest surveillance may well be through record linkage, rather than by questionnaires, interviews or medical examinations.

Which agency has jurisdiction? When a potentially toxic exposure has occurred, one can scarcely stop to puzzle out which government agency is responsible for dealing with it [33]. The simplest solution is for state health authorities to call the Center for Environmental Health of CDC in Atlanta; Dr. Glyn G. Caldwell for radiation exposures, Dr. Henry Falk for chemical exposures, or the Officer on Duty at night or on weekends. Their recent activities have included responses to medical problems related to explosions at Mt. St. Helens volcano and of the Titan II missile. Insofar as possible, local and state health departments should deal with small-scale contaminations without calling for federal assistance. It is extremely important, however, that such aid be sought promptly when it is really needed. Thus, the federal agency can share the responsibility with the local health authorities. In a comparable situation, the Regione Lombardia in Italy established an international committee of "garanti" to advise on medical activities after the dioxin contamination in Seveso.

THE SPECIAL SUSCEPTIBILITY OF THE FETUS AND CHILD

In 1973, a conference was held at Browns Lake, Wisconsin, on the special susceptibility of the fetus and child to chemical pollutants [34]. The proceedings, characteristically, were not widely read. Proceedings seldom are. Since the conference was held, we have learned of PBBs in Michigan; dioxin in Seveso, Moscow Mills and elsewhere; DBCP; kepone; the Love Canal and many other chemical dumps; heavy metal absorption in children living near smelters; the fetal alcohol syndrome; and asbestos dust from schoolroom ceilings. The effects include sterility, infertility, embryotoxicity, birth defects, possibly transplacental carcinogenesis, low birthweight, skin lesions, nerve impairment, immunologic disorders, cancer, and fear of late effects. To the usual modes for transmitting pollutants, we have added lactation, important in regard to fat-soluble chemicals, and dust worn home on the parent's work clothes.

Problems with pollutants have multiplied in the 7 years since the Browns Lake Conference. The reproductive process continues to be particularly sensitive to adverse effects. We need to use recent experience, so rapidly accumulated, to set priorities and formulate plans for evaluating current and future difficulties with environmental contamination.

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Dr. LEWTAS. There have been several reviews of these human monitoring methods especially as a result of the Love Canal problem and the inadequate chromosome study EPA was involved with. There have been several reports, both from the Office of Health Research in EPA and together with Health and Human Services which survey those techniques, those which we recommend, and how they could be applied in a scientifically adequate way. Of course it helps to have the largest population possible for such studies. Like Dr. Anderson suggested, it is important to have proper cohorts so you can decide whether or not the effect you see is really related to exposure to chemical wastes.

It is difficult, though, from such a study to say that one particular individual's chromosome abnormality is due to their particular exposure. That is the difficulty scientifically. But you can gather enough evidence that you can say there is a certain likelihood and this could make the basis for a decision.

Mr. ROWLAND. Are you familiar with the AMA position on dioxin espoused 3 or 4 weeks ago?

Dr. CARNOW. Are you talking about on the first day, or the fourth day?

Mr. ROWLAND. I am talking about their final position.

Dr. CARNOW. I think the final position was refutation of what appeared to be an irresponsible statement. I think that certainly it was not a studied remark. And unfortunately, I was in Paris at the time. I was quoted in the newspaper in relation to the toxicity of dioxin, not a direct comment on it. Again I think it was just something that was done in the heat of a convention resolution, and certainly those individuals in the AMA who were responsible refuted it as soon as they recognized the nature of the statement. It certainly was not a responsible scientific statement.

Mr. ROWLAND. Dr. Karrh, do you have a comment on that?

Dr. KARRH. I don't have much to add to what Dr. Carnow said, except the original statement itself, I don't think, addressed totally the toxicity of dioxin, except to imply at least that the problem posed by dioxin had been magnified by the press reports. This was passed by the AMA house and on the fourth day was rescinded by action of the same body. I think it was left, though, that the AMA position regarding dioxin is back where it was to start with. That is that they don't have a position on dioxin as such.

They still have a position on agent orange. The AMA Council on Scientific Affairs passed that in January. Agent orange became notorious because of the dioxin contamination.

Mr. ROWLAND. Medical specialists are confused, too, maybe.

Dr. KARRH. I think we are, Dr. Rowland.

Mr. LEVITAS. Dr. Anderson?

Dr. ANDERSON. Could I add something about the risk assessment approach? I think the reason for a lot of the confusion here is the reference to what we have done for years at EPA. It is exceedingly difficult to establish these effects in humans. And although it certainly is exceedingly important to continue trying, we feel that we have enough information, enough tools available on some compounds such as dioxin, to do a risk assessment with respect to different disease endpoints.

In this case we did evaluate the data and decided dioxin certainly has the potential of causing cancer, from animal studies, also reproductive effects. We established what we thought were plausible dose response curves for these diseases and we also did an exposure assessment that took into account soil levels and potential for human exposure. This information formulated the basis for concern in the areas such as Times Beach. And so I guess I wanted to just mention that we don't feel we have to wait for a smoking gun.

I think there might be eternal confusion about direct observation in humans. I realize it is the cutting edge of your bill. It is my own feeling that if you are going to do victim compensation, it is probably going to have to be based on some probability of occurrence rather than certainty because it is so exceedingly difficult to establish these kinds of things in humans.

So I just didn't want to leave you with the feeling EPA was sitting and waiting.

Mr. ROWLAND. Let me say I think what you did was correct, because you functioned under the authority that you were given, which says when there are releases to the environment of any pollutant or contaminant which may present an imminent and substantial danger to the public health. And I think you, EPA, acted entirely correctly from that standpoint.

Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you.

Before I recognize Mr. Sundquist I would just like to find out, Dr. Karrh, would you agree or disagree with what Dr. Anderson just suggested, that in establishing the standard for victim compensation, because of the difficulty in establishing what the standard would be if there were one, reasonable likelihood? Is that what you said?

Dr. ANDERSON. Some set of guidelines that would address potential possibilities or probabilities.

Dr. KARRH. I would agree as long as this were done on a case-by-case determination by a scientist or group of scientists and not have presumptions built into the Act where you automatically presume a certain trigger and once you pass that trigger, you compensate, because people in general develop diseases that tend to cluster. If you have an automatic presumption, automatic trigger, and just because of clustering you trigger that, then you have instigated something that perhaps should not have been in the first place.

But if you use some type of scientific judgment in each case, science panel or scientific board, so that you look at it on a waste site or geographic area at a time, then I would agree with what Dr. Anderson said.

Mr. LEVITAS. Mr. Sundquist.

Mr. SUNDQUIST. Thank you, Mr. Chairman.

Dr. Karrh, my impression from your testimony is that the wagons are in a circle and you are taking your last stand. I would say there is enough blame to go around in all areas. I think the Federal EPA standards today for dumps are not much better than the standards that allowed us to have a Love Canal. I think the chemical industry is continuing to admire the emperor's clothes when in fact he is naked. And there are some things we have to face up to. I think the industry position perhaps has created the situation where we are today where there is a lot of confusion.

My question is: On the first page of your statement you mention that you believe that individuals harmed by exposure to hazardous substances should be compensated by those that caused injuries. That makes sense to me. Then on page 2, though, you say that a special work group was put together to determine if there was a relationship between waste sites and widespread harm. You served as chairman of the group and found that claims of such relationship run counter to the experience of CMA member companies, stating "Moreover, independent surveys have failed to detect significant health problems."

On page 4 you talk about Hardeman County, TN. As I understand what you are saying, the blood tests for some of the residents who used water from a contaminated aquifer displayed a temporary abnormal liver function which disappeared in most cases after the source was removed. Do you have a copy of that study with you?

Could you furnish me with a copy of that study?

Dr. KARRH. I would be more than glad to furnish you a copy. I do have a copy with me but it has been punched and in a notebook. I will submit it to the record and send you a copy personally.

Let me first say, though, that I am sorry you get the impression from our testimony that we have our wagons in a circle and that we are taking our last stand, because we certainly are not.

Mr. SUNDQUIST. Let me tell you why I think that.

Dr. KARRH. Let me finish, if I may, sir.

Mr. SUNDQUIST. You may not want to finish if I tell you why. Let me tell you why. Because as I read through this, from page 1, you think there may be some injuries and responsible people ought to pay. Then on page 2, a study group was created which found that there really isn't much of a linkage. Then as I read on page 4, the case of Hardeman County, TN, the impression I get is that there is not really a problem in Hardeman County, Tennessee. That is the reason I concluded the wagons are in a circle.

I am not trying to blame anybody in particular. I think we ought to take a whole new approach to hazardous chemicals and encourage the private sector to recycle. But when I get to the middle of page 4 and read about Hardeman County, my impression is that you don't think there is a problem in Hardeman County, TN.

Dr. KARRH. If I may continue with the answer I started earlier.

Mr. SUNDQUIST. Absolutely. We want to give you a chance to give all your answers.

Dr. KARRH. It is because we feel there is a public perception of problems and there may be in certain areas where there has been a problem like Hardeman County, TN, where a small number of people did suffer some reversible effects as a result of waste, chemical waste contamination of their water acquifer. But what we are saying is that we think that before Congress passes some type of legislation to address an issue that may not exist, we ought to first determine does the issue exist; second, what is the magnitude of it; and, third, what is the best way to approach it from a legislative standpoint. Don't legislate and then go looking for a problem for which you have a solution, but look for the problem first, then fashion the solution that addresses that problem.

In Hardeman County, this was a rural area with a very small number of people who got their water from an acquifer that was contaminated. Drinking the water did not give them a problem. It was finally determined that they probably had the problem as a result of showering in the water that became heated and the volatiles that were in the water came off, and they inhaled them and developed some abnormal liver function test. As soon as they changed their source of water supply, their symptoms went away, liver function tests went back to normal and the problem from a health standpoint was corrected. That has not corrected the acquifer problem.

We feel before we jump in and do something precipitous we may later regret we ought to find out exactly what we should do.

Mr. SUNDQUIST. Now, the reason I made the statement that I made, now that you have had a chance to comment on it, is that Hardeman County is in my district, so I have some knowledge of Hardeman County. In a lawsuit currently pending before the U.S. district court, Judge Odell Horton has encouraged the attorneys, several times in the course of the trial, to work out a settlement. It is a $2.5 billion lawsuit. The judge has indicated that he is going to make a large judgment against Velsicol if they don't settle.

Dr. Alan Levin of San Francisco testified that Hardeman County residents are kind of sitting on a time bomb. Dr. Jack Sawyer, a clinical psychologist, found mass trauma after contamination of well water in the Toone-Teague area, saying, "You see these kinds of psychological effects in a refugee camp."

Steve Sterling, a Toone-Teague resident, said, "I don't know what's going to happen. Everybody's sick out there, kids and all."

From the Memphis Health Department, Everett C. Handorf, a sanitation engineer, said that chemicals buried there could contaminate Memphis water in 30 years—if not cleaned up.

Several doctors, liver and intestinal disease experts, testified that Toone-Teague residents may have a 10-percent greater likelihood of getting cancer.

One hundred two residents have been examined. A doctor from Vanderbilt, Dr. Robert K. Rhamy, testified that the specific illnesses of several plaintiffs probably were caused by chemically contaminated water. It is not just an isolated incident. It is not just a few people. It has not been a temporary abnormal liver function which is going away. It is a $2.5 billion lawsuit with permanent injury, with a lot of things involved, and I think in your testimony you are underestimating the extent of the problem.

In fact, from reading your testimony, I would have the impression—I don't know whether my distinguished colleagues would have—but I would have the impression that the study group that you headed found no relationship between waste sites and widespread harm.

Then I look at Hardeman County, and as stated here, that the blood tests for some of the residents displayed a temporary abnormal liver function which disappeared in most cases with removal of the source of exposure. From what I can determine the problem is much more serious than that. In the next few weeks we are going to find out how serious it is. I would also submit that the Federal Government has to share in a lot of the blame for where we are.

But I say that in statements like that which you have read and furnished us, you are viewing the emperor with clothes on.

Dr. KARRH. I, ~~think,~~ Mr. Sundquist, what we are saying here is that we have reviewed the literature, the data that are available to us. The Hardeman County study was written up in the scientific literature and was peer reviewed in the scientific literature. The data you have presented obviously came out of a lawsuit which we have not had access to. We probably could have, but we have not. But that is another indication that the current, existing system works. Because these people do have access to the courts and to the tort system.

There is not an indication from what you have just said that this tort system has broken down in this particular case such that there needs to be some other type of compensation mechanism. If we are faced with a situation after we have done other appropriate studies that indicates the tort system can't handle the load that is going to be presented to it or that there are indeed victims such as you have described here that have not been written up in the scientific literature yet, then the chemical industry will totally support that.

Our position is that if the system now works, let us look to see if we need a better system, because people do have access to the existing system.

Mr. SUNDQUIST. Doctor, my point is, with all due respect, that in the statement you just made you are talking about the issue we are discussing today. But in most of your testimony I think you are underestimating the problem to imply that we don't have the serious problems that do exist. When you say you will move your family to Times Beach, I would submit to you, you can buy a lot of property there very cheaply. It might be a good investment if you can find a lot of other people who will move there with you. I think what gets the industry in trouble is not recognizing that we do have a serious problem. There are people out there who do have claims, and let us talk about that, as opposed to trying to leave the impression, right or wrong, that there is not a problem in Hardeman County, TN, or there is not a problem in Times Beach, MO.

I think there is a problem. Now, let us talk about whether or not we need Federal legislation. That is what we ought to be talking about, as opposed to trying to induce the impression that we don't have serious problems with hazardous chemicals.

Dr. KARRH. I think, Mr. Sundquist, that is what we were addressing, is the fact we have reviewed the data that are available and have concluded that we cannot, in the scientific data, find there is a serious problem. There is a lot of talk about it. The public has a perception that there is a problem. We think we ought to do the studies to determine if there is a problem. We totally support the studies. But not just pick up the emotional side of it, assume there is a problem and then do something based on that assumption rather than adequately taking care of whether or not there is a problem and what is its magnitude.

Mr. SUNDQUIST. That concludes my questions. I will take another turn, Mr. Chairman.

Mr. LEVITAS. Thank you. We will be going around again.

Are you familiar, Dr. Karrh, with the proposed legislation that would require the chemical industry manufacturers to provide proof of efficacy of pesticides before they could be marketed and provide that information to the public or purchasers of the pesticide? Would you think that would be useful information for a purchaser to know that if he is buying a product, a pesticide or some other fungicide or herbicide, that it is effective?

Dr. KARRH. That is a pretty hypothetical question that you are asking. I would assume the companies and people who were purchasing it would not want it purchased unless it was effective. Our position, though, is that if there is a toxic characteristic of a material, whether we are selling it into commerce to other companies or whether we are selling it to the public, that we should provide adequate information as to what the toxic characteristics and hazards of that material is, if that is your question.

Mr. LEVITAS. I will take that as the basis for what I am going to ask.

Mr. ROWLAND. Mr. Chairman, you asked for the efficacy, didn't you?

Mr. LEVITAS. Yes. But he introduced toxicity.

Dr. KARRH. I think I answered the efficacy by saying that if we sold a product we would want it to work, or we wouldn't sell it.

Mr. LEVITAS. Let me take the toxicity because that might even be a better illustration of what I am trying to find out. I am sure Du Pont, for example, developed some sort of scientific or medical protocol before it markets a product to ascertain its toxicity.

Dr. KARRH. Yes, sir.

Mr. LEVITAS. What type of cause and effect determinations are made in terms that would exist between a given chemical product and health effect or health risk? What degree of certainty do you seek to achieve?

Dr. KARRH. As I mentioned earlier, we have a toxicology laboratory started in 1935. On any product we plan to introduce or any chemical substance we plan to use to which our employees may be exposed, if data do not already exist indicating what are the toxic characteristics of that product, then we will develop a battery of toxicity tests that will be done in this laboratory.This will include a lot of different tests.

The tests will depend largely upon what use is going to be made of the product, what the exposures are likely to be and how it is going to be handled. Then each of these tests are evaluated by scientists on an individual test basis. Based upon the results of the tests and results of their evaluation the scientists decide several different things on the chemical.

First is it too toxic to try to handle? If that is the case, we stop there and don't do anything else. Or does it have certain toxic characteristics that require special types of controls and medical surveillance? If it is the latter, then we decide what those are. When we introduce it into our plant sites we put in those controls to protect our employees. Then if we decide to introduce a product into commerce and sell it to other companies or sell it to the public, we will take the same data and decide first is it safe for other companies to purchase from us or for the public to use once they have purchased it from us, and if we decide yes, then we decide what precautions we should give to the people who will be handling or using the material.

Mr. LEVITAS. Would Du Pont market a chemical product, bulk chemical or drug or pesticide, without conducting tests so that it knows with 100-percent certainty that there are no adverse health affects that will result from exposure?

Dr. KARRH. Mr. Levitas, I don't think there is any material made that there is 100 percent certainty that no adverse effects will result from exposure. Table salt, water and other things are quite toxic if not handled properly. So the answer to your question is we market many chemicals that have toxic characteristics, some of which are quite toxic. But we know, based upon the data we have developed and our evaluation of the data that if the protocols we develop are followed, people will not suffer adverse health effects from exposure to these materials.

Mr. LEVITAS. Let me get back to my efficacy question. Would Du Pont market a product that it does not have 100-percent certainty would be efficacious?

Dr. KARRH. Since I am not sure how you are using that word, would you define it for me, please.

Mr. LEVITAS. Well, that it works.

Dr. KARRH. That is a marketing definition, not scientific, and I am not prepared to answer that question.

Mr. LEVITAS. You don't know the answer to that question.

Dr. KARRH. That is correct, I do not know.

Mr. LEVITAS. What degree of evidence does a company like Du Pont seek to achieve or attain either as to efficacy, or as to toxicity before it make a decision on marketing. I mean do not run every conceivable scientific test that might be available to evaluate what the health effects of the chemical would be, or what the efficacy achievement might be?

Dr. KARRH. I am going to have to excuse myself on answering the efficacy question because I really am not qualified to answer that. That is a marketing question and I am not in the marketing area. I can address the toxicity question. The answer to the question is no, we don't run every conceivable toxic test that you could run because then you couldn't afford to buy our products if we did. We run the tests that we feel, based upon our scientific evaluation of the compound and other compounds like it and our scientific evaluation of the early toxicity tests that we do, need to be done.

We will run every test we feel helps us determine or better define what the toxic characteristics are. But once we have decided that we have adequate toxicity information, that we know that chemical can be handled safely, then we probably will not run additional tests from then on.

But we do run adequate tests so that we can tell our customers and our employees what steps need to be taken to make sure people don't suffer harm from exposure to that material.

Mr. LEVITAS. So you run enough tests that satisfy yourself, that satisfies your company, that a product is safe enough to be used under whatever cautions are given even though it hasn't run all the tests capable of being run on that product?

Dr. KARRH. That is correct. We make a scientific determination on each chemical as to what are the toxic characterisics and what protective measures need to be taken.

Mr. LEVITAS. Let me tell you where that leads me. Congress has to make decisions. EPA has to make decisions and courts have to make decisions. And I believe on something short of absolute certainty as to cause and effect linkage.

Mr. Molinari is going to be asking some questions, I know, about the tort aspect of it. But from my point of view, in cases such as chemicals that are in hazardous waste sites, whereas all of you have testified, there may be long latency periods before we have actual manifestation of the particular abnormality that is caused or may be caused by that, maybe 10, 15 years down the line, I don't think that it is appropriate to ask Congress or EPA to wait and expose the population to those potential effects until we have had that 15 or 20 years of experience when the consequences of failing to take some action in the meantime will potentially expose large segments of the population to high risk. And that there has to be a place in time where, based upon scientific literature, reports of health consequences, individual health diagnoses, where there are statistically large numbers of occurrences that are different from what you would normally expect, that at that point, Congress ought to take some action, and so should EPA, without waiting for the long-term study that establishes beyond the shadow of a doubt the linkage, causal linkage that you seem to be suggesting should be done.

Dr. KARRH. Mr. Levitas, I think you probably think we are proposing to wait 10 or 15 years on each and every dump site. In fact, the chemicals that are in disposal sites now have been in use in industry for many, many years, some of them 30, 40, 50 years. The work force that has been exposed to those chemicals, at usually higher concentrations than what they would be exposed to at disposal sites, have been studied extensively.

The chemicals have been studied from a toxicological standpoint. The work forces have been studied. So we don't have to wait the 10 or 15 years. We know that once we determine what is in a waste disposal site and we go back to the animal data that we have and to the work place data that we have, we already have satisfied the 10- or 15- or 20-year, latency period in most cases.

Taking dioxin as an example, the first industrial accident in which large numbers of people were exposed was in 1949 in Nitro, WV. That population has been studied extensively since then. That is a period of 34 years. They are still being studied by the University of Cincinnati. So we don't have to wait for a dioxin disposal site like Times Beach for another 10 or 15 years. We have good data. That is precisely what my CMA work group did, was look at the occupational data we had on the chemical substances that were in disposal sites and decide whether or not there were likely to be health problems from these and, if so, what was the magnitude of them. We concluded that there was not likely to be any great number of health effects from these. And there was time to do the more in depth studies that need to be done, like the study we are going to be doing and like the studies the ATSDR will be doing.

Mr. LEVITAS. Let us take the dioxin situation where you say there has been a long period of time to study the health effects. There is a large body of evidence as to the properties of dioxin and we are constantly receiving reports of waste sites containing dioxin or dioxin-composed chemicals. We have a situation in Michigan which is current. There are several reports of sites in New Jersey, Times Beach, and I could go down the line, where dioxin is involved.

If I understand what you have said, based on your reading of the literature, that you don't see evidence of adverse health consequences from people living in proximity to these sites containing the dioxin.

Dr. KARRH. That is correct with the dioxin. Consequently, we feel further study needs to be done. Because we haven't seen these health effects, we are not putting people at risk during the relatively short period of time we are suggesting be taken to develop the magnitude—

Mr. LEVITAS. Consequently, the policy conclusion that would be drawn from that is therefore Congress shouldn't act until there is more proof—or proof, and EPA shouldn't act based on the present state of the data base.

Dr. KARRH. No, sir. We feel that EPA should get on with cleaning up the orphan land disposal sites now as Superfund required that they do. That was part of the lawsuit that was filed. We feel that the disposal sites should be cleaned up.

EPA should act in this area. We feel that CDC and EPA should act on the health studies.

Mr. LEVITAS. Why do you think that should be done? See, that is what I don't understand. I am going to get to Dr. Carnow in a minute. We are talking about in the case of Superfund, spending $1.6 billion for openers, and it is going to be a lot more than that in my judgment if the program goes forward, and I am satisfied it will.

But why should we spend that type of money if there is not evidence that there are health consequences resulting from the existence of these hazardous waste sites? Why should we do it?

Dr. KARRH. There are several reasons we should do it. First, the public perceives there is a problem and it should be taken care of for that reason.

Mr. LEVITAS. That is a lot of money to spend for cosmetics.

Dr. KARRH. Well, I think the chemical companies put it into the Superfund for that purpose. It is a type fund that once it is spent, EPA can come back for recovery of the funds they spent out of it from the people who created the hazard in the first place. So it would still be coming out of the chemical industry. The fund was created for that purpose.

Mr. LEVITAS. I don't want to get into that aspect of it because I would argue that any time there is a charge, a cost, it is going to be passed on to the public ultimately in some shape or form. So I am not really interested—I am just saying there is a lot of money that is being spent to do something.

Now, the only reason that you have given that it is being spent is because the public perceives that it is a problem. But your studies and working group has concluded there really isn't evidence of a health problem.

Dr. KARRH. The only reason that was the only reason I gave was because you cut me off before I could finish. These disposal sites that are out there, many have failed. They are leaky sites. They should be cleaned up and cleaned up expeditiously.

Mr. LEVITAS. Why?

Dr. KARRH. Thank you, sir. They should be cleaned up expeditiously because since they are leaking they will leak into areas where people can be exposed to them. So we should get on with the cleanup of those sites. We should make sure that our future efforts are such that we don't create sites like that. But we have to do some type of work, some type of study to determine which sites to clean up first.

We have 419 priority sites. EPA has added another hundred, or so, to that. EPA does not have the resources to clean that up. So we have to do something in setting priorities to clean them up under Superfund. But we think the sites should be cleaned up and certainly hope EPA will do something toward cleaning them up.

Mr. LEVITAS. Have you finished?

Dr. KARRH. Yes.

Mr. LEVITAS. You haven't answered my question. If those chemicals in those sites do not pose a health risk to people, or to use your words, there is no evidence of significant health problems from waste sites, if that is the case, I, for one, would strongly object to spending that kind of money to clean up these sites just for cosmetic reasons. If these sites were filled with benign materials, distilled water, and it was seeping out, I think it would be a dreadful mistake in terms of public policy and use of public resources to spend $1.6 billion cleaning up those sites.

Why do you think these sites should be cleaned up if they don't pose a significant health hazard?

Dr. KARRH. I think I have already covered that, but what we have said is, based upon the data we have now, we don't see a significant health hazard. There is a public perception of a problem. Some of the sites are leaking and are likely to create a problem in the future. So let us clean those sites up. Let us continue, before we pass some type of victims compensation legislation, to look at the scientific data and determine if there is a need to compensate victims.

We know we need to clean the sites up because the sites, some of them, are leaking, some of them have gotten into acquifers. Hardeman County, TN, is a good example. And those sites should certainly be cleaned up. But that doesn't mean that because the sites need to be cleaned up that we have to have victims before we clean them up. That is waiting, as some have said, to count the bodies. We don't think we ought to do that at all. We think that where a site may pose a hazard it ought to be cleaned up.

Mr. LEVITAS. Why would it pose a hazard?

Dr. KARRH. Because it may be leaking, it may be getting into acquifers.

Mr. LEVITAS. And the chemicals that are leaking could cause an adverse health effect in the future.

Dr. KARRH. They may in the future cause adverse health effects. The studies we have done indicates they are not significant adverse health effects at this time, but we don't know what will happen 10 or 15 years from now.

Mr. LEVITAS. We will take the public perception and put that over here so we don't need to address that anymore. Let us come back to the "may cause health effects in the future." And you would agree that the potential of causing health effects in the future is a justification for cleaning up these sites under Superfund?

Dr. KARRH. Yes, sir, I would agree with that.

Mr. LEVITAS. Thank you.

Dr. KARRH. But that doesn't mean we have health effects now.

Mr. LEVITAS. Dr. Carnow, would you care to comment on that briefly?

Dr. CARNOW. Briefly?

Mr. LEVITAS. Yes, I said briefly.

Dr. CARNOW. All right. You have to understand we are good friends, but we have some disagreements.

Mr. LEVITAS. I sensed that. I wanted to find out what the nature of those disagreements were.

Dr. CARNOW. First of all, I think that there are at least 400 priority sites and there have not been anywhere close to 400 studies. It is really presumptuous to say there is no health effect. I think, for example, if you did a study of dioxin in a remote area in Canada you probably would not find health effects. If you do them in Times Beach, I submit you will find those.

The fact is that we have difficulty with some studies. There is an enormous amount of data on health effects of dioxin. We have to take that because that seems to be this contention point. EPA says in its document it is the most deadly of all manmade chemicals. I think they say it is possibly, or probably the most—I think we all recognize that it kills in incredibly small amounts.

The study or studies to which Dr. Karrh referred, first of all, were studies which were funded by Monsanto. We have examined people from Nitro. I can assure you that our results are different than the results of those studies. And when we can we will publish them. They are in litigation now. We can't publish them. The studies were done by a dermatologist who was mostly interested in chloracne. And the fact is that the studies at BASF in 1953 and the reports in the literature which again I know have 23 volumes of reprints on the health effects of dioxin, and that does not constitute an exhaustive group of reprints.

There is no question it is extraordinarily toxic. I think to deny that is to deny reality. I think again in terms of the problems we face, I don't know the extent of the problem. I don't have the confidence Dr. Karrh has that there is no problem, or that we find no problem. I feel that out there is an extraordinary potential problem. Dr. Karrh agrees.

If this stuff leaks out, it is dangerous. I submit that some of that has been there for 50 years and it has leaked out, and we don't know how dangerous it is. I am not suggesting that all of us are going to die imminently. I am suggesting it requires intensive effort. But while we make those intensive efforts, if we find levels of dioxin in the community, then we have to take every measure to protect that community.

I understand the staggering costs. I understand all those things. But I certainly would not put my family into that community. I have examined children from those communities and they are very sick children. They are children who have brain damage. They are children who have seizures. They are—I just examined a 6-year-old child that has a blood cholesterol that would stagger you. You don't see that in 6-year-old girls.

Again I just—I don't want to be accused of emotionalism. I think people become emotional because they feel threatened. I don't think people feel threatened because they are emotional. I think there is a basis for all of this emotion. I think people are concerned about their families and so on.

I think our response should not be an emotional response. A scientific response. But I think it has to be a serious response that looks not only at what is in the dumps, but looks at what has happened to communities around them. They are going to be very expensive studies.

But on the other hand, if we know that there is a dump, and if we know that there is leaking, and if we find any levels at all in soil or water that may be a problem, we have to either at least examine people promptly as you point out, you can't wait, or else we move people away until we can make a determination of their health conditions or the problem that exists.

I can only tell you in looking at—they are very insidious problems. Looking downwind from two smelters in two different States we found little children who are retarded. That represents a serious cost to society. An enormous cost. We estimate $260,000 to raise that child who has mental retardation. Very subtle. We have to look with great care. I don't think we can say we may not have a problem at all, and we are going to clean it up, because people have emotional problems in this country.

Mr. **Levitas**. Let me give Dr. Karrh his due on that. I think he finally said that we ought to clean it up, not just for public perception reasons but also because there is the potential of adverse health effects.

Dr. **Karrh**. That is right. After I got a chance to start back again.

Dr. **Carnow**. And I said we don't know whether there are potential or determined health effects.

Mr. **Levitas**. We have made some progress. Let me say this one thing and then conclude. I know Dr. Karrh wants to make some response on what Dr. Carnow has said. If we could or should take action based on potential health effects in the future, and therefore clean up hazardous waste sites which are leaching out hazardous materials into surface waters and groundwaters and the soil, then should we not also consider providing a system for compensating the human beings who are being affected or exposed to those materials at the present time rather than waiting a long number of years to bring about this scientific certainty that you would—in other words, shouldn't we provide as much concern and response on the likelihood of this problem occurring to individuals as we do for cleaning up the property where the materials are stored?

Dr. KARRH. If we have determined that people have been exposed to these materials and are suffering health effects as a result of that and a scientific determination is that there is in fact the reason for it, then sure they should be compensated. But we feel that if we clean up the sites as Superfund is supposed to, we wouldn't run as great a risk of having people who have to be compensated.

We don't feel we need to put in some type of mechanism now to compensate people if we don't know people out there need to be compensated. But we do need to get on with cleaning up the sites and determine if there are people who need to be compensated. That was part of my testimony. Although we have not seen evidence of significant health problems and while that evidence leads us to believe there is not likely to be a significant health problem, we fully agree the evidence in this area requires a thorough scientific review.

Dr. Carnow's comment regarding the Nitro, WV, study, the study group was headed by a dermatologist who has a special interest in chloracne, but he is also head of the Kettering Institute which has a multidisciplinary approach and probably is the best recognized occupational and environmental health laboratory. He did not do the study by himself. He had a team of several different disciplines.

Mr. LEVITAS. Dr. Rowland asked me to yield. I will now do so, then recognize Mr. Molinari.

Mr. ROWLAND. Thank you for yielding, Mr. Chairman. You mentioned, Dr. Carnow, children who had brain damage, I believe. Maybe I missed the point somewhere. Were these people who were exposed to dioxin?

Dr. CARNOW. No, the children I am talking about were children—you may have come in after I discussed this—were children who lived downwind from lead smelters.

Mr. ROWLAND. Thank you, Mr. Chairman.

Mr. LEVITAS. Was one of those sites the Dallas situation that we have investigated?

Dr. CARNOW. No. The first one was found theoretically. I was asked by the city of El Paso to look at the question of sulfur oxides. When I looked at the emission data I found that 800 tons of lead had come out of that smelter, and suggested to the health commissioner that they might have a lead problem. He suggested they had never had a case of lead poisoning in El Paso or that area.

We then examined some of those children for him. And we found that large numbers of them did, in fact, have chronic blood poisoning. But it wasn't the kind of blood poisoning you see in Chicago where a child eats some leaded paint and develops an encephalopathy. It was very chronic, very quiet, very subtle. The children were weak, anemic, retarded, dropped out of school. That kind of problem.

This has been found since that time in El Paso. That has been found in other areas around the country in relation to some of these things. This doesn't mean, of course, that every industrial plant is spewing out poison. I am not implying that.

Mr. LEVITAS. I understand. Mr. Molinari?

Mr. MOLINARI. Dr. Karrh, I would like to pick up again at the point where the gentleman asked you about the question of cleanup. I would like to see if I correctly understand you and your recommendations, that we do need to clean up the sites. What sites were you referring to?

Dr. KARRH. I think any site, Mr. Molinari, that presents a hazard or may present a hazard should be cleaned up as soon as we possibly can. Then any other site that is on the priority site list should be cleaned up. But we have to have some order of priority because our resources are finite.

Mr. MOLINARI. Let us see if we can narrow this a bit. Would you agree that there is a need to clean up the 419 priority sites that were established by EPA?.

Dr. KARRH. Based upon what I know about those sites, from having read only EPA's very short summary, I would say yes there is a need to clean them up but they should be cleaned up in some order of priority so we make sure we take the worst first.

Mr. MOLINARI. I think we all agree to that. What if there is not enough money in the Superfund to achieve that purpose? Would you agree that more funds should be collected according to the present formula in order to have an adequate sum to complete the job?

Dr. KARRH. I think we first ought to start using the money we have in there for the purpose for which it was intended and begin to clean up the sites and then determine whether or not there will be enough money. That $1.6 billion fund was to clean up the orphaned sites. The Government could first use the money to clean up the sites, and then at the same time, the legal side of the Government could try to recoup that money back from the people who put the material there.

So if it works the way it should, the fund would replenish itself based upon what was being brought in from the legal action that was taken. If later it is determined that this is not adequate to clean up the sites, then there ought to be a need for further funding. But we ought to start cleaning up the sites——

Mr. MOLINARI. Aren't they in fact doing that now?

Dr. KARRH. I am not aware that large numbers of sites have been started. I know EPA has started some sites but I can't tell you which ones.

Mr. MOLINARI. Are you allowing for the number of sites where it is apparent from experience that EPA is not going to be able to collect reimbursement because the companies involved are bankrupt or otherwise out of business?

Dr. KARRH. That is what the Superfund was set up for; the orphaned sites where a responsible party could not be determined. In most of the cases where there is a responsible party, EPA does have redress back to that responsible party.

Mr. MOLINARI. It would appear obvious from your answers that there is not going to be enough money to clean up the 419 sites. You identify them as orphaned sites, although you do recognize the fact that there are some responsible parties from whom we can collect a contribution. The reason I ask this is that we have had testimony that indeed there wouldn't be enough money to clean up 100 sites.

Dr. KARRH. I think until we start cleaning them up and until EPA starts going back to the parties that they can identify as responsible and taking the appropriate action to recoup what they have spent cleaning up that site, we really wouldn't know whether there will be enough money or not. We should proceed with the clean up and see if there is enough.

Mr. **MOLINARI**. I am asking a simple question. Assume hypothetically there is not going to be enough. That in fact the testimony we have is accurate. That we only have enough to clean up 100 sites. Do you recommend we go further and collect more moneys and proceed to clean up the remaining sites?

Dr. **KARRH**. I didn't come prepared to make a legislative recommendation to you at this time. What I would like to do is just submit something back to the record on behalf of the chemical industry.

Mr. **MOLINARI**. Fine. I would be glad to accept that.

Dr. **KARRH**. Thank you.

[The information received from Dr. Karrh follows:]

E. I. DU PONT DE NEMOURS & CO.,
Wilmington, DE, August 22, 1983.

U.S. HOUSE OF REPRESENTATIVES,
Subcommittee on Investigations and Oversight, Committee on Public Works and Transportation, Rayburn Building, Washington, DC.

Mr. Molinari had asked that CMA comment on whether Congress should go further and collect more monies for Superfund (see transcript p. 111 and 112). In response to this question we wish to state that it is not possible at this time to determine whether or not the $1.6 billion to be raised through Superfund taxes will ultimately prove to be adequate. It is clear, however, that there is now plenty of fund money available and that EPA and Congress have time to assess the fund adequacy issue carefully before acting to impose additional taxes. As of May 31, 1983, the U.S. Treasury Department, Bureau of Government Financial Operations, Division of Financial Management, Trust and Revolving Funds Branch, reported a net balance in the fund of over $470 million. Thus, the fund contains substantial monies.

The amount of money in the fund is not, however, the sole factor to consider in determining its adequacy. The level of federal funding needed will depend on several key factors. Perhaps the most important are the number of sites needing cleanup, the cost per site, and the degree to which private funding will be used for cleanup (either "up front" or to reimburse fund expenditures).

It is too early in the administration of the Superfund cleanup program to make accurate projections on any of these key factors, but experience thus far clearly shows that private funding is being used a great deal. Many sites are being cleaned up totally from private funds, and many others are being cleaned up with substantial percentages coming from private funding. According to EPA, over $118 million had been collected by the end of 1982 through private party contibutions to Superfund waste site cleanup.

One example is the Fridley site in Minnesota (which is the No. 1 priority site on EPA's proposed list of 419 sites). There the FMC Corporation recently agreed to perform a surface cleanup and groundwater monitoring program for approximately $6 million. In addition, last month the Minnesota Mining and Manufacturing Company committed to clean up certain of its sites in Minnesota for $6 million.

There are also recent examples of groups of companies agreeing to contribute shares towards the cost of cleanup. At the Bluff Road site in South Carolina, 14 companies contributed $1.8 million. At the Chem-Dyne site in Ohio, 112 companies contributed $2.4 million. At the Deerfield site in Ohio, 8 companies contributed $2 million. At the Wade site in Pennsylvania, 32 companies contributed $1.7 million. Thus, it is clear that private funds are a significant source of cleanup funds in addition to Superfund.

In addition, we should note that EPA is now preparing a comprehensive report, in compliance with Section 301(a)(1) of CERCLA, assessing these key factors in order to "project any future funding needs". CERCLA Section 301(a)(1)(C). This report is due in December 1984. In light of the facts that there is now a balance in the fund of almost ½ billion dollars and that EPA is currently carefully assessing future funding needs, we believe that it would be inappropriate for Congress to impose new taxes now.

I am also enclosing the following for inclusion in the record:

1. The study of residents in Hardeman County, Tennessee, as requested at page 83, line 1896, entitled "An Environmental Health Survey of Drinking Water Contamination by Leachate from a Pesticide Waste Dump in Hardeman County, Tennessee", from the January/February 1982 *Archives of Environmental Health*, Vol. 37, No. 1.

2. A copy of the UAREP study, as requested on page 113 of the transcript, entitled "Report on the Feasibility of Organizing a Study to Assess the Potential Health Hazards of Waste Disposal Sites Within the United States", by Universities Associated for Research and Education in Pathology, Inc., dated June 22, 1983.

3. A report entitled "Epidemiologic Study of Workers Exposed to Acrylonitrile", by Maureen T. O'Berg, Ph.D., from *Journal of Occupational Medicine*, Vol. 22, No. 4, dated April 1980.

4. A report entitled "Angiosarcoma of Liver in the Manufacture of Polyvinyl Chloride", by J. L. Creech, Jr., M.D., and M. N. Johnson, M.D., from *Journal of Occupational Medicine*, Vol. 16, No. 3, March 1974.

5. A study entitled "Mortality Study of Workers in the Manufacture of Vinyl Chloride and its Polymers", by Irving R. Tabershaw, M.D., and William R. Gaffey, Ph. D., from *Journal of Occupational Medicine*, Vol. 16, No. 8, dated August 1984.

6. A report entitled "Respiratory Cancer in Relation to Occupational Exposures Among Retired Asbestos Workers", by Philip Enterline, *et al.*, Dept. of Biostatistics, Graduate School of Public Health, University of Pittsburgh, from *British Journal of Industrial Medicine*, Vol. 30, 1973.

7. A report entitled "Lung Cancer After Employment in Shipyards During World War II", by William J. Blot, et al., from *The New England Journal of Medicine*, Vol. 299, No. 12, dated September 21, 1978.

8. A report entitled "Dust Exposure and Mortality in Crysotile Mining, 1910-75", by J. C. McDonald, *et al.*, from *British Journal of Industrial Medicine*, Vol. 37, 1980.

9. Report entitled "The Pathogenesis of Asbestos-Associated Diseases", by J. D. Craighead and B. T. Mossman, from *The New England Journal of Medicine*, Vol 306, No. 24, dated June 17, 1982.

The above reports (3 through 9) of workers incidence of disease resulting from exposure to acrylonitrile, vinyl chloride, and asbestos are for inclusion at page 125 of the transcript.

I also enclose for the record preliminary reports relating to studies of the Brookfield landfill by The Bureau of Preventable Diseases of the New York City Department of Health. This work was discussed at page 155 of the transcript. Also enclosed for the record is a critique of these reports.

Sincerely,

BRUCE W. KARRH, M.D.,
General Director,
Medical, Safety and Fire Protection.

Mr. MOLINARI. You make reference in your prepared text on page 7 that you were going to submit a copy of the NAREP feasibility study for the record, but apparently you don't have a copy of the study with you.

Dr. KARRH. I have a copy of the study with me. I do not have enough copies to submit the number required. I can submit it for the record either by giving it to you now, or I can submit it to the record later, whichever you would prefer.

Mr. MOLINARI. I would like to have as many copies as you have available. We would like one submitted for the record.

Mr. LEVITAS. Without objection it will be made part of the record.

Dr. KARRH. I only have one copy with me I can submit to the record at this time. Actually the one I have with me is a draft. I will have to submit a copy.

Mr. LEVITAS. Do you have one now we can reproduce?

Dr. KARRH. No; the one I have is a draft. I thought I had one of the originals with me, but I don't.

Mr. MOLINARI. The study was released on July 7. I take it it is in final form?

Dr. KARRH. That is correct.

Mr. MOLINARI. If you would, turn to page 4 of your statement.

Dr. KARRH. I have a final copy of the study, but this is my personal copy that has my scribbling.

Mr. MOLINARI. We wouldn't want that.

Dr. KARRH. There is nothing I have in here that I would mind you seeing, but I think I would prefer to keep this one and submit one for the record.

Mr. MOLINARI. Fine; if you would turn to page 4, toward the bottom of the page you talk about occupational exposure and health data providing an indication of the health effects that might be expected if similar exposure patterns were encountered by the public. Then you go on to say:

> Since we saw no significant increased incidence of chronic disease among employees in the chemical industry, we concluded that widespread increased incidence of chronic disease among the public is also unlikely.

Now, before I heard you testify that there should be a differentiation made between the various chemicals. Here you are prepared to make the statement that regardless of the chemical that has been produced by the industry you represent, you have seen no significant increase in incidence of chronic disease. Is that a fair statement?

Dr. KARRH. I am not sure I understand what you are asking.

Mr. MOLINARI. When we asked the question before about the exposure to toxic chemicals, I think you made a point, a very definite point, that we have got to think in terms of what chemicals we are talking about. There are some that will not cause a problem. There are others that are more serious and have to be looked at more carefully, and we should be careful to distinguish what we are talking about.

But here you are making a general statement that there was no increased incidence of chronic disease among employees in the chemical industry. Now, I take it that that means all chemicals prepared or manufactured by the chemical industry.

Dr. KARRH. It means potentially all chemicals. What we were doing here was trying to look at the data that were available. We determined that there was not much data available that was specific to waste disposal sites and to people who may have been exposed to that.

In order to try to get some understanding of what may be the magnitude of the health problem from waste disposal sites if there was one, we went to the next data that we had that indicated, people would be exposed to chemicals and for which we had some health data. That is employees of the chemical industry.

We evaluated those data. Based upon the results of the studies that have been done in that industry and the fact that chemical industry employees would be expected to be exposed to larger concentrations of the material, than would people living out in the community that may be close to a disposal site——

Mr. MOLINARI. How do you come to that conclusion?

Dr. KARRH. I haven't reached a conclusion yet.

Mr. MOLINARI. OK.

Dr. **Karrh**. We were able to make inferences that the likelihood of people in the industry suffering adverse health effects from chemical exposure would be greater than people in the areas of the waste disposal site because of the differences in the potential exposure. That tells us that, although we cannot conclude that people in the waste disposal sites would not have health effects, at least it tells us that there should not be a pressing, imminent problem that means that we have to take action now without doing the proper studies that need to be done to determine if there is a problem.

Mr. **Molinari**. In the case of those working in the chemical industry, don't they wear protective gear that is supposed to insulate them from adverse health effects?

Dr. **Karrh**. They would wear protective gear if they were exposed to materials at concentrations that had been shown or were likely to cause adverse health effects, but generally that is at a much higher level than what one would expect to find out in the community where there are a lot of different factors than at work, such as an open air system where the concentrations of the material would be dissipated.

You have to determine first is there any exposure occurring out in the community. In many cases the material would not be released in concentrations in which there could be exposure as large as chemical industry people would be exposed to.

Mr. **Molinari**. Are you telling us that there are cases in the industry where people are working and are exposed to chemicals that could cause a health problem and that the industry is not in those cases providing protective clothing?

Dr. **Karrh**. I am telling you that there are cases where people in the chemical industry are exposed to chemical substances. Based upon the scientific data that we have, though, we have been able to determine at what level the chemical substance is likely to cause a problem. We then put in a factor to make sure the employees aren't exposed at that level. You can't operate a chemical plant at zero exposure to a material.

The only way you can is by what is called remote operation. There are very few processes that people are willing to run through remote operation because of the difficulty of running, so you do have some exposure.

Mr. **Molinari**. It is fair to say the rate of exposure, then, would differ from chemical plant to chemical plant, and from chemical to chemical as well?

Dr. **Karrh**. That is correct, between companies, between plants and between chemicals.

Mr. **Molinari**. OK. So taking your statement that you can't operate at zero exposure, are there workers then that would be seriously exposed in the industry?

Dr. **Karrh**. The companies, an individual company, based upon the data they have, will determine what would be the safe level for an employee to be exposed to a material for the period of time that that employee would be working. They will put in a safety factor below, but the employees may be exposed at some level of exposure. This is taken into account by the Occupational Safety and Health Act where they give you a level above which you can't expose people.

Mr. MOLINARI. Isn't that supposed to be a safe level?

Dr. KARRH. Yes; based upon the information that is available and we draw our scientific conclusions from it.

Mr. MOLINARI. Dr. Carnow, I saw you shaking your head rather vigorously. I certainly welcome your comment on what has just been said.

Dr. CARNOW. I didn't notice my head shaking. However, the TLV, which is the threshold limiting value, which is one which has been assigned as a level workers can theoretically work at 8 hours a day, 40 hours a week for a working lifetime, every one of those that has been reviewed in the last 10 years has been changed.

I think when we think about TLV's, we have to think of them as convenient levels for industrial hygienists so when they do measures for air quality they have some kind of guidepost. I don't think anybody suggests these levels are appropriate for all workers.

In addition, like toxic waste dumps, many workers are exposed to multiple materials which may impact on a single target organ. So while TLV's are useful, like toxic waste dumps, I think there is no assurance that those levels are safe.

Let me just suggest that the arsenic level has been reduced two or three times; asbestos level, four or five times; vinylchloride level remarkably, and so on and so on. So I think that there has to be that—of course, I guess that is why I shook my head.

Mr. MOLINARI. There are, I think, 168 hours in a week. If we have a person who lives near a toxic dump who works out in the garden or is otherwise exposed for 168 hours a week, and were we to attempt to draw a comparison to somebody who works in the chemical industry who has the latest state of the art protective gear, could we draw a comparison between the two and make any assumptions that are reasonable assumptions?

Dr. CARNOW. You can make comparisons between animals and humans and various humans, but you have to be very careful about how you extrapolate your data. For example, at the level of 5 parts per million of sulfoxide, it was considered safe in the workplace. Studies we did suggested that at .14 parts per million, individuals with chronic bronchitis became ill. At .3 parts per million, less than one, some individuals who are marginal died. So it depends on the population.

We also carried out a series of studies with the National Academy of Sciences on the relationship between air pollution and cancer, particularly lung cancer. It suggests that there is a great variability of risk in individuals. And an individual who was exposed from the time he or she is born until old age to something may present a very different problem from that of a worker who in fact is part of a survival population.

Workers are the healthiest individuals in our society. Many of them, if they are working in a place where the nose or throat or chest—that can't handle the TLV, they find another job. Again, you have to be very careful about extrapolation of those kind of data.

Also, the caveats you introduced about protective devices, children eating snow and playing snow angel are different from somebody who is wearing a respirator and body suit.

Mr. **Molinari**. Doctor, wouldn't it be a fair statement in some cases that continuous low level exposure by unprotected individuals could be more harmful than the exposure experienced by somebody working in the chemical industry who, during the limited period of time that he is working with the protective gear, is exposed to higher concentrations; that the person in the first category could have a greater health risk? I say, depending upon the chemicals.

Dr. **Carnow**. There are a lot of variables. An individual working in a plant where three or four people get sick, people zero in on it. You do air quality measurements and make corrective measures. In a community, you have individuals going to various doctors. They may be going to a doctor with a rash. One doctor says it is cosmetics; another says it is tight pants, which is what was said with one group of people who were exposed to dioxin.

Again, I think that the problem we have in the community is that most of these are subtle. Acute problems are easy. If you have a spill and people are sick and dying, everybody moves in on it. It is this very insidious low-grade, seeping into a water supply, getting into the air and dust—those are the kinds of things I think we have to be concerned about here—extraordinarily difficult problems, but those are the ones we must be concerned about.

Yes; there are very significant differences. Low-level chronic exposure may very well be a serious problem. The body can compensate for a certain amount of some toxic agents. We can get rid of 300 micrograms of lead a day from our body, but when we exceed that we begin to accumulate it. So low-level, long-term exposure may be a significant problem.

Mr. **Molinari**. I am pleased to hear you say that. Not pleased, but it corroborates my reading on the subject. I question the statement, "Since we saw no increased evidence of disease among employees in the industry, we concluded that widespread incidence of chronic disease among the public is also, unlikely." I think the comparison there is not a fair comparison, and the conclusion is not a fair conclusion.

Dr. **Carnow**. Well, in a sense it is, because we can extrapolate, and we do extrapolate from effects, because the occupational setting is a microcosm of the community. I think we just have to be very careful about how we extrapolate the data.

Dr. **Karrh**. May I comment on that, Mr. Molinari?

Mr. **Molinari**. You sure may.

Dr. **Karrh**. The point I have made on that—and I have spoken it several times here today—is that we were using that to draw inferences for ourselves as to whether or not there is a pressing problem that requires immediate action. Or do we have time to address the problem properly by evaluating its extent, magnitude and what is the best solution.

All we wanted to do was see if we had something that today we had to take some type of action, or if we had 6 months or 12 months or a little bit of time to more thoroughly evaluate the problem. That is the only reason we resorted to those data.

We agree with Dr. Carnow that these data don't give us direct information, but they certainly give us some very valuable inferences that we can use in deciding our future course of action.

Mr. **Molinari**. Do you have any studies available that we could look at which show high incidence or abnormal incidence of disease where workers have been exposed to toxic chemicals on the job?

Dr. **Karrh**. Yes; we do. I will be glad to submit some for the record for you.

Mr. **Molinari**. Is there a title for that work?

Dr. **Karrh**. Acrylonitrile Epidemiological Study reported by myself to the Federal Government in May 1977, and was subsequently published in a journal that shows an increased incidence of cancer among workers exposed to acrylonitrile. That was at a time when the Government standard was much higher than what industry was operating on itself.

[The study referred to follows:]

Epidemiologic Study of Workers Exposed to Acrylonitrile

Maureen T. O'Berg, Ph.D.

This retrospective cohort study of 1,345 male employees with potential for exposure to acrylonitrile analyzes cancer incidence and mortality from 1956 to 1976. Expected numbers based on company and national rates have been computed based on person-years. The analyses consider calendar time, payroll classification, occupation, duration of exposure, severity of exposure, and latency. Overall, 25 cases of cancer occurred, with 20.5 expected based on company rates. Of these, eight were respiratory cancer cases, with 4.4 expected. Excesses were found primarily during the 1970-76 time period among wage roll employees who had worked during plant start-up. A trend toward increased risks was seen with increased duration and severity of exposure. Twenty cancer deaths were found, with 17.4 expected according to company rates. Since many cancer cases were recently diagnosed and are living, it may be premature to evaluate mortality statistics. These findings, coupled with results from tests in laboratory animals, raise the serious suspicion that acrylonitrile may be a human carcinogen.

Acrylonitrile is a chemical frequently used in the production of plastics, synthetic rubber, and fibers. It has been estimated that in the United States 125,000 workers are potentially exposed to acrylonitrile.[1] The chemical is structurally similar to the human carcinogen vinyl chloride, and, in fact, is known synonymously as vinyl cyanide. In 1977, Maltoni et al[2] reported that rats exposed to acrylonitrile by inhalation and gavage showed "borderline oncogenic effects." Preliminary results of rat studies conducted at Dow Chemical Company for the Manufacturing Chemists' Association[3] revealed excesses of papillomas of the stomach, Zymbal gland carcinomas, tumors of the central nervous system, and masses of the mammary region.

Although previous studies in man have identified acute effects of acrylonitrile,[6,7] none has specifically addressed the question of its carcinogenicity. This study was undertaken to determine whether workers potentially exposed to acrylonitrile at a DuPont textile fibers plant in Camden, S.C., exhibited increased risks of developing cancer.

Acrylonitrile has been used at the Camden plant since 1950. Work history cards showing demographic information and dates of assignment to areas where there was potential for exposure to acrylonitrile have been maintained at the plant and are available for both present and former employees in production and in maintenance.

Cohort Selection and Exposure Classification

Through an examination of more than 10,000 work history cards, 1,345 men who had had potential exposure to acrylonitrile at some time between start-up in 1950 and the cutoff date of December 31, 1966, were identified. These men were followed up until December 31, 1976. December 31, 1966, was chosen as the cutoff date for cohort eligibility so that a minimum of 10 years of follow-up would be possible for all members of the cohort. No women were included in the cohort, as none had held assignments in potential exposure areas during the years 1950 to 1966.

Plant environmental monitoring data unfortunately are not available for the 1950-1966 period. In the absence of quantitative exposure data, a qualitative assessment of severity of exposure in jobs was made by the author and a committee of six employees with long-term service in the acrylonitrile area. This committee included representatives from production, maintenance, engineering, and personnel. Manufacturing process changes and personal protection were documented chronologically in establishing the exposure levels. Three categories were used to indicate relative level of exposure: high, moderate, low. Each level is a composite measure of the intensity and the frequency of exposure, reflecting the guidelines established by the committee.

After the exposure potential of jobs had been assessed,

From the Epidemiology Section, Medical Division, E. I. DuPont de Nemours and Company, Inc., N-11400, Wilmington, DE 19898.

Completed in partial fulfillment of the Ph.D. degree in Epidemiology at the University of Pennsylvania.

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JOM 1980

three methods were used to determine whether a specific individual had ever held any of the jobs which involved potential exposure to acrylonitrile.

1. *Record Examination* — Work history cards were thoroughly examined. This was sufficient for determining exposure potential in most cases for wage roll employees and in many cases for salaried employees.

2. *Recall* — Because information contained in work history cards for wage roll personnel who worked in general services maintenance and for salary roll personnel does not always include specific assignments, it was necessary in some instances to rely on memory in determining whether or not an individual had held a job in which there was potential for exposure. Those individuals involved in recalling the jobs of persons in the cohort were members of supervision who had long service and were familiar with both the jobs and the people under consideration.

3. *Survey* — Work history cards for salary roll employees are not as detailed as those for wage roll employees. All active salary roll employees whose work history cards indicated they had worked in an area where they could have had exposure to acrylonitrile were asked to fill out a form indicating which of their assignments, if any, did constitute exposure to acrylonitrile.

Follow-up Procedures

After coding procedures had been established, pertinent data were recorded at the plant. The coded sheets were sent to the epidemiology section of the medical division where a computer tape was created. Medical data for individuals in the cohort were added to the tape. Sources of medical data include: (1) The DuPont Mortality File. This file was begun in 1957 and contains information on deaths which have occurred among all active employees and among pensioners. (2) The DuPont Cancer Registry. The Registry was begun in 1956 and includes diagnoses of cancer among active employees enrolled in the company's accident and health insurance program and a record of deaths which have occurred among active employees. Approximately 95% of company employees are enrolled in this health insurance program. Claims must involve disability of at least eight consecutive days, must indicate the diagnosis, and must be signed by the attending physician. (3) Death Certificates. Death certificates were obtained for former DuPont employees who were identified as deceased by the Social Security Administration (SSA) and were a source of additional cause-of-death information. A more detailed description of these data sources can be found in a previous publication.[8]

Of the 475 former employees whose names were sent to SSA, the vital status of 102 could not be determined. The name and date of birth of each of these persons were sent to the Motor Vehicle Division of the Department of Highways and Public Transportation in Columbia, S.C. This division provided information indicating the most recent date of driver's license renewal, when available. Former employees for whom the Motor Vehicle Division had such information were considered alive until the date of license renewal.

Additionally, a list of the names of former employees of unknown status was given to the plant committee members with instructions that vital status, when known, be recorded on the list.

Following these procedures, it was concluded that, of the 475 former employees, 41 were deceased, 402 were alive, and the status of 32 was unknown as of December 1976. In summary, vital status was determined for 98% of the cohort.

Coding of diagnoses and causes of death was done by trained nosologists in the medical division, according to the International Classification of Diseases. Death certificates were obtained from various state health departments for all 89 members of the cohort whose deaths were known to have occurred between 1957 and 1976. Furthermore, pathology slides were available for 16 of the 25 diagnosed cancer cases. These were sent to the Armed Forces Institute of Pathology, whose examination of tissue specimens confirmed in all instances the original diagnosis in the DuPont files.

Analytic Methods

The cancer incidence and the mortality experience of the cohort were analyzed. The numbers of person-years of observation were calculated as described below. For analyses of incidence (cancer diagnosed among persons actively employed at DuPont) the numbers of person-years were calculated from the date of first exposure after January 1, 1956, when the Cancer Registry was begun, until the earliest of the following events: date of cancer diagnosis, date of death, date of termination (resignation, discharge, or retirement), or December 31, 1976. Periods of unemployment (e.g., lay-off or military leave) were not included in the total number of person-years. Time periods were rounded to the nearest tenth of a year.

For mortality analyses, the numbers of person-years were calculated from the date of first employment after January 1, 1957, when the DuPont Mortality File was begun, until the date of death, or the date lost to follow-up, or until December 31, 1976, whichever occurred first. Table 1 presents, by calendar year and payroll status, the compilations of person-years of observation used in the morbidity and mortality analyses.

Expected numbers were computed using the packaged USDR program developed by Monson[9] in which age-specific rates are multiplied by the appropriate numbers of person-years to give expected numbers. The expected numbers are then summed over all age strata, resulting in an overall age-adjusted expected number. This number is compared statistically to the observed number, using

Table 1. — Person-Years of Observation of Acrylonitrile Cohort, Camden Plant

Year	Wage	Salary	Total
Morbidity analyses			
1956-64	5482	901	6383
1965-69	3563	658	4221
1970-76	4591	837	5428
Total	13636	2396	16032
Mortality analyses			
1957-64	7438	901	8339
1965-69	5511	735	6246
1970-76	7586	1019	8605
Total	20535	2655	23190

Table 2. — Observed and Expected Numbers of Cases, All Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976.

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with < 6 Months Exposure (n = 217)						
1956-1964	0	0.2	0	—	0	0.2
1965-1969	0	0.3	0	0.1	0	0.4
1970-1976	1	0.8	0	0.1	1	0.9
Total	1	1.3	0	0.2	1	1.5
Men with ≥ 6 Months Exposure (n = 1128)						
1956-1964	2	3.5	1	0.8	3	4.3
1965-1969	1	3.6	1	1.0	2	4.6
1970-1976	17	8.3*	2	1.8	19	10.1*
Total	20	15.4	4	3.6	24	19.0

*$p < 0.01$

probability tables for the Poisson distribution (one-tailed).

Cancer incidence rates obtained from the DuPont Company and from the 1969-1971 survey conducted by the National Cancer Institute (NCI) were used in the calculations. Several sets of mortality rates were used: DuPont Company rates, rates for U.S. white males, rates for South Carolina white males, and rates for white males in the five counties contiguous to the plant.

For comparison purposes, DuPont rates are preferable to those derived from the general population because the general population includes persons hospitalized or otherwise too sick to be employed and thereby yields "expected numbers" which are higher than those determined using a healthier employed population as the referent. The magnitude of an excess risk, then, is underestimated when expected numbers are based on general population rates because of the so-called "healthy worker effect." Furthermore, ascertainment of cases is generally consistent within DuPont, whereas methods employed elsewhere might differ.

Incidence Data

Through a search of the DuPont Cancer Registry from 1956 to 1976, 25 cases of cancer were identified among employees from the acrylonitrile cohort. Superficial skin cancers are not included in this count, since these tumors are not uniformly reported and seldom involve either a week's disability or death. The 25 cases of cancer include eight lung, three colon, three prostate, two bladder, and one each of thyroid, nasopharynx, penis, esophagus, brain, leukemia, lymphosarcoma, Hodgkin's disease, and malignant melanoma.

Table 3. — Observed and Expected Numbers of Cases, Respiratory Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976.

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with < 6 Months Exposure (n = 217)						
1956-1964	0	—	0	—	0	—
1965-1969	0	0.1	0	—	0	0.1
1970-1976	0	0.2	0	—	0	0.2
Total	0	0.3	0	—	0	0.3
Men with ≥ 6 Months Exposure (n = 1128)						
1956-1964	2	0.4	0	0.1	2	0.5
1965-1969	0	0.9	0	0.2	0	1.1
1970-1976	6	2.1*	0	0.4	6	2.5*
Total	8	3.4*	0	0.7	8	4.1

*$p < 0.05$

Table 4. — Observed and Expected Numbers of Cases, All Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with ≥ 6 Month's Exposure)

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with First Exposure in 1950-1952						
1956-1964	2	2.9	0	0.4	2	3.3
1965-1969	1	2.7	1	0.5	2	3.2
1970-1976	17	5.6†	2	0.8	19	6.4†
Total	20	11.2*	3	1.7	23	12.9†
Men with First Exposure after 1952						
1956-1964	0	0.6	1	0.3	1	0.9
1965-1969	0	0.9	0	0.5	0	1.4
1970-1976	0	2.7	0	0.9	0	3.6
Total	0	4.2	1	1.7	1	5.9

*$p < 0.05$
†$p < 0.01$

Two additional cases of lung cancer in individuals who should have been included in the cohort but were inadvertently omitted due to clerical error were discovered from examination of work history files for all employees with cancer at the Camden plant. Both of the employees had short duration of exposure: two months in one instance, three months in the other. Both are now deceased. These cases/deaths are excluded from the tables which follow, since proper adjustment of expected numbers is impossible. An estimate of the "miss-rate" among noncases was made by sampling one out of every 14 plant records. Among this sample of 465 employees, there were five similar omissions, giving a 1% error rate.

The numbers of cases observed were compared first with the numbers expected based on DuPont Company rates. The time span of 1956-1976 was divided into three periods. For each time period company rates for wage roll employees were applied to the number of person-years accrued among wage roll employees in the cohort. Similarly, rates for salary roll employees were applied to the number of person-years accrued among the salary roll group. Rates were computed after exluding the experience of the cohort.

Table 5. — Observed and Expected Numbers of Cases, Respiratory Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with ≥ 6 Months' Exposure)

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with First Exposure in 1950-1952						
1956-1964	2	0.4	0	0.1	2	0.5
1965-1969	0	0.7	0	0.1	0	0.8
1970-1976	6	1.5†	0	0.3	6	1.8*
Total	8	2.6†	0	0.5	8	3.1*
Men with First Exposure after 1952						
1956-1964	0	—	0	—	0	—
1965-1969	0	0.2	0	0.1	0	0.3
1970-1976	0	0.6	0	0.2	0	0.8
Total	0	0.8	0	0.3	0	1.1

*$p < 0.05$
†$p < 0.01$

Table 6 — Observed and Expected Numbers of Cases, All Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with ≥ 6 Months' Exposure) by Occupation

	Wage	
	Obs	Exp
Operators		
1956-1964	0	1.1
1965-1969	0	1.3
1970-1976	6	3.5
Total	6	5.9
Mechanics		
1956-1964	2	2.0
1965-1969	0	1.9
1970-1976	10	3.7*
Total	12	7.6
Laboratorians		
1956-1964	0	0.4
1965-1969	1	0.4
1970-1976	1	1.1
Total	2	1.9

*$p < 0.01$

The cohort was dichotomized so that results for men with exposures of less than six months and for those with exposures of at least six months were considered separately. Results for cancer, all sites combined, are presented in Table 2 and for cancer of the respiratory system, in Table 3. Numbers for other sites are too small for detailed comparisons. In the overall cohort, however, three cases of colon cancer were observed, compared with 2.2 expected, and three cases of prostate cancer were observed, with 0.9 expected, based on DuPont rates.

It can be seen in Table 2 that among the 217 persons with less than six months of exposure only one case of cancer was observed, compared to 1.5 expected. There will be no further discussion of cancer incidence among persons with less than six months of exposure. Among persons with at least six months of exposure 24 cases were observed, compared to 19.0 expected. Among these cases were eight respiratory system cancers, compared to 4.1 expected (Table 3).

Table 7 — Observed and Expected Numbers of Cases, Respiratory Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males 1956-1976 (Men with ≥ 6 Months' Exposure) by Occupation

	Wage	
	Obs	Exp
Operators		
1956-1964	0	0.1
1965-1969	0	0.3
1970-1976	3	0.9
Total	3	1.3
Mechanics		
1956-1964	2	0.3*
1965-1969	0	0.5
1970-1976	3	1.0
Total	5	1.8*
Laboratorians		
1956-1964	0	–
1965-1969	0	0.1
1970-1976	0	0.3
Total	0	0.4

*$p < 0.05$

Table 8 — Observed and Expected Numbers of Cases, All Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with ≥ 6 Months' Exposure) by Duration

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Duration < 5 years						
1956-1962	0	0.9	1	0.4	1	1.3
1963-1969	1	1.6	0	0.6	1	2.2
1970-1976	3	2.9	1	0.9	4	3.8
Total	4	5.4	2	1.9	6	7.3
Duration 5-9 years						
1956-1962	0	1.2	0	0.1	0	1.3
1963-1969	1	1.5	0	0.2	1	1.7
1970-1976	5	2.3	1	0.3	6	2.6*
Total	6	5.0	1	0.6	7	5.6
Duration > 10 years						
1956-1962	0	0.2	0	0.0	0	0.2
1963-1969	1	1.3	1	0.2	2	1.5
1970-1976	9	2.8†	0	0.4	9	3.2†
Total	10	4.3*	1	0.6	11	4.9*

*$p < 0.05$
†$p < 0.01$

The experience of men first assigned to the area in 1950-1952 during start-up operations was analyzed separately from that of men assigned after 1952. For the men whose exposure began in 1950-1952, results for cancer all sites combined, are presented in Table 4 and for cancer of the respiratory system, in Table 5. Table 4 shows that among persons with at least six months of exposure beginning after 1952, fewer cancers were observed than expected (1 compared to 5.9). Among those whose first exposures occurred during the period 1950-1952, 23 cases were observed, compared to 12.9 expected ($p < 0.01$). Twenty of these cases developed among wage roll men, with 11.2 expected, and three occurred among salary roll men, with 1.7 expected.

Generally, wage roll employees have greater potential for exposure than do salary roll employees since they are more apt to work continuously where the process is carried on. Among the wage roll employees in the 1950-1952 group, a slight deficit of two cases is seen in each of the first two time periods, whereas a significant excess of 11

Table 9 — Observed and Expected Numbers of Cases, Respiratory Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with ≥ 6 Months' Exposure) by Duration

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Duration < 5 years						
1956-1962	0	0.1	0	–	0	0.1
1963-1969	0	0.3	0	0.1	0	0.4
1970-1976	2	0.7	0	0.2	2	0.9
Total	2	1.1	0	0.3	2	1.4
Duration 5-9 years						
1956-1962	0	0.2	0	–	0	0.2
1963-1969	1	0.3	0	–	1	0.3
1970-1976	2	0.6	0	0.1	2	0.7
Total	3	1.1	0	0.1	3	1.2
Duration ≥ 10 years						
1956-1962	0	–	0	–	0	–
1963-1969	1	0.3	0	–	1	0.3
1970-1976	2	0.7	0	0.1	2	0.8
Total	3	1.0	0	0.1	3	1.1

Table 10. — Observed and Expected Numbers of Cases, All Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with > 6 Months' Exposure) by Severity

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with Only Low Level Exposure						
1956-1964	1	1.0	0	0.3	1	1.3
1965-1969	1	1.0	0	0.4	1	1.4
1970-1976	4	2.9	1	0.8	5	3.7
Total	6	4.9	1	1.5	7	6.4
All Others						
1956-1964	1	2.5	1	0.5	2	3.0
1965-1969	0	2.5	1	0.6	1	3.1
1970	13	5.5*	1	1.0	14	6.5*
Total	14	10.5	3	2.1	17	12.6

*$p < 0.01$

cases is noticeable in the period 1970-1976. This pattern is largely due to the distribution of respiratory cancers among wage roll men, there were eight cases observed, compared to 2.6 expected ($p < 0.01$). Six of these cases were diagnosed during the period 1970-1976, 1.5 were expected (Table 5).

Wage roll employees include operators, maintenance mechanics, and laboratorians. Table 6 shows, by occupation, the observed and expected numbers of cancers of all sites combined for wage roll employees with at least six months of exposure.

It can be seen that the excess cancer among wage roll men during the period 1970-1976 occurred principally among maintenance mechanics (10 cases observed, compared to 3.7 expected) and to a lesser extent among operators (6 compared to 3.5). From 1970 to 1976, three respiratory system cancers were found among maintenance workers, compared to 1.8 expected, and three among operators, with 0.9 expected (Table 7). Among laboratorians cancer incidence was close to expected.

In an effort to explore the possibility of a dose-response relationship, duration and severity of exposure have been analyzed. In the analysis of duration, numbers of person-years have been distributed so that the contribution to a given calendar year is evaluated according to duration of exposure as of that year. Thus, for a man exposed from 1960 to 1970, the number of person-years of exposure has been tallied in the 0-4 years duration-of-exposure category for 1960-1964, in the 5-9 years category for 1965-1969, in the 10+ years category after 1969. An internally developed computer program was designed for this purpose.

Table 11. — Observed and Expected Numbers of Cases, Respiratory Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Company Incidence Rates for Males, 1956-1976 (Men with ≥ 6 Months' Exposure) by Severity.

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with Only Low Level Exposure						
1956-1964	1	0.1	0	—	1	0.1
1965-1969	0	0.3	0	0.1	0	0.4
1970-1976	1	0.7	0	0.2	1	0.9
Total	2	1.1	0	0.3	2	1.4
All Others						
1956-1964	1	0.3	0	0.1	1	0.4
1965-1969	0	0.6	0	0.1	0	0.7
1970-1976	5	1.4*	0	0.2	5	1.6*
Total	6	2.3*	0	0.4	6	2.7

*$p < 0.05$

Table 12. — Comparison of Expected Numbers of Cases, All Cancer, According to Referent Population, Acrylonitrile Cohort, Camden Plant, 1956-1976

	Observed	Expected Based on NCI Rates (1969-1971)	Expected Based on DuPont Rates
1956-1964	3	6.0	4.5
1965-1969	2	6.4	5.0
1970-1976	20	13.1	11.0
Total	25	25.5	20.5

The analyses of duration are presented in Tables 8 and 9. No trends are seen among salary roll employees. Among wage roll men with a least six months of exposure the risk for cancer of all sites appears to increase with increasing duration of exposure, as summarized below.

Duration	**Observed**	**Expected**	**Ratio**
<5 years	4	5.4	0.7
5-9 years	6	5.0	1.2
≥10 years	10	4.3	2.3

The trend in ratios is stronger in the 1970-1976 period, which approximates a 20-year latency period since first exposures in the early fifties.

Duration	**Observed**	**Expected**	**Ratio**
<5 years	3	2.9	1.0
5-9 years	5	2.3	2.2
≥10 years	9	2.8	3.2

The numbers of respiratory system cancers are quite small, therefore, ratios have not been calculated.

In considering severity of exposure, results for men with only "low level" rankings have been presented separately from those for the remainder of the cohort, which includes men with some "moderate" and "high" rankings. In Tables 10 and 11 it can be seen that, in the "low level" group, cancer incidence was close to expectation: seven cases observed, compared to 6.4 expected, for all sites, two observed, compared to 1.4 expected, for respiratory system. In the "all others" category, which represents higher exposure levels, 17 cases were observed, compared to 12.6 expected, for all sites, six were observed, compared to 2.7 expected, for respiratory cancer. Again the comparisons for the 1970-76 period are more striking: 14 observed, compared to 6.5 expected, for all

Table 13. — Observed and Expected Numbers of Deaths, All Causes, Acrylonitrile Cohort, Camden Plant, Based on DuPont Mortality Rates for Males, 1957-1976

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with < 6 Months' Exposure						
1957-1964	1	2.0	0	0.1	1	2.1
1965-1969	4	2.5	0	0.1	4	2.6
1970-1976	12	6.2*	1	0.3	13	6.5*
Total	17	10.7*	1	0.5	18	11.2*
Men with ≥ 6 Months' Exposure						
1957-1964	9	11.4	2	1.7	11	13.1
1965-1969	17	13.9	2	2.2	19	16.1
1970-1976	38	32.3	3	4.7	41	37.0
Total	64	57.6	7	8.6	71	66.2

*$p < 0.05$

Table 14. — Observed and Expected Numbers of Deaths, by Cause, Acrylonitrile Cohort, Camden Plant, Based on DuPont Mortality Rates for Males, 1957-1976 (Men with ≥ 6 Months' Exposure).

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Neoplasms	15	12.8	2	2.1	17	14.9
Cerebrovascular disease	1	3.3	1	0.4	2	3.7
Diseases of the heart	29	25.3	2	4.0	31	29.3
Other cerebrovascular diseases	1	1.2	—	0.2	1	1.4
Accidents	12	6.7	1	0.6	13	7.3
Suicides	3	2.2	1	0.2	4	2.4
Other causes	3	6.4	—	1.0	3	7.4
Total	64	57.9	7	8.5	71	66.4

sites, five observed, compared to 1.6 expected, for respiratory.

The expected numbers presented thus far have been based on DuPont Company rates. In Table 12, the expected numbers based on NCI rates (Third National Survey, 1969-1971) are presented along with those based on DuPont rates. It can be seen that the expected numbers based on NCI rates are consistently higher than those based on DuPont rates. Further presentation of comparisons based on NCI rates has been omitted since the DuPont rates provide a more sensitive indicator of risks.

Mortality Data

Eighty-nine deaths were known to have occurred between 1957 and 1976 among persons in the acrylonitrile cohort. Death certificates were obtained in all instances. Table 13 presents the comparison of observed and expected numbers of deaths, based on DuPont Company rates for 1957-1976, in three time periods. Results show that among men with less than six months of exposure, 18 deaths occurred, compared to 11.2 expected. Among men with at least six months of exposure, 71 deaths occurred, compared to 66.2 expected.

Cause-specific mortality among men with at least six months of exposure is shown in Table 14. The numbers of deaths due to cancer and heart disease were slightly higher than expected. There was also excess mortality for accidents and suicides.

A more detailed presentation of mortality for cancer of all sites and for cancer of the respiratory system can be found in Tables 15 and 16 respectively. A total of 20 cancer deaths occurred in the entire cohort, compared to 17.4 expected, based on DuPont Company rates. The slight excess (18 observed, compared to 15.2 expected) was confined to wage roll men. As has been shown to be the case with incidence, any excess occurred during the period 1970-1976 (15 deaths observed, compared to 10.5 expected). Mortality from respiratory system cancers in the period 1957-1976 differed little from expectation: 8 deaths observed, compared to 7.1 expected.

Additional mortality comparisons have been made based on rates from the general white male population of the United States, the white male population of the state of South Carolina, and the white male population of the five counties contiguous to the Camden, S.C., plant. These comparisons are displayed in Table 17, along with the DuPont-based expected numbers. It can be seen that, as was the case with morbidity, the numbers of expected deaths based on the experience of the U.S. white male population are higher than those based on DuPont figures. Expected numbers of cancer deaths based on the state and county rates are slightly higher than those based on the U.S. rates, and again are higher than the DuPont expected numbers.

Discussion

The incidence data in this study show increased cancer risks for some subgroups within the acrylonitrile cohort. These data are limited to persons who develop cancer while still employed at DuPont. The mortality follow-up is more comprehensive in that it includes pensioners and former employees. However, most of the diagnoses have been recent — too recent in many instances to have resulted in death. Thus far, mortality findings in this study do not reveal an association between acrylonitrile and cancer, but it may be premature to evaluate mortality properly.

Table 15. — Observed and Expected Numbers of Deaths, All Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Mortality Rates for Males, 1957-1976

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with < 6 Months' Exposure						
1957-1964	0	0.4	0	—	0	0.4
1965-1969	1	0.5	0	—	1	0.5
1970-1976	2	1.4	0	0.1	2	1.5
Total	3	2.3	0	0.1	3	2.4
Men with ≥ 6 Months' Exposure						
1957-1964	1	2.2	0	0.4	1	2.6
1965-1969	3	2.9	0	0.5	3	3.4
1970-1976	11	7.8	2	1.2	13	9.0
Total	15	12.9	2	2.1	17	15.0

Table 16. — Observed and Expected Numbers of Deaths, Respiratory Cancer, Acrylonitrile Cohort, Camden Plant, Based on DuPont Mortality Rates for Males, 1957-1976.

	Wage		Salary		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
Men with < 6 Months' Exposure						
1957-1964	0	0.1	0	—	0	0.1
1965-1969	0	0.2	0	—	0	0.2
1970-1976	1	0.6	0	—	1	0.6
Total	1	0.9	0	—	1	0.9
Men with ≥ 6 Months' Exposure						
1957-1964	0	0.5	0	0.1	0	0.6
1965-1969	1	1.0	0	0.1	1	1.1
1970-1976	5	3.1	1	0.4	6	3.5
Total	6	4.6	1	0.6	7	5.2

Table 17. — Comparison of Expected Numbers of Deaths According to Referent Population, Acrylonitrile Cohort, Camden Plant.

	Observed	DuPont	U.S. White Males	Expected Based on S.C. White Males	White Males in Counties Contiguous to Plant
All deaths	89	77.4	121.1	156.0	143.5
Cancer deaths	20	17.4	22.4	24.2	24.5
Respiratory cancer deaths	8	7.1	7.4	9.7	10.3

Causal relationships are difficult to establish in epidemiologic studies, since rigorous control of pertinent variables is impossible. Several criteria are used in determining whether an association is likely to be causal.[10] The strength of the association is considered. In this study the overall cancer standardized incidence ratio (SIR) for cancer based on DuPont Company rates for persons with at least six months of exposure is 126. The standardized mortality ratio (SMR) is 113. Within the entire cohort, the wage roll group works most directly with the acrylonitrile process. Furthermore, it was during start-up (1950-1952) that exposures were least contained. If the strength of the association is examined for wage roll employees who had at least six months of exposure and who began assignments during start-up, the SIR increases to 179 and the SMR to 143.

A dose-response relationship lends credence toward establishing causality. In this study, small numbers and a lack of quantitative exposure data hinder attempts to study dose-response. Nonetheless, a trend in the direction of increased risks can be gleaned from comparison of observed and expected incidence in three subgroups with increasing duration of exposure: less than 5 years, 5-9 years, and at least 10 years. The SIR increases from 0.7 to 1.2 to 2.3, respectively. Incidence analyses using the qualitative severity assessments show that persons with only "low" level exposures have close to expected cancer experience (SIR = 109), whereas those with higher exposures have higher risks (SIR = 135).

Temporal correctness of the association is an important consideration. In this study, the data are consistent with the hypothesis that a carcinogen appeared in 1950 and that excess cancer appeared 20 years later. Of the 24 cases which developed among men with area assignments of at least six months, 17 occurred at least 20 years after initial exposure. Of the eight cases of respiratory system cancer, six developed 20 years after first exposure. For the period 1970-1976, the SIR for cancer, all sites, for wage roll men who had had at least six months of exposure and who had worked during start-up is 304 and the SMR is 177.

The latency issue is important in the study of carcinogenicity. Most cancers develop long after initial contact with causative agents. Latency periods of 20 to 30 years are quite common with many known human carcinogens, e.g., asbestos, betanaphthylamine, chromium, and vinyl chloride.[11] The temporal relationship between initial exposure and increased risks in this study is consistent with existing knowledge.

Another criterion used to determine whether an association is causal is the consistency of the association across many studies. No other epidemiologic studies addressing the carcinogenicity of acrylonitrile have been published, and hence results of this study cannot yet be supported or refuted by other research. Animal studies have demonstrated carcinogenic response by several routes of administration. These offer some support and insight. A variety of tumors was found in rats, as was the case in this cohort. There is no correspondence of primary sites between the rat studies and this study. Specificity of response and organ consistency between animal species and man would provide a stronger case for causality. The multiplicity of primary sites may not be without biologic plausibility though. Acrylonitrile is carried throughout the bloodstream in humans, and thus could reach many target organs and systems.

Other factors which must be considered require discussion. Cigarette smoking is one. Smoking histories were unavailable at the Camden plant, but an effort was made to obtain information on the smoking habits of all persons in the cohort who developed cancer. Contact was made with employee associates and members of supervision to determine the cigarette smoking history of these individuals. It was possible to obtain this information for all except one of the employees who developed lung cancer. All for whom this information was available were smokers; however, this does not rule out an etiologic role for acrylonitrile, either alone or in synergy with cigarette smoking.

Other exposures must also be considered. The cohort was explicitly selected because of assignment to areas where acrylonitrile was present. This exposure did not occur in isolation, however. Exposure to dimethyl formamide, methyl acrylate, and other chemicals could also have occurred in many instances, but these substances are not suspected carcinogens. Previous or subsequent employment could also have involved other exposures. Personal factors such as diet, use of medication, and ethnicity have not been ascertained. These factors are known to affect the development of cancer. The problem of confounding factors is not unique to this study, nor to occupational studies in general, but it must be kept in mind when interpreting results.

Summary and Conclusions

Among 1,345 male employees who had potential for exposure to acrylonitrile at the Camden plant, 25 cases of cancer occurred, compared with 20.5 expected, based on DuPont Company rates. The excess was primarily due to respiratory cancer, with eight observed cases versus 4.4 expected.

These cancer cases were diagnosed primarily during the period 1970-1976, when highly significant excesses of all cancers and of respiratory cancers were seen. The excesses were found among wage roll employees who worked during the 1950-1952 plant start-up. A trend toward increased risks of cancer of all sites was seen with increased duration of exposure and with higher severity of exposure.

Eighty-nine deaths occurred among men in the cohort. A very slight excess of cancer deaths was found (20 deaths vs 17.4 expected). Since many cases were diagnosed recently in persons who are still alive, it may be premature to interpret mortality statistics.

These findings, coupled with results from tests in laboratory animals, raise the serious suspicion that acrylonitrile may be a human carcinogen. Further studies are needed to confirm or refute what appears to be an increased risk in certain subgroups of this cohort.

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Mr. MOLINARI. Any other studies that you can recall?

Dr. KARRH. Back in the 1930's——

Mr. MOLINARI. Any more recent ones? I am talking about the last 10 years or so.

Dr. KARRH. Well, some of the vinylchloride studies where there are some increased incidences, some asbestos studies. These are all occupational studies. I will be glad to submit some for the record.

Mr. MOLINARI. I would appreciate it if you would.

[The information received follows:]

ER-0001 REV 2-81

DUPONT
ESTAB. 1802

E. I. du Pont de Nemours & Company
INCORPORATED
Wilmington, Delaware 19898

EMPLOYEE RELATIONS DEPARTMENT

BWK/File

July 26, 1983

The Honorable Donald K. Sundquist
The United States House of
  Representatives
515 Cannon House Office Building
Washington, DC 20515

Dear Congressman Sundquist:

During my testimony on July 21, 1983, I discussed a study of Hardeman County residents. You asked that I provide a copy of the study. It is attached and will also be submitted for inclusion in the record with the other information requested by the Subcommittee on Investigations and Oversight of the Committee on Public Works and Transportation.

Very truly yours,

Bruce W. Karrh
General Director
Medical, Safety & Fire Protection

BWK:mdm
Enclosure

Submitted for publication October 8, 1981; accepted for publication November 29, 1981.

Requests for reprints should be addressed to: Jane Q. Koenig, Department of Environmental Health, SC-34, University of Washington, Seattle, WA 98195.

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An Environmental Health Survey of Drinking Water Contamination by Leachate from a Pesticide Waste Dump in Hardeman County, Tennessee

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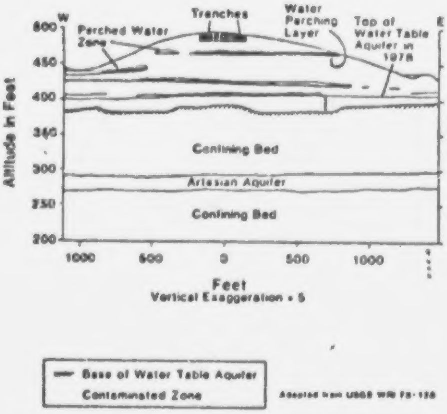
ABSTRACT. Ground water that was used as a source of potable water by residents of a small community became contaminated by leachate from a 300,000 barrel pesticide waste dump. An environmental health survey of the residents and an apparent control group was conducted to determine if any adverse health effects resulting from exposure to the toxic compounds, many of which were hepatotoxins, could be detected. The survey utilized a health questionnaire, a clinical examination, and a biochemical screening. The latter included analysis of serum for liver and kidney function parameters, determination of hepatitis A and B serology, and bile acid determinations on fasting and postprandial serum and urine specimens. Air and water from representative homes and urine from study participants were analyzed for selected organic compounds. The biochemical screening was conducted on two occasions 2 months apart. The initial hepatic profile testing revealed elevated concentrations of the serum enzymes, alkaline phosphatase and serum glutamic oxaloacetic transaminase, in residents who had used the contaminated water. During follow-up testing of these same persons 2 months later, these values were significantly reduced, as were postprandial serum concentrations of the bile acid of cholyglycine. Six individuals in the exposed group had slight hepatomegaly compared to one individual in the intermediate exposure group and none in the controls. Concentrations of carbon tetrachloride were as high as 18,700 μg/L in the well water samples. The biochemical and clinical observations are suggestive of a transitory liver injury probably related to exposure to the contaminated drinking water.

TOXIC WASTE DUMPS are a major source of environmental pollution in many areas of the country and are being recognized with increased frequency. In Hardeman County, Tennessee, wastes from a Memphis pesticide manufacturer have contaminated the groundwater aquifer serving approximately 60 nearby residents. During the years of operation, 1964–1972, an estimated 300,000 barrels of liquid and solid waste were buried in shallow trenches dug into the ridges at the 200-acre dump site. The dump was closed by state authorities in 1972 because contamination had been detected in test wells proximal to the burial areas.[1,2] The analyses performed at that time did not reveal any contamination in the three private water wells closest to the dump. During the fall of 1977, however, nearby residents became alarmed upon detection of unusual and unpleasant tastes and odors in their well water. Within a short time, the residents reported an unusually high number of symptoms including skin and eye irritation; weakness in the upper and lower extremities; upper respiratory infection; shortness of breath; and severe gastrointestinal symptoms, including nausea, diarrhea, and abdominal cramping. In general, these symptoms appeared to subside once ingestion of the contaminated water was stopped. A 1978 investigation by the U.S. Geological Survey confirmed the residents' observations that contamination of the local water supply aquifer had occurred[3] (Fig. 1). In an effort to determine the extent and possible etiology of the signs and symptoms of illness, a limited health screening study was conducted in November, 1978, when the U.S. Environmental Protection Agency strongly advised that all use of the water cease. The results from this study showed biochemical changes suggestive of liver injury in some of the residents whose water was contaminated. A second more extensive evaluation was conducted in January, 1979.



Adapted from USGS WRI 78-138

Fig. 1. Conceptual diagram of contaminant percolation below the disposal trenches.

Background. The Hardeman County toxic waste dump first came to our attention during the course of a seroepidemiologic research study of wastewater treatment plant personnel in Memphis, Tennessee, located approximately 60 miles from the dump site. One of the two treatment plants in this study served an area which included the pesticide manufacturer whose waste had formerly been deposited in the Hardeman County toxic waste dump. A portion of the wastes became airborne at the treatment plant because of the aeration process utilized, and because of the open basins that are generally used in such facilities. Workers complained of eye irritation and respiratory distress in the spring of 1978 during periods of time when chemical odors at the treatment plant were unusually intense. In conjunction with the ongoing study at the treatment plant, a urine screening study of the workers was conducted in May and June, 1978, and air samples were collected at various locations in the treatment plant. Hexachlorocyclopentadiene (HEX) and hexachlorobicycloheptadiene (HEX-BCH), two compounds produced by the pesticide manufacturer, were detected in air, wastewater, and urine specimens obtained at that time. In the June testing, workers at the Memphis North Treatment Plant had increased concentrations of the HEX-BCH compound in urine specimens collected late in the work shift compared to concentrations in urine specimens collected early in the work shift.[4]

By late 1978, the U.S. Environmental Protection Agency and the State of Tennessee Department of Health had detected more than 12 chlorinated organic compounds in the wells serving individuals living near the dump site.[5] Many of these chemicals (Table 1) are on the National Priority Pollutant Consent Decree List.[6] Carbon tetrachloride was the most abundant contaminant detected. Two of the other compounds in the dump are HEX and HEX-BCH.

Carbon tetrachloride, a highly toxic substance,[7,8] can be absorbed through the lungs, gastrointestinal tract, and skin; gastrointestinal and hepatorenal injuries are most prominent in poisoning from ingestion, and nervous system injuries predominate in inhalation poisoning. In all poisonings, the liver is the most drastically affected organ, but kidney injury may be more prominent clinically. A committee of the National Academy of Sciences–National Research Council has suggested that at a dose of 1 μg/day, a 5×10^{-8} lifetime risk of cancer exists.[7]

Among the most toxic chemicals known to have been dumped at the Hardeman County site are HEX and HEX-BCH. The occupational air standard[9] for HEX of 100 μg/m^3, much lower than that for carbon tetrachloride

($65,000 \mu g/m^3$), reflects that it is a much more toxic compound. Hexachlorocyclopentadiene is a potent irritant and is readily absorbed through the skin. Industrial workers often experience irritation of the eye and upper respiratory tract, and headache upon exposure to HEX vapors; burns result upon contact with the liquid. Animal studies have indicated that long-term exposures may result in a number of nonspecific adverse health effects.[10] No epidemiologic studies of workers who are occupationally exposed to HEX have been reported,[11,12] and there is no information available concerning the potential health effects resulting from chronic environmental exposure to HEX via contaminated air and/or water. Even less is known of the toxic effects of HEX-BCH, but there are indications that HEX-BCH may be as toxic as HEX.[13]

Populations studied. Sixty-one exposed individuals in 16 households along the Toone-Teague Road in Hardeman County, Tennessee, used water from wells containing carbon tetrachloride in concentrations exceeding 150 µg/L. Fifty-nine of these individuals participated in at least one of the health evaluations; 31 participated in both evaluations. An intermediate exposed group of 33 participants consisted of 8 individuals from two households along Toone-Teague Road whose wells contained up to 45 µg/L carbon tetrachloride and other people who were either close neighbors of or frequent visitors to families with contaminated wells. The location of the affected wells in relation to the dump site is shown in Figure 2. The "unexposed" control population was recruited from civic organizations and church groups elsewhere in Hardeman and adjacent counties.

Table 1.—Contaminants Detected* in Private Wells Serving Exposed Group Participants in Toone-Teague Area of Hardeman County, Tennessee

ompound	NP/NT†	Range (µg/L)	Median (µg/L)
Benzene‡	7/ 7	5-15	12
Carbon tetrachloride‡ (CCl_4)	15/15	61-18,700	1,500
Chlordene	5/24	Trace-0.81	Trace
Chlorobenzene‡	23/25	Trace-41	5.0
Chloroform‡ ($CHCl_3$)	14/15	2.1-1,890	140
Hexachlorobutadiene‡	22/28	Trace-2.53	0.15
Hexachloroethane‡	19/31	Trace-4.6	0.26
Hexachlorobicyclohepta-diene (HEX-BCH)	24/31	Trace-2.2	0.05
Methylene chloride‡	11/11	1.5-160	45
Napthalene‡	13/31	Trace-6.7	ND§
Tetrachloroethylene‡	27/28	Trace-2,405	3.5
Toluene‡	14/24	0.1-52	0.6
Xylenes	2/ 3	0.07-1.6	0.07

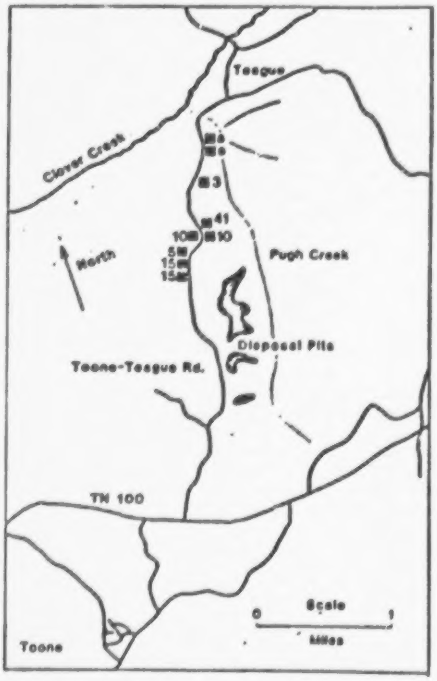
*Adapted from U.S. Environmental Protection Agency, Region IV, March 9, 1979, Summary of USEPA and State of Tennessee Analyses.[8] Endrin, heptachlor, hexachlorocyclopentadiene and octachlorocyclopentene were not detected.

†NP = Number of samples with detectable amounts of the substance tested; NT = total number of samples tested.

‡On National Priority Pollutants Consent Decree List.[6]

§D = Not detected.

Fig. 2. Location of contaminated private wells near Hardeman County toxic waste dump (▪) and percent of exposed group using each well.



a. These wells were contaminated to a lesser degree and were used by two intermediate-exposed households.

Participants who had been occupationally exposed to pesticides (including those who worked at the dump site); who had consumed alcohol 24 hr prior to the collection of the blood specimens, or who regularly consumed in excess of 30 units of alcohol per month; or who had a history of a liver disorder were deleted from the analysis of data. The age and sex profile of the resulting exposed, intermediate exposed, and control groups is presented in Table 2.

METHODS

The protocol for the November, 1978, and January, 1979, health evaluations is outlined in Tables 9 and 10.

Many of the households had stopped consuming the contaminated water by May, 1978, and all apparently intended to heed the U.S. Environmental Protection Agency's advice (November, 1978) to stop all other uses of the water. The initial limited health screening was conducted in November, 1978, in an attempt to evaluate the residents while any physical or biochemical changes were still evident. Because of this limited time, and because of limited personnel, the study was designed to include: (1) analysis for selected organic compounds in air and water samples collected from some homes where wells were affected; (2) urinalysis for detection of selected organic chemicals; (3) blood analysis to determine liver profile and nonfasting serum bile acids; and (4) a questionnaire, including alcohol and drug intake, as well as past history of hepatitis or other liver abnormalities, to be administered by the public health nurses involved in the study. This study was conducted primarily to ascertain whether these chemicals, many of which are known hepatotoxins, were associated with liver dysfunction in the concentrations at which they were found, and to exclude other sources of liver disease.

The blood specimens were centrifuged and the serum was frozen and shipped to the Clinical Laboratory of Cincinnati General Hospital for performance of routine liver function determinations, including serum gamma glutamic transaminase (SGGT), serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphatase, and total bilirubin. Urine specimens and water samples were frozen immediately after collection and an aliquot of each was saturated with sodium chloride and extracted with an equal volume of petroleum ether. The petroleum ether extracts were stored in a freezer until time of analyses.

Air samples were collected using glass sampling tubes containing 150 mg of pre-extracted chromosorb 102 and desorbed with petroleum ether. All specimens were analyzed by electron capture gas chromatography using glass columns, packed with OV-101 or OV-17/QF-1, at 175°C, and were performed at the laboratories of the Department of Environmental Health at the University of Cincinnati, College of Medicine, Cincinnati, Ohio.

A follow-up study in January, 1979, included a more detailed questionnaire regarding past medical history, alcohol intake, drug consumption, history of hepatitis and other liver abnormalities, as well as a general health survey. In addition, experienced physicians conducted a blinded clinical examination to assess blood pressure, liver span (by percussion), and other physical parameters. While participants were fasting, serum and urine were obtained for determination of bile acids; liver and renal profiles and hepatitis serology were also performed. Postprandial serum and urine specimens were collected 2 hr after a breakfast of eggs, bacon or sausage, juice, toast, and coffee or milk, for analyses for bile acids.[14]

Representative air and water samples were collected from residences of the exposed, intermediate-exposed, and control groups. In some cases, indoor air samples were collected in bathrooms with either the bathtub or the shower tap running.

Statistical analysis included testing the dependent variables for normality and making transformations when necessary. Analysis of covariance was used to assess the effects of age, sex, monthly alcohol consumption, and their interactions on the dependent control variables. Adjustments were made for those covariables found to be significant. Analysis of variance was used to test whether significant differences between the exposed, intermediate-exposed, and control groups existed. When all three groups were tested simultaneously, analysis of variance with 2 degrees of freedom was used. Since this analysis involved multiple comparisons, the significance level was set at .02.

To test whether the individuals in the study group who were tested in both November, 1978, and January, 1979, showed a significant change in their hepatic profile and non-fasting serum bile acids, the differences between the results of the two periods were analyzed. A *t* test was then performed on the differences. Informed consent was

Table 2.—Age and Sex Profile of Exposed, Intermediate-Exposed, and Control Groups in Hardeman County Study (January, 1979)

Age (yr)	No. Exposed*		No. Intermediate-Exposed		No. Controls	
	Male	Female	Male	Female	Male	Female
0-14	6(5)	13(9)	3	12	4	7
15-24	2(2)	3(2)	0	2	1	4
25-34	5(4)	8(8)	5	7	7	10
35-44	3(0)	1(1)	0	0	3	4
45-54	0(0)	1(2)	0	0	2	2
55-64	3(3)	1(0)	1	1	1	7
65 and over	1(0)	2(0)	1	1	1	3
Total	20(14)	29(22)	10	23	19	38†

*Age and sex profile of population observed in November, 1978, indicated in parentheses.
†The age of one female was unknown.

Table 3.–Indoor Air Concentrations of Selected Organic Compounds in Houses with Contaminated Groundwater Supply (November, 1978)

Compound	No. Samples	No. Sample Concentrations above Detection Level	Concentration Range ($\mu g/m^3$)
Hexachlorocyclopentadiene	5	3	0.06-0.10
Hexachlorobicycloheptadiene	5	0	—
HEX-VCL*	5	1	0.6
Chlordene	5	0	—
Carbon tetrachloride	5	5	3.6-41
Tetrachloroethylene	5	4	2.4-5.7

*Heptachlorobicycloheptene

obtained from all study participants after the study procedures had been fully explained to them.

RESULTS

Water use. From May to December, 1978, a trailer-mounted water tank served as a temporary supply for kitchen and drinking uses for many residents. A second temporary supply, piped from an underground holding tank to a number of the residences, was installed in December, 1978. A permanent water supply from the village of Toone was connected to most of the residents in 1979. On the day blood and urine collection from Hardeman County resi-nts in November, 1978, many people in the affected area received certified letters from the U.S. Environmental Protection Agency (Region IV) advising them not to use their well water for any purposes because of high contamination with carbon tetrachloride. Some of the residents had stopped potable use of the water (i.e., drinking and cooking) beginning as early as the fall of 1977. A large group stopped use in May, 1978, after being advised by local public agencies that the water was unsafe. By August, 1978, practically all had ceased potable use of the contaminated water. Thus, any abnormalities reflected by the specimens collected in the November, 1978, survey would, for the most part, result from current exposure from washing and toilet uses, and prior exposure from drinking and cooking. By the time of the January, 1979, survey all uses of the contaminated water had ceased except for some use of contaminated water by two intermediate-exposed households.

Environmental monitoring. Detectable levels of HEX were found in the indoor air of three of the five exposed households examined in November, 1978 (Table 3). The highest concentration was 0.10 $\mu g/m^3$. Another related component, heptachlorobicycloheptene, was detected at 0.60 $\mu g/m^3$. Carbon tetrachloride was detected in all five homes and tetrachloroethylene in four of the five. In one of these homes two bathroom air samples were collected earlier in August, 1978: one sample with no water running nd the second with the shower on and the door closed. he concentration of carbon tetrachloride was 23 $\mu g/m^3$ in the first sample and 3600 $\mu g/m^3$ in the second. The higher value is approximately 5% of the American Conference of Governmental Industrial Hygienists (ACGIH) recommended occupational standard for workplace air.[9]

Sources of domestic water for the homes of the January, 1979, participants were analyzed. A summary of indoor and/or outdoor air and water samples collected from 37 homes are presented in Table 4. Water samples were designated as temporary (T), individual wells (I), and public (P). A temporary water supply was in use for all homes of exposed participants. Inside air samples were collected in the bathrooms with the shower or bathtub tap running. Water samples containing the highest concentrations of carbon tetrachloride and tetrachloroethylene were from the homes of exposed and intermediate-exposed participants. In general, samples from the control group had typical background levels.[15,16] Carbon tetrachloride and tetrachloroethylene were detected in most air samples, regardless of the group tested. Of the six control homes using a public water supply, one had an inside air sample containing more than 1 $\mu g/m^3$ carbon tetrachloride. Low levels of carbon tetrachloride (i.e., $\leq$ 1.1 $\mu g/L$) were detected in some routinely chlorinated public water supply samples. Individual private well samples, except those along Toone-Teague Road, did not contain detectable amounts of either compound.

Analysis of urine samples for selected organic compounds. Urine specimens collected from the exposed group in November, 1978, and from 137 participants from the three groups of participants in January, 1979, were screened for the presence of HEX, HEX-BCH, hexachlorobicycloheptene, chlordene, and heptachlor. No sample showed the presence of the selected chlorinated organics. A select group of nine individuals judged to have relatively high potential for exposure to carbon tetrachloride in January, 1979, were sampled for determination of carbon tetrachloride. Five of the nine persons were from the exposed group and four from the intermediate-exposed group. These intermediate-exposed participants included individuals who were using water containing about 50 $\mu g/L$ of carbon tetra-

Table 4.—Comparison of Analyses* of Water and Air Samples of Residences of Study Participants (January, 1979)

Group and Sample Type§	Carbon Tetrachloride† Water ng/L NP/NT	Carbon Tetrachloride† Air Samples‡ (µg/m³) Outside NP/T	Carbon Tetrachloride† Air Samples‡ (µg/m³) Inside NP/T	Tetrachloroethylene† Water ng/L NP/NT	Tetrachloroethylene† Air Samples (µg/m³) Outside NP/NT	Tetrachloroethylene† Air Samples (µg/m³) Inside NP/NT
Exposed						
T	0/2	2/2# (0.5-75)‡	2/2 (0.6-6.3)	0/2	1/2 (0.4)	2/2 (1.0-1.2)
I∥	1/1 (29,450)	1/1 (326)	NS**	1/1 (9.0)	1/1 (1.2)	NS
Intermediate-exposed						
I	3/10 (0.3-96)	4/6 (0.4-19)	6/6 (0.6-681)	3/10 (0.2-6.4)	5/6 (0.3-7.2)	6/6 (1.0-14.3)
P	0/1	NS	1/1 (0.7)	0/1	NS	1/1 (3.0)
Control						
I	0/17	3/4 (0.2-11.2)	3/5 (0.2-0.6)	0/17	4/4 (0.3-3.0)	5/5 (0.3-0.8)
P	3/6 (0.7-1.1)	6/6†† (0.1-13.5)	3/6 (0.2-63.1)	3/6 (0.4-1.4)	6/6 (0.1-11.8)	6/6 (0.2-40.8)

*Samples were also analyzed for: hexachlorocyclopentadiene (HEX), hexachlorobicycloheptadiene (HEX-BCH), heptachlorobicycloheptene, chlordene, and heptachlor. Unless otherwise noted, none of these substances were detected.

†All numbers in parentheses represent the range.

‡Inside samples were collected in a bathroom with water running. Outside samples were collected within 10 m of the house.

§The types of water supplies in use were: T = temporary supply provided for homes with contaminated wells; I = individual wells; and P = public supply.

#One outside air sample also contained 3.2 µg/m³ of HEX-BCH.

∥This sample was obtained from a well known to be contaminated and thus no longer in use. The sample was also found to contain 2.5 µg/L HEX-BCH and 390 mg/L chloroform. The air sample, collected during and after water was drawn from the well, contained 0.6 µg/m³ HEX-BCH.

**NS = No sample obtained.

††Two indoor air samples also contained 4.6 and 1.5 µg/m³ of heptachlor.

chloride. No evidence was found for carbon tetrachloride in any of these urine specimens. These findings with human urine specimens are consistent with animal studies[17, 18] which show that (1) carbon tetrachloride administered to mammals is largely excreted through the lungs, (2) only a small fraction of absorbed material is metabolized, and (3) it is not excreted as the parent compound in the urine. It was, therefore, concluded that analysis of additional urine specimens for carbon tetrachloride was not warranted at that time.

Hepatitis serology. To determine if the results of the tests of liver function were due to a prior history of infection with hepatitis, antibodies to hepatitis A and B were determined. A log-linear model accounting for age was used to determine that the incidence of positive hepatitis serology was not significantly different between the groups.

Liver function tests. The results of the comparisons of the exposed group in November, 1978, and the controls are presented in Table 5. Significant differences were found for several of the tests; alkaline phosphatase and SGOT levels were significantly higher in the Hardeman County specimens ($P = .016$ and .010, respectively).

A comparison of the results for the 31 exposed individuals who were tested in both November, 1978, and January, 1979, showed a statistically significant change for all 4 variables with alkaline phosphatase, SGGT, SGPT, and SGOT—all decreasing from November, 1978, to January, 1979 (Table 6). No significant differences were detected between the results for any of the groups in January, 1979.

Renal function tests. Results of renal function tests were normal and showed no differences between the groups.

Bile acids. For 23 individuals in the exposed group, group bile acid determinations were made on non-fasting serum specimens collected in both November, 1978, and January, 1979. A comparison of the November, 1978, and January, 1979, results (Table 7) revealed that serum SLCC concentrations were significantly higher in November, 1978, than in January, 1979, suggesting a degree of hepato-

Table 5.—Hepatic Profile Comparison of Hardeman County Exposed Group [illegible] and Control Group

Parameter*		Results November, 1978 Exposed Group	Control Group	Significance of Difference (t test)
Alkaline phosphatase (32-72 mU/ml age > 21, 25-150 mU/ml age < 21)	Mean†	88.1	61.5	.016
	Range	34-360	31-220	
	No. above normal /total tested	17/36	8/56	
Serum gamma glutamic transaminase (5-29 mU/ml)	Mean†	9.47	11.56	.430
	Range	2-54	4-56	
	No. above normal /total tested	3/36	3/56	
Albumin (3.5-5.0 gr/dl)	Mean	4.35	4.93	.0001
	Range	3.9-4.8	4.2-6.2	
	No. below normal /total tested	0/36	0/57	
Total bilirubin (0.1-1.1 mg/dl)	Mean†	.240	0.51	.0001
	Range	0.1-0.8	0.2-1.7	
	No. above normal /total tested	0/31	4/52	
Serum glutamic pyruvic transaminase (5-25 mU/ml)	Mean†	15.9	14.25	.324
	Range	9-50	6-70	
	No. above normal /total tested	5/36	4/56	
Serum glutamic oxaloacetic transaminase (8-22 mU/ml)	Mean†	19.5	16.08	.010
	Range	12-36	9-140	
	No. above normal /total tested	11/36	7/56	

*Normal range indicated in parentheses.
†Geometric mean.

Table 6.—Comparison of Hepatic Profile Test Results for Exposed Participants Tested in November, 1978, and January, 1979, Studies

Variable	N	Mean Difference*	Standard Error	t	df	P
Hepatic profile						
Alkaline phosphatase	31	13.3	6.3	2.125	30	< .0409
SGGT [log (5-SGGT)]	31	-2.6	1.1	2.302	30	< .0284
Albumin	31	- .5	.13	3.922	30	< .0004
Total bilirubin	26	- .2	.03	6.405	25	< .0001
SGPT	31	3.6	1.2	3.044	30	< .0048
SGOT	31	4.4	1.0	3.989	30	< .0003

NOTE: SGGT = serum gamma glutamic transaminase; SGPT = serum glutamic pyruvic transaminase; and SGOT = serum glutamic oxaloacetic transaminase.
*November results minus January results.

toxicity with resolution.

Results of analyses of fasting and non-fasting serum for specific bile acids—sulfated conjugates of lithocholate (SLCC) and cholyglycine (CG)—appear in Table 8. The mean non-fasting serum SLCC values were significantly lower in the exposed group than in the control. The clinical significance of this finding is unknown, since SLCC increases in most liver diseases; however, the lower levels may reflect a decrease in SLCC synthesis resulting from an enzyme inhibition.[19]

As expected, the mean change in SLCC and CG from the fasting to the non-fasting serum specimens was positive for all groups. The results of urine bile acid determinations were not different between the groups, except that the mean change in CG was negative for the exposed group and positive for the controls.

Medical histories. Medical questionnaires, which focused on illnesses during the 12-month period prior to the study, revealed that 11 Hardeman County residents were hospitalized. Complaints included gastrointestinal abnormalities, respiratory difficulties, neurological symptoms, muscular complaints, and high fever. Other individuals reported symptoms such as dizziness, headache, skin rashes, nausea and vomiting, numbness of limbs, and menstrual irregularities. During this same time period the only birth was a low birth weight infant with gastrochisis and eventration of intestines. Seven adults and five children reported vision problems. One individual in the control group had a severe myocardial infarction and was hospitalized. Another person in the control group had choroid retinitis.

Physical examination. One hundred eighteen individuals were examined: 48 in the exposed group, 24 in the intermediate-exposure group, and 46 controls. During physical examination, 7 individuals (6 exposed and 1 intermediate) had borderline liver enlargement (slight hepatomegaly). A significant difference between the groups was found ($P = .034$) using the Pearson chi-square test. No significant skin or eye abnormalities were detected.

DISCUSSION AND CONCLUSIONS

Concentrations of carbon tetrachloride, a suspected carcinogen, found in the contaminated well water of the

Table 7.—Comparison of Serum Bile Acid Levels Found in Samples Collected in November, 1978, with Levels in Serum Samples Collected from the Same Individuals in January, 1979

Serum Bile Acids	*N*	Mean Difference*	Standard Error	*t*	*df*	*P*
SLCC-nonfasting [log (SLCC + 1.5)]†	23	0.22	0.09	2.475	22	< .0211
CG-nonfasting	23	0.32	0.23	1.401	22	> .1745

NOTE: SLCC = sulfated conjugates of lithocholate and CG = cholyglycine.
*November results minus January results.
†For SLCC the data required a log transformation of the serum concentration plus a constant to produce a normal distribution.

Table 8.—Comparison of Mean Bile Acid Levels in Fasting and Postprandial (2 hr) Serum Samples Collected from Participants in January, 1979 Study

Serum Bile Acids	Groups					
	Exposed Mean ±	SE*	Intermediate-Exposed Mean ±	SE*	Control Mean ±	SE*
SLCC-fasting	0.769	.091(48)	0.878	0.136(29)	0.835	.066(57)
SLCC-nonfasting	1.106	.078(42)†	1.490	0.220(24)	1.526	.112(56)†
SLCC-difference‡	+.466	.071(42)	+0.628	.157(24)	+0.683	.087(56)
CG-fasting	0.268	.046(48)	.255	0.053(29)	0.375	.063(57)
CG-nonfasting	1.299	.135(42)	1.081	0.133(24)	1.237	.101(55)
CG-difference‡	+.987	.123(42)	+0.861	.157(24)	+.861	.105(55)

NOTES: Values in parentheses are number of observations. SLCC = Sulfated conjugates of lithocholate; GC = cholyglycine.
*SE = Standard error.
†Significance between groups at $P < .02$.
‡Nonfasting result minus fasting result.

exposed group (median 1500 μg/L and maximum 18,700 μg/L) are considerably higher than the 2.6 μg/L level that has been proposed as a safe level for drinking water.[20] In comparison, the releases of carbon tetrachloride into the Ohio River in February, 1977, ranged only up to approximately 340 μg/L.[21] The highest concentrations of carbon tetrachloride found in air samples during this study were 326 μg/m³ in outside samples and 3600 μg/m³ in interior air. These concentrations are below the lowest concentrations in air that have produced toxic effects in man, 130,000 μg/m³.[7]

The highest concentration of HEX detected in the air of Hardeman County homes was 0.10 μg/m³–0.1% of the occupational standard.[9] In comparison, air levels at the Memphis North Treatment Plant ranged up to 39 and 278 μg/m³ for HEX and HEX-BCH, respectively. The highest concentration of HEX-BCH detected in this study was 3.2 μg/m³ in an outside air sample. In practice, when evaluating air exposure levels for two or more hazardous substances, their effects should be considered to be additive unless other information is available. This practice implies that for complex exposure situations, i.e., those that might be encountered with contamination of a groundwater supply and the air by a multi-component toxic dump leachate, determination of exposure levels of a single chemical may not adequately reflect the actual exposure burden experienced by an individual. In such case, consideration of the exposure levels of several substances is required.

Preliminary toxicity and mutagenicity testing[22] has shown that contamination of some well water samples was sufficient to cause a toxic response and, in one individual, a slight mutagenic response. Well water samples were tested for this biological activity without any concentration step in the laboratory procedure. Municipal drinking water from Cincinnati or New Orleans, for example, requires concentration of the organic components to elicit a biological response. Hexachlorobicycloheptadiene, reported to cause a mutagenic response in the Ames assay,[23] was detected in the contaminated well water, as were the suspected human carcinogens, chloroform and carbon tetrachloride.

The water sample collected from a heavily contaminated well in March, 1979, contained the highest concentration of carbon tetrachloride found in that area up to that time. This result indicates that contamination of the aquifer is increasing even with decreased overall local usage of the aquifer as a water supply. Future contamination of the much deeper artesian aquifer used at a location approximately 50 miles away by Memphis, Tennessee, is a distinct possibility through abandoned well shafts to this aquifer which could be plugged at the present time and, therefore, not detectable by the static head tests performed by the U.S. Geological Survey.[24]

Exposure to many agents, including drugs, alcohol, and a variety of chemicals, as well as disease states including viral hepatitis can cause liver damage. This study, which excluded these potential causes, indicated the presence of a subclinical and transitory liver injury that appears to be associated with the water consumption from wells contaminated by the leaching of material from the pesticide waste dump located in that area. The acute symptomatology and abnormal liver function tests have in all but a few cases returned to normal. However, it is impossible at this time to predict whether there will be any future chronic effects as a result of this exposure. Six of the seven individuals found to have enlarged livers were in the exposed group. A medical follow-up of those studied in this investigation is warranted.

Table 9.–Study Elements of the Two Surveys

Study Performed	Survey November 1978	 January 1979
Air and water monitoring	X	X
Chemical analysis of urine	X	X
Hepatitis serology		X
Biochemical screening	X	X
Health questionnaire	X	X
Physical examination		X

Table 10.–Biochemical Screenings for the Two Surveys

Test Performed	Survey November 1978	 January 1979
Liver function profile*	X	X
Renal function profile†		X
Fasting serum bile acids‡		X
Non-fasting serum bile acids	X	X
Fasting urine bile acids		X
Non-fasting urine bile acids		X

*Serum gamma glutamic transaminase, serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, alkaline phosphatase, and total bilirubin.

†Sodium, potassium, chloride, total carbon dioxide, glucose, blood urea nitrogen, and creatinine.

‡Sulfated conjugates of lithocholate and cholyglycine.

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75TH AIR POLLUTION CONTROL ASSOCIATION ANNUAL MEETING & EXHIBITION, The Rivergate Convention Center, New Orleans, Louisiana, June 20–25, 1982. The technical sessions (June 21–25) and the exhibition (June 22–24) will cover the full spectrum of air pollution control technology, including emission control, instrumentation and systems. Preceding the meeting (June 18–20) one- and two-day continuing education courses will be held. For preliminary program and registration forms, write or phone: Air Pollution Control Association, PO Box 2861, Pittsburgh, PA 15230 [(412) 621-1090].

REPORT ON THE FEASIBILITY OF ORGANIZING A STUDY TO ASSESS THE POTENTIAL HEALTH HAZARDS OF WASTE DISPOSAL SITES WITHIN THE UNITED STATES

Submitted by

UNIVERSITIES ASSOCIATED FOR RESEARCH AND EDUCATION

IN PATHOLOGY, INC.

June 22, 1983

UAREP, with technical assistance from the Life Sciences Research Office (LSRO) of the Federation of American Societies for Experimental Biology (FASEB), has completed the feasibility study requested by CMA. A detailed report, presented in two parts, is submitted herewith. Part I describes the feasibility study, and Part II describes the proposed comprehensive review. We have concluded that within the time constraint of 10 months it is feasible to carry out a review of existing data and prepare a report covering the following general areas:

1. Prepare a list of the chemicals most likely to occur in a majority of waste disposal sites and which are most likely to create health hazards.

2. Identify the acute and, to a limited extent, the long-term toxic effects likely to result from human exposures to such chemicals. This would largely be based on published data in the peer review literature and make use of certain secondary sources of material available from industry as well as state and federal agencies.

3. Estimate the levels and duration of exposure to these chemicals at or above which toxic effects might be expected.

4. Identify those clinical and laboratory investigations which are likely to be most useful in detecting health effects in exposed populations and the epidemiological techniques to be utilized in so doing.

5. Develop scientific criteria for identifying immediate health hazards requiring urgent investigation or attention.

In carrying out such a review UAREP proposes to utilize a blue-ribbon panel of senior scientists and two special task forces assisted by professional and support staff.

We believe the review can be completed in 8 to 10 months, that adequate data is available and that the scientific and professional expertise necessary for organization of the panels, task forces, and staff can be obtained.

Kenneth M. Endicott

Kenneth M. Endicott, M.D.
Executive Officer

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I. REPORT OF UAREP FEASIBILITY STUDY GROUP TO CMA ON THE POSSIBILITY OF ORGANIZING A STUDY TO ASSESS THE POTENTIAL HEALTH HAZARDS OF WASTE DISPOSAL SITES WITHIN THE UNITED STATES

A. INTRODUCTION

Public concern over the potential health hazards of waste disposal throughout the United States has reached a high level. The U.S. Congress and several state legislatures have enacted laws aimed at identifying hazardous sites and taking remedial action. Several thousand sites have been identified and the U.S. Environmental Protection Agency has designated that over 400 require "priority attention".

The problem of hazardous wastes has been the subject of considerable media coverage but the scientific documentation is more limited. Among the scientific publications are "Assessment of Health Effects at Chemical Disposal Sites" (Life Science and Public Policy Program of the Rockefeller University, 1981), and "Management of Hazardous Industrial Wastes: Research and Development Needs" (National Research Council, 1983). The former emphasizes the difficulties of making risk assessments in view of the complications involved in evaluating exposures. The latter reviews and makes certain recommendations regarding waste disposal and also recommends that "both public and environmental risks should be covered in a manner that allows comparison with existing risks that are acceptable to society," and that "an educational program be established to provide the public and industry with soundly based information on the options and risks that are acceptable to society" (page 14). The technical and practical problems of health effects on an individual basis are outlined in the study of the Love Canal (EPA, 1980) and, more recently, Heath (1983) has examined the epidemiology difficulties.

Whereas the National Research Council study does provide a certain consensus regarding the generic and practical issues involved in waste management, the nature and extent of health problems associated with waste disposal sites are much more controversial. Accordingly, though a small number of sites have been the subject of intensive study, the relevance and extent of these findings as related to the problem on a national scale is very uncertain and requires more intensive investigation.

In view of these facts and the delay in carrying out adequate in-depth studies of health effects on an individual waste disposal site basis and the need to obtain more factual information of potential health effects, the Chemical Manufacturers Association (CMA) concluded that it would be of value to all concerned if a group of knowledgeable and prestigious scientists would examine available scientific data and reach an interim conclusion regarding the nature and extent of health hazards which may exist at waste disposal sites as a basis for interim strategy and policy. Further, such a group would outline the scientific criteria for identifying potentially hazardous sites and indicate the methodology of investigation.

In March 1983, CMA entered into an agreement with Universities Associated for Research and Education in Pathology, Inc. (UAREP) to carry out a Feasibility Study to learn how and whether such aims might be achieved through the organization of a scientific review based on the expertise of the academic community.

B. SCOPE OF THE FEASIBILITY STUDY

A small group was set up within UAREP and the Life Sciences Research Office (LSRO) of the Federation of American Societies for Experimental Biology (FASEB) to initiate the feasibility study. The goals of the group were (1) to determine what the aims and objectives of such a scientific study would be, i.e., health effects, exposures, etc.; (2) to determine how such a study would be organized and conducted; (3) to identify sources of information relative to (1) and (2) above; and (4) if possible, to identify what additional studies should be considered, if necessary. The group took into consideration the following areas as pertinent to the objectives of such a scientific review:

1. Information on i) the criteria of health effects to be used in evaluating endpoints of hazards, ii) the methodology of determining how such causal associations are to be determined, and iii) the extent and limitations of the universe of substances likely to be associated with waste disposal sites and their potential exposures for humans.

2. Identification of sources of data, as well as their relevance and potential significance in assessing health effects in waste disposal sites.

3. Information as to the availability of appropriate scientific experts in academia who might be available to participate in carrying out the study.

4. Information relevant to the organization of the interim study.

Initially, the Feasibility Study Group identified a number of parameters that might be addressed during the scientific review, including:

1. Determination of what inferences could be drawn from knowledge of hazardous chemical substances present in waste disposal sites and the probability of human exposures in relation to all types of health effects.

2. Identification of what inferences can be drawn regarding the effects of such substances based on other studies on humans or on animals, i.e., in environmental, occupational, or other settings, and included reports from existing waste disposal site studies. This also included the problem of assessing the limitations of epidemiology and other techniques in evaluating the effects of low level exposures to potential agents.

3. The possibility of carrying out preliminary correlation surveys based on i) known waste disposal sites, ii) studies on states known to have a high degree of environmental pollution, and iii) correlation studies based on general investigations in the U.S. in terms of pollution whether urban or rural, and involving air or water.

4. The possibility of the interim group establishing scientific criteria for identifying the existence of imminent health hazards in relation to chemical waste disposal sites and what would be the criteria or endpoints in terms of clinical and pathological data.

5. The possibility of the study group indicating further areas as requiring investigation in depth for the purpose of making the final reports more meaningful.

These various points were examined and an appropriate planning document prepared and circulated to a number of senior scientists as a background for discussion.

A number of experts representative of epidemiology, environmental waste disposal, toxicology, experimental biology, and biostatistics were approached (*see* Appendix I). In addition, a number of individuals in the field of experimental pathology and two representatives of environmental organizations were contacted. A considerable amount of effort was expended on contacting individuals within the Environmental Protection Agency (EPA) to determine the data and material available in its archives. The CMA advised UAREP/LSRO on contacts within the chemical industry; member companies of CMA prepared and submitted material for consideration by the Feasibility Study Group. Some individuals prepared reports and submitted them to UAREP/LSRO. Others were visited by members of the Feasibility Study Group or visited UAREP in Bethesda.

C. VIEWS OF EXPERTS

As anticipated, there was a wide range of opinions regarding the objectives and organization of any study relative to waste disposal sites. All were agreed on the inherent difficulties involved, but differed as to their importance in relation to an overall review. Some emphasized that if the problem was to be investigated, it should be done well or not at all. The majority agreed that, by far, the best way to investigate the overall problem was to carry out systematic examinations of individual sites. However, some experts also recognized the logistical difficulties associated with doing so in the immediate future, and concluded that some form of interim scientific evaluation of the extent and nature of the health problem was essential to provide some basis for rational public health policies and strategies in the next few years. For example, the Centers for Disease Control (CDC) have investigated only five sites in depth, although there is a considerable amount of material available on an additional twenty. All experts emphasized the fundamental importance of determining human exposures in relation to waste disposal sites, but none could offer exact guidance as to how this might be accomplished except through study of individual sites.

The point was raised by a number of individuals that in the past, few human health effects have been demonstrated to be caused by waste disposal sites and, therefore, it is probable that despite considerable efforts, little additional information may be obtained in future efforts of this type. If true, such a finding is clearly of importance in terms of future public health strategy and control of waste disposal sites since it suggests that more emphasis should be placed on methods of diminishing waste disposal, as done by the Japanese, than on the investigation and clean-up of existing sites. Many consultants indicated their doubt that the public will readily accept such a conclusion in view of preconceptions as to health hazards which have developed over the last several years and because of extensive media coverage of "crises". These aspects undergird the importance of ensuring the credibility of any study which carries out an overall investigation of this problem.

D. IDENTIFICATION OF POTENTIAL DATA SOURCES

The Feasibility Study Group discussed sources of data with the consultants and carried out preliminary surveys of the general literature. UAREP/LSRO staff members reviewed data retrieved from computerized literature services (Medline, Cancerline, Toxline, and Enviroline). They also reviewed such publications as the monograph series of the International Agency for Research on Cancer, and the Environmental Health Criteria documents of the World Health Organization on toxic hazards. In addition, a number of key articles were reviewed relative to the availability of specific material regarding waste disposal sites (*see* Appendix II). In brief, it can be stated that the amount of material available in the literature with respect to specific studies on hazardous waste disposal sites is very small. On the other hand, there are considerable data available regarding individual chemicals and their health effects based on a variety of studies including animal toxicology and epidemiology studies of occupational and other populations which might be of use for drawing inferences as to possible health effects.

There is a large amount of material available in the CDC on the potential health effects of waste disposal sites. Whereas some of it has been published in the general literature, the greater part is available as internal technical reports. However, the CDC staff indicated that this material would be made available to UAREP, and they would be pleased to collaborate directly with the UAREP study group in evaluating and identifying data pertinent to the overall problem. The experts at CDC were of the opinion that only waste disposal sites at Love Canal, New York; Triana, Alabama; Woburn, Massachusetts; and Times Beach, Missouri had been investigated adequately.

Key EPA personnel were contacted but little additional information seemed to be readily available regarding the problem, despite the fact that other experts indicated to the Feasibility Group that EPA has the best data on exposures. Several EPA contacts indicated that the desired information might be obtained from the CDC in Atlanta, but the CDC stated they had exposure data only for sites in which they had carried out some initial health investigations. CDC referred UAREP back to EPA. In conclusion, it proved impossible to identify who in EPA had such material; it was decided to await further approaches until after the organization of a review group.

Industry made material available to UAREP regarding exposures to suspected problem chemicals in the work place. A number of reprints and drafts of studies underway were made available to the Feasibility Study Group which were clearly pertinent and valuable in drawing inferences (*see* Appendix II). Key reference material is identified in Appendix III. In some cases these documents can be amplified by the literature in peer review journals. These data will be valuable and pertinent relative to information on the potential effects at specific exposure levels, but such data again raise the perennial question of how to deal with the effects of exposure at very low levels which would be fundamental to much of the assessment of the study group.

Preliminary contacts were made in the United Kingdom, and it would appear that within the European Common Market a considerable amount of data have been collected relative to hazardous waste disposal. Apparently, studies have also been carried out on potential health effects in some areas, but relatively little has been published. It is intended to investigate in further depth what material is available later this year, but preliminary investigations indicate that a number of individuals in Europe will be eager to collaborate in this context.

In conclusion, there appears to be less published data regarding the specific effects of waste disposal sites than had been originally anticipated. Although it is clear that much pertinent information is present in archives of various sorts, it will require a concerted effort to obtain and analyze this material. On the other hand, a considerable amount of published literature is available which contains important material on which inferences may be drawn by appropriate experts regarding potential health effects. The weakest area appears to be in relation to potential exposure levels. In this context, preliminary correlation studies may be of value. The Feasibility Study Group further explored what already had been done in this context.

1. Studies of Individual Waste Sites

While all agreed that a comprehensive study of individual waste disposal sites would be the most satisfactory rational method in identifying or excluding potential health effects, to do so would require extensive resources in manpower and expertise and a considerable period of time to implement. Clearly, it is exceedingly difficult to evaluate the potential danger of a waste disposal site unless accurate information, not only on the chemicals present but also on the probable degree of human exposure, is available. This suggests that for the study it will be necessary to draw up some general guidelines as to possible approaches. It will be highly desirable to explore what material is available within CDC in the form of internal reports in this context. CDC had considered but dropped the possibility of using certain chemicals as indicators of pollution. These CDC reports cover the PCB problem in the North Hollywood dump in Tennessee; the PBB problem in Michigan; the asbestos problem in Globe, Arizona; arsenic at Woburn, Massachusetts; and in a mixed deposit at Tar Creek, Kansas and Oklahoma. The reason many reports have not been published is that the data are essentially negative, far from complete and, therefore, "uninteresting". Thus, if relatively little evidence of exposure was found in a population group, no further action relative to health effects was undertaken. It would appear essential to explore further the importance of these negative findings as it may be fundamental to the objectives of the interim study to determine what are the conditions in which exposures are likely to occur and the period that may elapse before a site begins to break down and become a hazard. This will require further contact with appropriate experts in that area.

2. State Correlation Studies

Drs. R. S. Levine and D. Chitwood of the Department of Epidemiology and Public Health at the University of Miami prepared an "Analysis of Existing Data Pertaining to Potential Health Problems from Hazardous Wastes in Florida" in April 1983, acting under Prof. J. Davies. This was a rapid survey of the state of Florida covering areas of waste disposal sites and associated health indices in relation to such deposits. In general, no health effects were found in most areas of the state that could be definitively correlated with the EPA designated sites, although in some sites, there was a trend toward lowe. indices of health. However, in other areas of the state where pollution was known to be considerable, no health effects were evident. In fact, these often were the most healthful areas relative to the rest of the state and the U.S. as a whole. No definitive conclusions could be drawn on the health effects of such sites. On the other hand, Davies believes that such an approach can demonstrate that, while a health hazard due to a waste disposal site cannot be completely excluded, such studies can provide most useful data as to the probable existence and extent of the health problem. Interestingly enough, this Florida study indicated that an immense amount of material relative to existing sites had already been collected and was present in the State Department of Health. This suggests that in planning and investigating exposures, it may be desirable to use these sources in addition to EPA or CDC. *The Miami workers were convinced, therefore, that this approach did provide data of relevance and should be tried out in several other states*. They estimated that each such study could be carried out at a cost of approximately $100,000 within a three month frame utilizing the resources of local universities and departments of health. However, these costs may be unrealistically low because for the Florida study personnel, facilities, computers, and related equipment and materials were already in place.

The Feasibility Study Group felt that this approach should be explored further during the review as a potential source of data in carrying out future follow-up investigations to place some limits on the probable extent of the hazard.

E. FEASIBILITY STUDY RESULTS

1. Availability of Data

There is extensive literature available regarding the toxicology of many chemicals, but specific information relating to the amount of each chemical in individual waste disposal sites, the degree of population exposures, and the potential hazards involved are very limited. Thus, unless specific assumptions are made, the problem theoretically can be so huge as to defy analysis. Accordingly, it is estimated that the study will need to confine its review to a limited universe of chemicals, to drawing specific inferences that might be pertinent to the overall problem, and to defining and justifying such assumptions on a scientific basis.

2. Availability of Expert Consultants and Staff

Conversations with a number of experts disclosed a broad interest in toxic waste disposal problems and a willingness to participate in a scientific approach to solving these problems. Recruitment should not present a major problem although some desirable candidates will be unavailable due to other commitments or serious conflicts of interest.

A preliminary list of potential consultants is presented in Appendix IV. This list will be expanded as necessary in the course of the study, especially after the first meeting of the Executive Scientific Panel.

Based on previous experience, it was decided that resolution of problems such as conflict of interest and willingness to testify should not be attempted during the feasibility study but, rather, should await authorization to proceed with the comprehensive review.

F. CONCLUSIONS

1. The Feasibility Study Group concluded that it is both desirable and feasible to carry out a comprehensive review to evaluate the substantial body of available data for the purpose of permitting an interim conclusion as to the nature of the health problems likely to be associated with waste disposal sites. A detailed presentation of the Feasibility Study Group recommendations on the organization and implementation of the comprehensive review study follows (Part II). Such a study would have the following objectives:

i) Preparation of a universe of chemicals most likely to occur in waste disposal sites and which are most likely to create health hazards.

ii) The levels and duration of exposure to these chemicals above which toxic effects might be expected would probably have to depend largely on inferences. Thus, the perennial question of the presence or absence of low exposure level effects would require emphasis and standard methodological approaches. However, a considerable amount of material is available regarding water and air pollution within the U.S. which can be used in determining such possibilities. It will not be possible, however, to utilize all the material available within EPA and other institutions.

iii) Identification of the acute and, to a more limited extent, the long-term toxic effects likely to result from human exposures to such chemicals, and whether they are real or imagined. This review would be based principally on published data in the peer review literature. It would make considerable use of secondary sources of material in view of the great difficulty of evaluating the vast amount of primary and raw material in EPA or state archives, unless the latter had been adequately analyzed.

iv) Identification of those clinical and laboratory investigations which are likely to be most useful in detecting health effects in exposed populations and the epidemiological techniques to be utilized in so doing.

v) Development of scientific criteria for identifying an immediate health hazard requiring urgent investigation or attention.

A comprehensive review is considered desirable and feasible because it would be comparatively inexpensive in terms of money and manpower. While it will add relatively nothing new to the universal literature, it will provide a unique source of evaluated data for scientists and the public regarding the potential health hazards and scientific issues associated with investigating waste disposal sites. It will provide a mechanism whereby the scientific community can be directly involved in investigating such issues and correcting the misconceptions that are widely held by the public. It will identify problems that are likely to occur frequently and thus involve academia in a search for scientific rational solutions. This may be pertinent to the further development of more intensive studies on an individual waste disposal site basis which may be necessary over the next decade.

Such a review might be more credible if it had multiple sponsors and if it were perceived as an interim measure to be followed up by additional studies.

2. The Feasibility Study Group concluded that as the proposed review is being completed, it would be desirable to follow up with studies aimed at compiling and analyzing raw data in the files of EPA, CDC, and various states agencies, carrying out in other states procedures modeled after the Florida study, analyzing the NHANES II data bank and results of the Health Interview Surveys conducted by NCHS to develop baseline health values, and testing the validity of the criteria developed in the review in a limited number of specific toxic waste disposal sites.

Such follow-up studies would require participation of various agencies and organizations and should also be funded from a number of sources probably including government, industry, and private philanthropy. The follow-up studies would also require wider and more extensive involvement of the academic community in developing correlation studies and utilizing data from all sources, as well as more active involvement and cooperation between the academic community and CDC, as well as other government laboratories. In addition, follow-up studies would enhance the probability of producing new data and providing better baselines for developing new scientific and technological criteria for hazardous waste disposal.

The only major disadvantage associated with follow-up studies is the potential cost and the necessity for developing considerable manpower and resources in academia. On the other hand, these costs are likely to be relatively modest in the context of the vast expenses incurred in cleaning up waste disposal sites if this is not done on a rational basis.

II. FEASIBILITY STUDY GROUP RECOMMENDATIONS ON THE ORGANIZATION AND IMPLEMENTATION OF THE COMPREHENSIVE REVIEW STUDY

A. OBJECTIVES:

1. To prepare a list of the universe of chemicals associated with waste disposal sites which could prove hazardous to man as the result of excessive exposure. Identify, if possible, the levels of human exposures likely to occur in association with waste disposal sites.

2. To define the probable health effects likely to occur in association with waste disposal sites and classify these according to their nature, real or imagined, acute, subacute, chronic, or carcinogenic. This is to be based on inferences from the available studies within the literature and inferences drawn from occupational, animal toxicology, and other sources.

3. To identify the clinical and laboratory and results that should be utilized in investigating such problems, and the epidemiological criteria necessary to establish causal relationships. Special emphasis should be given to the evaluation of negative data in relation to low level exposures.

4. To identify the scientific criteria for describing a specific or individual waste disposal site as representing an imminent health hazard.

5. To decide to what extent these conclusions (#1-4) provide a basis for identifying which follow-up studies may be carried out.

B. IDENTIFICATION OF PROBLEM CHEMICALS AND THEIR EVALUATION

Crucial to the development of the study will be the establishment of guideline criteria for identifying the universe of substances in waste sites and for establishing causal associations between exposures and potential human health effects.

1. Universe of Substances and Waste Sites

For logistical reasons, the list of hazardous chemicals must be defined and limited but, at the same time, hazards of perceived potential major significance must not be excluded. Identification of such chemicals and their exposure levels will be fundamental to the success of the study because, above all, it will be necessary to review the effects of probable human exposures likely to occur, or to have occurred, in relation to hazardous waste disposal sites as a basis for any epidemiological approach.

In attempting to analyze the universe of hazardous substances, the following lists of chemicals have been examined during the Feasibility Study:

i) Chemicals identified in the Superfund Priority Sites list of 418 hazardous waste disposal sites. The list of chemicals was prepared by Dr. Blaine McKusick of du Pont.

ii) A list of chemicals provided by CMA and designated by six major chemical-producing companies as those considered to be the 20 most likely to cause risks or public perception of risk. Each company had prepared its own list of 20 problem chemicals. A few chemicals received five to six votes, but most received two or three, indicating that the interests of the six companies are not necessarily similar. However, the final list of 20 represents a consensus of the six chemical producers.

iii) A list of chemicals prepared for the State of California and published in Chemical and Engineering News 1982.

iv) EPA list of relative carcinogenic potencies among 53 chemicals evaluated by the Carcinogen Assessment Group (dated April 5, 1983.)

v) Other sources of material relating to general or local environmental pollution, such as the Love Canal list and lists provided by several consultants.

It is interesting to note that there is a certain degree of overlap among the chemicals on each of the lists mentioned above.

2. Evaluation of Exposures

Estimation of exposures are recognized to vary widely and, until now, no satisfactory methods of calculating these have apparently been developed. Thus, each waste disposal site must be investigated according to a highly systematic plan. The possibility, however, that an appropriate group will be able to provide guidelines for the development of appropriate models as to those chemicals most likely to pose a danger according to the construction, location and nature of a deposit will receive careful attention. Some attempt must be made to distinguish between waste disposal sites that are well constructed and likely to exist for a prolonged period, and those where there is evidence already of immediate breakdown and release. It is recognized that while air and direct exposures may occur, they are unlikely to present an important hazard and that greatest concern relates to potential water contamination and, possibly indirectly, through diet resulting from contamination of local vegetation.

Most important, the overall significance of small waste disposal sites must be examined. For example, these were believed to be the greatest problem in Florida. It will be necessary, therefore, to explore all these aspects of the problem and to ensure that the Executive Panel and two Task Forces contain experts who have had experience in movement of substances from sites. Thus, evidence in this area will depend on i) material from sites which have already been examined, ii) literature reports in this and other countries, iii) the availability of further material of the type that was obtained in Florida from local resources, and iv) other sources. It may be possible that, after reorganization, EPA may be able to provide further information to the study group than they have been able to provide to the Feasibility Group. An additional important source of information will be studies on wild and domestic animals in the region of the sites.

Considering the importance of this aspect of the total problem, the material available from known sites should be examined and analyzed first. Then, the possibility of developing a background database of the overall exposures in the general population of the U.S. relative to suspected compounds should be explored. What is known regarding excess exposures in different situations will be established and specific areas in which appropriate exposure studies should be conducted will be defined. Conversely, there may be sufficient material already in the literature to permit evaluation of the general effects (or lack thereof) at higher levels of exposure, but this, again, almost certainly will require review by a group of experts.

3. Evaluation of Health Effects

In view of the many potential chemicals involved, no attempt will be made to generate primary data or evaluate raw data unless it is absolutely essential. Thus, data on health effects will be predominantly generated from previous reviews or assessments available within the general literature and other pertinent sources which are available to the public. Some of the various aspects that can be studied include:

i) Health Effects

Health effects will be inferred from what is known regarding potential levels of exposure and inferences drawn from high, intermediate, and low levels of exposure in other situations. Any attempt to relate low exposure level health effects to the multitude of chemicals which may occur, not only in relation to waste disposal, without considering the general background of the United States will be difficult and require careful evaluation but must be done. Thus, some attempt may be necessary to develop a profile on what is already known regarding exposures of the human population within the U.S., in terms of tissue deposits, etc., and their significance in disease patterns. Much of this material may, in fact, be available within the literature and could be the subject of further in-depth evaluation.

ii) Acute Effects

It will be important to define end points that can be examined with relative ease because they are not dependent on prolonged latent periods. Such effects should be closely related temporally to exposures if they are significant. Therefore, acute health effects may be susceptible to review whether as indicated by clinical, or clinico/pathological, or biochemical examination. Investigations of acute health-effect criteria should be standardized to a certain extent; criteria should be established to distinguish between organic and psychosomatic lesions. In this context, inferences drawn from occupational, accidental, and similar exposures at higher levels will provide pertinent data.

A list of end points will have to be examined to determine which are most likely to provide information of importance in evaluating health effects related to waste site exposures. Such end points would include:

a) blood studies;
b) urinary studies;
c) analysis of breast milk and other fluids and tissues;
d) evidence of respiratory, kidney, liver and other dysfunctions;
e) evidence of damage to infants, such as congenital malformations, cytogenic abnormalities, etc.

iii) Chronic Effects

Chronic effects, notably cancer, will require careful evaluation in light of the associated long latent period. In view of the small number of individuals liable to be exposed in relation to a particular waste disposal site and considering the known carcinogenic effects to be anticipated from suspected chemicals, it is quite possible that no conclusions can be reached regarding negative or positive findings in this context.

iv) Teratogenic and Mutagenic Effects

Following exposure to suspected chemicals, teratogenic and mutagenic effects may occur rapidly and are identifiable through congenital malformations, but many limitations apply to interpretation of the significance of several tests for mutagenesis. The assistance of CDC can be very valuable in this respect; toxicologists and epidemiologists with special expertise will be required to evaluate the findings.

v) Baseline Data

To compare the health status of individuals exposed to waste disposal sites deposits with that of the general population of the U.S., baseline data are required. Data from the ongoing Health Interview Survey and the second National Health and Nutrition Examination Survey (NHANES II) 1976-80 carried out by the National Center for Health Statistics would be used to identify a basic health profile of the U.S. population. With this information in hand, it may be possible to determine, on a broad basis, whether any indices can be attributed to proximity to waste disposal sites for an exposed population.

vi) Experience in Japan and Europe

Direct contact will be made with appropriate scientists and experts in Europe and Japan, who will participate as consultants to the task forces.

vii) Studies on Flora and Other Markers of Toxicity

A review of data from wild and domestic animals will be made and evaluated in terms of their implications for human disease.

In conclusion, the Feasibility Group agreed that a vast amount of material was available on a national and international scale from which significant and important inferences could be drawn as to the probable nature and extent of ill-health effects that could be anticipated as a result of previous waste disposal practices. In the past this material has been scattered and considered usually in terms of individual chemicals. However, a number of studies have been carried out on air pollution. In contrast, studies on chemically polluted water -- probably the most feared result of waste disposal -- are very poor and in terms of human disease are essentially negative, although the importance of water as a major source of human exposure has been shown for many of the suspected chemicals.

The possibilities of such a review are probably greater than originally believed and further analysis will be of great value in terms of the involvement and understanding of the academic research community.

4. Epidemiological Studies

In view of the small numbers of individuals likely to be involved in most waste disposal site exposures, epidemiological studies will be difficult to perform and will provide little information in most cases. Thus, the comprehensive review will of necessity have to contain an evaluation of the problems and limitations inherent in assessments of health effects related to individual sites. In addition, other methodologies for collecting material on a wider scale will have to be explored.

This particular aspect of the problem will be the subject of a specific task force. In this context, it will be desirable to further explore the experience of the Miami group in Florida as a base for calculating the overall extent of the problem.

5. Identification of Scientific Criteria for Classifying a Waste Disposal Site as an Imminent Health Hazard

This will arise out of the general discussion in the early portions of the survey and be addressed later by the Executive Panel which may, however, wish to redirect the question to one of the Task Forces.

C. ORGANIZATION OF THE STUDY APPROACH

The comprehensive review study will require an Executive Scientific Panel (ESP), two Task Forces (TF1 and TF2) and a full time scientific and support staff secretariat. The ESP will consist of 12 to 15 eminent academic scientists of international reputation. The ESP will be multidisciplinary in membership, including epidemiology, pathology, toxicology, biostatistics, oncology, occupational carcinogenicity, industrial hygiene, biochemistry, public health, toxic waste engineering, and other closely allied fields.

TF1 will address the universe of chemicals and aspects of exposure. TF2 will concentrate on human health effects. Both Task Forces will focus on additional issues and individual problems identified by the ESP and call upon appropriate experts from all sources to provide information and evaluation of the technical and scientific problems inherent in the investigation. A preliminary list of scientists identified as potential candidates for the ESP and Task Forces is attached (see Appendix IV).

In view of the nature of the subject and its unusual political and scientific ramifications, it is difficult to give an immediate outline of the procedures to be adopted by the ESP. These will depend, to a certain extent, on organization of a compatible panel of experts and their preliminary review of the problems and resources available. Accordingly, this necessitates a flexible approach to the initial scientific analysis and structure; the procedures outlined below should not be taken as definitive. It is a general principle that every effort will be made to take cognizance of and acquire any scientific information from any source pertinent to the overall problem, and these data will be freely available at the conclusion. Further, members of the committee will avoid obvious conflicts of interest. Lastly, adequate disciplinary representation and expertise should be included among the members to ensure wide exploration of all scientific viewpoints and to ensure credence in both the academic and public domain.

1. Management and Organizational Procedures (ESP)

While procedures must remain flexible, there are several aspects common to all evaluation studies. These include the following:

i) Preliminary conferences are held with key individuals to explore extant knowledge concerning hazardous waste disposal sites and to further identify experts with access to additional information.

ii) The purpose of these preliminary conferences is not only to identify additional individuals who may have pertinent knowledge or are engaged in research in related relevant topics, but also to define those scientific activities which may be most closely related to the particular type of evaluation. These discussions are used to generate a list of individuals as well as a list of key words to be used in computerized literature searching. Subsequent identification of key review articles and important information sources provide further expansion of the lists of potential consultants and pertinent key words.

iii) A definitive listing of pertinent parameters to be examined by the Executive Committee and Task Forces is prepared as appropriate based on an expanded review of current literature by the staff, current information on other aspects related to scientific evaluation, and consultation with key individuals concerning the scientists identified initially. In this case, care is taken to select all potentially pertinent information which will be balanced by the inclusion of work of investigators involved in related topic areas, or scientists holding divergent views concerning the subject.

iv) Typically, at this point a working paper will be prepared for the ESP by the professional scientific secretariat. This working paper describes the background of the issue and the extent of information available concerning the study topic. These papers, along with selected references, are sent to each participant prior to the ESP meeting at which the subject will be discussed.

v) Experience suggests that discussions with individual investigators or knowledgeable persons, as well as deliberations of the ESP at one to three-day meetings, coupled with a comprehensive literature review and analysis by the Task Forces, constitutes a reasonable basis for drafting an evaluation study. This draft report is prepared by the secretariat based on information obtained from the Task Forces, scientific literature, and from the discussions of the ESP. The draft report reflects documentation from the scientific literature and other valid information sources, as well as pertinent analysis and opinions of the Task Force members and/or ESP members. Where a controversial area exists, all aspects are discussed in considerable detail, and scientific references are given wherever possible. In instances where a consensus opinion of the ESP cannot be derived, an effort is made to include all divergent views.

vi) Drafts on individual topics are submitted to all members of the ESP as appropriate and, on occasion, to additional experts for their critical review and comment. Revision of the draft report is made by the staff based upon comments and emendations submitted by all participating consultants. The final draft may be resubmitted to the ESP for subsequent review and concurrence.

vii) The report of the ESP will be submitted to the UAREP Board for review and approval. This secondary review process ensures that the report will meet acceptable standards for scientific objectivity and fulfillment of study objectives.

viii) The Final Report will be published *in toto* and a synopsis will be published in the scientific literature.

2. Management and Organizational Procedures (TF)

The two Task Forces will be organized and will operate in an analogous manner, with certain exceptions. First, the ESP will play a major role in identifying qualified experts for membership on the two Task Forces. Second, membership on the Task Forces will be based primarily on scientific and disciplinary expertise with less emphasis on international reputation and possible conflict of interest.

Task Force reports, or series of reports, will be considered internal documents and will be exchanged between Task Forces, but delivered to the ESP. Task Forces are also considered ad hoc and may change in membership as determined by the ESP and secretariat. At least two ESP members will be on each Task Force.

3. Procedures to Assure the Adequate Identification as well as the Quality, Relevance, and Completeness of Data

Once specific problem areas have been identified relative to the universe of substances, exposure routes, health effects, etc., the ESP or a steering group of the ESP will explore i) information concerning a particular chemical, ii) toxic effects, and iii) identification of appropriate experts for consultation by the ESP for appointment to TF1 or TF2. Where appropriate, staff will visit investigators at their laboratories. The process of identifying and evaluating pertinent information will continue throughout the evaluation process, but it will probably not be possible to utilize sources of unanalyzed raw data.

Quality, relevance, and completeness of scientific data are a critical aspect of scientific evaluation. Experience dictates that the quality of data is related to the completeness of data and where these data may be found. In the final analysis, a reasoned judgment of the quality, relevance, and completeness of available data is a matter of a determination by the members of the ESP. The validity of the determination rests with the experience and reputation of the individuals who are selected to serve on TF1 and TF2, as well as with those on the ESP.

4. Elimination of Potential Bias and Conflicts of Interest Among Members of the ESP and TF1 and TF2

Evaluation studies of this type must use guidelines that include ad hoc self-terminating approaches, emphasis on the minimization of bias, and avoidance of quasi-permanent committees and appearance of vested interest. The following steps and techniques have proven successful in maximizing the objectivity of the input to evaluation studies:

i) Key individuals assist in the identification of potential ESP members and TF participants. These include members of the UAREP Board and other representatives of scientific societies who are able to identify knowledgeable scientists with expertise in particular study topics. The multiplicity of input provides ample opportunity to identify a wide diversity of potential participants. Every effort is directed towards balancing potential participants by the inclusion of consultants who may hold divergent views on a particular topic.

ii) In the course of developing an intensive literature database, it is possible to identify the specific contributions of potential participants to the study. Quality and objectivity of scholarly publications as well as reputation are evaluated by the project's staff.

iii) The final lists of ESP and TF members are reviewed by the UAREP Board. This review procedure is included to eliminate bias and potentially avoid vested interests following which an effort is made to informally contact each individual a second time to invite him or her to participate in the study.

iv) ESP and TF members are asked to complete a conflict of interest form. This form provides information on the extent of financial obligations and involvement in the public and private sectors which may bear upon the particular topic under evaluation. Procedures have been developed for maintaining the confidentiality of these statements; and an internal review is conducted by members of the UAREP Board. Specifically, the Executive Officer of UAREP, the President of UAREP, and the Business Manager of UAREP reviews each statement of conflict of interest for evidence of potential conflict. Where no conflict exists, forms are signed, sealed, and held until completion of the project. Where potential conflict is noted, the opinion of legal counsel is sought.

If and when conflict of interest is apparent, the scientist is informed, while a decision is made as to whether or not he or she should be given further consideration. If in the affirmative, the individual must agree prior to the evaluation study that, if at any point this conflict of interest is raised by the Executive Officer of UAREP or the project director, he or she would state for the meeting record non-participation in the deliberations or decision-making process of the Task Force or ESP.

5. Procedures to Resolve Conflicting Opinions

During Task Force meetings or during the deliberations of the ESP, the respective chairmen will ensure informal, harmonious discussions in which all participants are allowed to present data and comment on information presented. The chairmen must be selected for their experience in this context. Draft reports, revisions, and preparation of the final reports by the secretariat are written to reflect opinions of participating scientists and other knowledgeable individuals, documentation from the scientific literature, and pertinent analysis. Because the staff rapporteurs are recognized as professional scientists by all, the credibility of the document is further enhanced. Every effort is made to present and document divergent views, but to give a consensus of scientific opinion where appropriate. If a consensus is not appropriate, then divergent views are documented as completely and objectively as possible.

In cases where minority opinions are necessary, they are written by the secretariat based upon discussions with those dissenting members. Members with dissenting opinions are asked to indicate their identity and their concurrence with the material that has been independently prepared by the staff. This aspect is further strengthened by the subsequent secondary review and approval of the report by the UAREP Board.

6. Proposed Study Plan

The overall study comprises specific steps as follows:

Month 1: Identify Executive Scientific Panel and Chairperson

The ESP will be comprised primarily of members of the academic community with expertise in epidemiology, toxicology, biostatistics, experimental pathology, oncology, occupational carcinogenesis, industrial hygiene, biochemistry, public health, and toxic waste engineering. Emphasis will be placed upon obtaining the cooperation of individuals from the academic community who, insofar as possible, have no conflicts of interest or biases. These will be individuals recognized for their expertise in the particular relevant disciplines. There will be approximately 12-15 individuals on the Executive Panel. Budgetary considerations are based on a 15-member Executive Panel and five two-day meetings (or some combination of 10 days of meeting time).

One of the first individuals to be identified would be the Chairperson of the ESP. This individual should be available 100% of the time during the study period and should be recognized for his or her general expertise in review and evaluation of public health and scientific issues, rather than only for expertise in one particular discipline. Because most knowledgeable scientists in industry and government have *ipso facto* a conflict of interest, an effort will be made to recruit academic members of the scientific and medical community as ESP members.

Nominations will be accepted from all sources, including public announcements and specific requests to various communities concerned with toxic waste sites, such as industry, government, environmentalists, academic scientists, scientific societies, professional organizations, and other sources (e.g., the National Academy of Sciences, Carnegie Mellon Foundation, Ford Foundation, etc.). *The primary criteria for membership on the Panel will be scientific expertise and freedom from conflict of interest or bias.*

A list of potential invitees as members of the ESP will be prepared by the staff and forwarded to the UAREP Board for concurrence and selection. Individuals selected will be asked to serve on the Panel and to complete the conflict of interest information.

Months 2-3: Initial ESP Meeting

A two-day meeting for organizational purposes will be conducted. Prior to this Panel meeting, all members will have been invited and informed of the conditions of the appointment and the scope of the study. In addition, they will receive a working paper describing the project and an agenda for the meeting. At the meeting, the consultants will be asked to:

i) review and concur with the study plan or make modifications as they deem desirable,

ii) identify the need for Task Force activities and to provide a scope of work for those Task Forces, and

iii) identify individuals to serve on the several Task Forces.

It should be noted that at least two members of the ESP will serve on each Task Force as chairperson and vice chairperson (or recorder or rapporteur). Members of the Task Forces will be selected on the basis of scientific expertise and may include representatives of industry, government, academia, and other communities as required by the scope of Task Force activities.

Months 3-4: Appointment of the Task Forces

At the present time, we believe that only two Task Forces will be necessary.

Universe of Chemicals and Human Exposures Task Force (Task Force #1): The first activity of Task Force #1 (TF1) will be to evaluate the several lists of chemicals that describe the universe of substances found in hazardous waste disposal sites, and to finalize a finite list of substances likely to prove problematic in terms of hazardous waste disposal sites. It should be noted that at this stage, TF1 may require additional outside expertise from both government and industry as consultants.

The second activity of TF1 will be to examine information available on the substances in the finalized universe of chemicals, and to identify levels of probable exposure, methods or route of exposure, and duration of exposures that may prove to have some impact on human or animal health, and on the environment. TF1 will have seven to nine members and may utilize an additional five consultants. TF1 will meet four times for two days each.

Health Effects Task Force (Task Force #2): TF2 will consist of nine to eleven persons and will provide guidance in the evaluation of probable health effects that are to be anticipated in relation to hazardous waste disposal exposures of those chemicals within the universe of substances that have been determined to be potential exposure problems.

TF2 will consider acute and chronic health effects that are likely end points of exposure, information available from previous epidemiological studies and from occupational medicine, scientific evaluation of toxicity in laboratory animal studies, and data from other waste disposal sites. TF2 may wish to organize itself into subgroups. Additionally, it is expected that TF2 will require a greater input from staff in terms of literature and data analysis. TF2 may also require more time and meetings than TF1. TF2 will meet five times for two days each.

Months 3-6: Meetings of Task Forces

It is anticipated that TF1 must meet first and that TF2's progress will be dependent upon the output from TF1. The budget reflects a total of six meetings for the two Task Forces; however, it is anticipated that TF1 would meet twice and TF #2 would meet four times. Both TF1 and TF2 would draft reports, with the reports of TF1 being submitted to TF2.

The final output from TF1 would be a finalised list of problem chemicals, and information concerning environmental, animal, and human exposures that may prove to have demonstrable health effects. Emphasis would be upon human health effects. Clearly, this background information would be essential for TF2 in categorizing the likely end points or criteria for human health hazards and in identifying sources of data where the Health Effects Task Force would look for information relative to the toxic effects and potential health effects of the substances.

Months 4-7: ESP Meeting

Following the completion of the reviews of TF1 and TF2 and the completion of their draft reports, the ESP would utilize this information to create a draft of the review study report.

The conclusions of the review study draft report would point out what should be looked for in any waste disposal situation based upon extant scientific knowledge concerning the potential hazard to man, rather than upon political expedience, public complaints, or other reasons. A strong case would be developed for a reasoned judgment on scientific evidence of potential risk. The Tentative Final Report of the ESP would be published and made publicly available for comment.

Month 8: Open Public Forum

After publication of the Tentative Final Report of the ESP, an opportunity would be provided for all members of the public and the several communities of interested parties to come and address, either in writing or orally, the ESP at an Open Public Forum. The topic for discussion would be a review of and comments on the Draft Report in terms of garnering opinion as to the approach being presented and identification of any additional needs or deficiencies. This open public forum would be recorded and a transcript of the entire forum would be made available at cost to all participants. The ESP would be seated in front of the room and would listen to and accept all comments. The purpose of the Open Public Forum is information gathering and there would be no emphasis on entering into a dialogue or presenting the information in an adversarial manner. The organization and conduct of the open forum would be disseminated as widely as possible. In making arrangements for this, those individuals who wish to testify orally would be asked to provide written statements. Those not wishing to testify orally would be given deadlines for submission of written material. By negotiation, all oral testimony would be confined to a finite period of time.

Months 9-10: Review of Information Received

The ESP would meet a third time to discuss all information presented at the Open Public Forum and to revise as necessary the Tentative Final Draft of the Report. Based upon the decisions reached by the ESP in a review of the included comments, an attempt would be made to reach a consensus by the panel and a Final Report would be prepared. This Final Report would be provided to the sponser of the study and would be disseminated widely and made available at cost to the public.

D. ESTIMATION OF COSTS

In analyzing the necessity of input from Task Forces and ESP, and to provide adequate time for review of the information collected by Task Forces, for preparation of Task Force reports, and for subsequent analysis of this information by the ESP and preparation of the Final ESP Report, as well as a desire to have informed public input, it seems logistically necessary to provide for a minimum of a 10-month period for the Comprehensive Review Study. The budget has been developed on the basis of 10 months. The budget also contains additional information concerning the number of meeting days for members of the Task Forces and panel.

COMPREHENSIVE REVIEW STUDY

10-Month Budget

	Annual Salary Basis	10-Month Budget	
Personnel[a]			
Project Director[b]	$ 62,400		
Staff Scientists[c] (2)	101,400		
Associate Scientists[d] (2)	58,800		
Administrative Assistant	24,953		
Technical Data Analyst	19,394		
Technical Literature Specialist	19,394		
Senior Secretary	17,958		
Clerk	12,226		
Subtotal	$316,525	$263,771	
Fringe Benefits & Payroll Taxes @ 28%		73,856	
Subtotal Personnel			$337,627
Consultants[a]			
a) Executive Scientific Panel 15 members @ $500/day 5 meetings - 2 days each		75,000	
b) Task Force #1 9 members @ $450/day 4 meetings - 2 days each		32,400	
c) Task Force #2 11 members @ $450/day 5 meetings - 2 days each		49,500	
Subtotal			$156,900

Travel and Per diem[f]		
Travel @ $600; per diem @ $100		
a) Executive Scientific Panel		
5x15x600	45,000	
5x2x15x100	15,000	
b) Task Force #1		
4x9x600	21,600	
4x2x9x100	7,200	
c) Task Force #2		
5x11x600	33,000	
5x11x2x100	11,000	
Subtotal		$132,800
Other Direct Costs[g]		
Telephone @ $600/month	5,400	
Postage and mailing (includes overnight mail) @ $975/month	8,775	
Expendible supplies	3,600	
Computerized literature retrieval @ $240/month	2,160	
Word processing @ $320/month	2,880	
Report preparation (2TF + 1 tentative + 1 final) 4 reports @ 150 pp. each @ $27/p., 550 cc.	16,200	
Duplication & photocopying, est. 216,000 @.07/p.	15,120	
Open public forum:[h]		
Hall rental for 2 days	1,100	
Transcript preparation	2,350	
Conference center expenses[i]		
1 meeting - 4 days	2,400	
Miscellaneous	975	
Subtotal		$ 60,960
TOTAL DIRECT COSTS		$688,287
INDIRECT COSTS[j]		
(39.6% of salaries, wages and consultant fees = $494,527)		$195,833
TOTAL COSTS		$884,120
FIXED FEE @ 10%		88,412
TOTAL CONTRACT		$972,532

BUDGET NOTES

a) All personnel are 100% FTE for 10-month period. It is essential that scientific staff be full time; some salary lapse with support staff may occur during months #1 and #10, but wage figures are not adjusted.

b) The Project Director would be a senior scientist (M.D. and/or Ph.D.) whose international reputation and scientific credentials are impeccable. His or her expertise would be in toxicology, pathology, and epidemiology.

c) Staff Scientists (2) would be mid-level M.D. or Ph.D. investigators with at least 10 years of professional experience and relevant education. Each would be initially assigned to a Task Force as staff coordinator. Staff scientists would be responsible to the Project Director and would play a major role in data collection and analysis, and preparation of reports.

d) Two M.S. or B.S. level associate scientists with at least 3-5 years experience in data analysis and review, as well as skills in scientific writing.

e) Consultant fees are based on the mid-point of daily fees paid by government contracts and private industry. Fees are necessary to attract and hold the most highly qualified persons as well as the necessity to require that they contribute substantially to the ESP and Task Force reports. Fees for ESP members are based on $500/day and those for Task Force members, $450/day. The budget assumes expert consultants from industry or government (as appropriate) would receive no compensation from UAREP.

f) $600 per trip is based on the average travel cost experience of UAREP and LSRO for 1982-83. Per diem is based on the daily rates for the Marriott Hotel, Bethesda, rate plus $24.00 per day.

g) Estimates derived from current projects and previous contractual activities of analogous scope and size.

h) Based on $550 per day (local Bethesda hotel rate).

i) One ESP meeting may be a four-day meeting outside the Bethesda area.

j) UAREP Indirect Cost rate at 39.6% estimated for 1983-84. Rate is based on total direct personnel salaries, wages, and consultant fees.

APPENDIX I

List of Persons and Organizations Consulted in the Feasibility Study

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APPENDIX II

Literature and Reference Material
Reviewed by UAREP/LSRO in the
Feasibility Study
(March - May 1983)

100 - 199

Memoranda (originating outside FASEB)

File No.	From	Subject	Date
100	McKusick, B.	Potentially hazardous chemicals at disposal sites	12/1/82
101	McKusick, B.	Superfund priority sites	1/7/83
102	McKusick, B.	Corn Report (file #500)	1/4/83
103	Foege, W.	Transmits CDC report on long-term followups of populations exposed to toxic hazards, #506	6/21/82
104	Tozzi, J., OMB	Toxic Torts -- evaluating merits of alternative legislative proposals (sent by T. O'Leary, CMA)	4/18/83

CMA Literature File

200 - 299

Letters (Incoming)

File no.	From	To	Subject	Date
200.0	Umland, C., Exxon	Karrh, B., du Pont	CDC's Superfund Responsibilities	12/22/82
200.1	Umland, C.	Endicott, K.	Transmits draft of on-going study (file #550(a),(b))	3/28/83
201	Karrh, B., du Pont	Zoll, D., CMA	Transmits summary of mortality study of chemical workers (file #620)	1/21/83
202	Thomas, L., Sloan-Ket.	Gov. Carey, New York	Transmits Love Canal report (file #505)	10/8/80
203.0	McKusick, B.	Higginson, J.	Transmits study summaries (file #204, 441(b), and 609)	3/16/83
203.1	Mckusick, B.	Higginson, J.	Transmits Contra Costa study (file #509)	3/16/83
203.2	McKusick, B.	Higginson, J.	Transmits summary of Contra Costa study (file #510)	2/28/83
203.3	McKusick, B.	Higginson, J.	Transmits study summaries (file #611-618)	3/16/83
203.4	McKusick, B.	Higginson, J.	Transmits Triana summary (#1017) & article on CA waste rules (#812)	4/13/83
204	Senger, J., Monsanto	Karrh, B., du Pont	Contains study summaries. (Enclosure to letter #203.0)	1/3/83
205	Parker, H. Union Carb.	McKusick, B du Pont	Transmits 2 studies on petrochemical workers (#440 & 441)	3/15/83
206	O'Leary, T., CMA	Higginson, J.	Transmits lists of banned chemicals from CFR (file #1013 & 1014), from EPA (file #1015), and CO_2 study (file #437)	4/19/83
207	Duchie, W., Shell Oil	Higginson, J.	Transmits 7 documents on McColl Site, California, file #519-525	4/13/83

Letters (Cont'd.)

File no.	From	To	Subject	Date
208	Gaffey, W.R., Monsanto Co.	Altman, P.	Transmits reprints (#445-447) and paper (#529) on 2,4,5 T production accident in W. Va.	4/20/83
209	Cole, P., U. of Alabama	Higginson, J.	Response to Dr. Endicott's letter of 4/5/83 on Feasibility Study	4/20/83
210	Gehring, P.J., Dow Chemical	Endicott, K.	Same as for #209	4/21/83
211	O'Leary, T.F., CMA	Endicott, K.	Transmits an OMB memo on toxic torts, file #104	4/22/83
212	Kissell, R.G., du Pont	Altman, P.	Transmits 2 letters from Kissel to CMA Superfund Task Group, file #213 and 214	4/28/83
213	Kissell, R.G., du Pont	CMA Superfund Task Group	EPA Expenditure Status	3/15/83
214	Kissell, R.G., du Pont	CMA Superfund Task Group	Analysis of Proposed National Priority List	2/16/83
215	Dourson, M.L., EPA	Altman, P.	Transmits EPA Document Listing, file #1018	4/29/83
216	Silbergeld, E., Env. Def. Fund	Fisher, K.	Response to UAREP letter of 4/5/83 on Feasibility Study	5/3/83
217	Levine, R.S., U. of Miami	Higginson, J.	Transmits report on hazardous wastes in Florida, and a summary, file #532(a) & (b)	4/28/83
218	Damstra, T., NIEHS	Fisher, K.	Transmits 3 lists of chemicals found in Superfund sites, file #1037 - 1038	5/11/83
219	Buffler, P.A., U. of Texas	Higginson, J.	Comments on Feasibility Study strategy	5/16/83
220	Paulson; Lundy, Audubon Society	Endicott, K.	Response to UAREP letter of 4/5/83 on Feasibility Study	5/17/83
221	Heath, C.W. CDC, Atlanta	Higginson, J.	Inquiried if further input was required of him for waste study	6/5/83

CMA Literature File

300 - 399

Books

File no.	Author	Title
300	Epstein, et al.	Hazardous Waste in America
* --	Brown, M.	Laying Waste: The Poisoning of America by Toxic Chemicals
301	Bridges; et al., editors	Banbury Report 13: Indicators of Genotoxic Exposure

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* Not numbered, to prevent damage, because book on loan from Geraldine Cox, CMA.

CMA Literature File

400 - 499

Journal Articles

File no.	Author(s)	Journal	Title
400	Sun, M.	Science	Dioxin's Uncertain Legacy
401	Anonymous	Inside EPA	SAB Criticizes RCRA Degree of Hazard Plan As Not Useful in Reg Decisions
402	Zuesse, E.	Reason	Love Canal--The Truth Seeps Out
403	Pell, S.	Transactions of N.Y. Acad. Sci.	Identification of Risk Factors in Employed Populations
404	Karrh; Pell	Annals of N.Y. Acad. of Sci.	Brain cancer in the du Pont Company
405	Hoar; Pell	J. Occup. Med.	A Retrospective Cohort Study of Mortality and Cancer Incidence Among Chemists
406	O'Berg, M.	J. Occup. Med.	Epidemiologic Study of Workers Exposed to Acrylonitrile
407	Pell, S.	J. Occup. Med.	Mortality of Workers Exposed to Chloroprene
408	Bonnett; Pell	J. Occup. Med.	Du Pont's Health Surveillance Systems
409	Pell; O'Berg; Karrh	J. Occup. Med.	Cancer Epidemiologic Surveillance in the du Pont Company
410	Pell, S.	J. Occup. Med.	Epidemiological Requirements for Medical-Environmental Data Management
411	Dow Chemical	The Point Is...	No Double Standard of Risk
412	Ott; Holder; Langner	J. Occup. Med.	Determinants of Mortality in an Industrial Population (have 2 copies)
413	Townsend; et al.	Am. J. Epidemiol.	Survey of Reproductive Events of Wives of Employees Exposed to Chlorinated Dioxins
414	Kramer; Mutchler	Amer. Indus. Hyg. Assoc. J.	The Correlation of Clinical and Environmental Measurements for Workers Exposed to Vinyl Chloride
415	Ott; Holder; Gordon	Archives of Environ. Health	Respiratory Cancer and Occupational Exposure to Arsenicals
416	Ott; Langner; Holder	Archives of Enriron. Health	Vinyl Chloride Exposure in a Controlled Industrial Environment
417	Ott; et al.	J. Occup. Med.	A Health Study of Employees Exposed to Vinylidene Chloride
418	Roush; Ott	Amer. Indus. Hyg. Assoc. J.	A Study of Benzene Exposure Versus Urinary Phenol Levels

Journal Articles (Cont'd.)

File no.	Author(s)	Journal	Title
419	Ott; Gordon; Schneider	J. Occup. Med.	Mortality Among Employees Chronically Exposed to Caustic Dust
420	Ott; et al.	Archives of Environ. Health	Mortality Among Individuals Occupationally Exposed to Benzene
421	Townsend; Ott; Fishbeck	J. Occup. Med.	Health Exam Findings Among Individuals Occupationally Exposed to Benzene
422	Kramer; et al.	Archives of Environ. Health	Health of Workers Exposed to 1,1,1,-trichloroethane: A Matched-Pair Study
423	Ott; Holder; Olson	J. Occup. Med.	A Mortality Analysis of Employees Engaged in the Manufacture of 2,4,5-trichlorophenoxyacetic acid
424	Ott; Scharnweber; Langner	British Journal Indus. Medicine	Mortality Experience of 161 Employees Exposed to Ethylene Dibromide in Two Production Units
425	Venable; et al.	J. Occup. Med.	A Fertility Study of Male Employees Engaged in the Manufacture of Glycerine
426	Ott; Kolesar; Scharnweber; Schneider; Venable	J. Occup. Med.	A Mortality Survey of Employees Engaged in the Development or Manufacture of Styrene-Based Products
427	Cook; Townsend; Ott; Silverstein	J. Occup. Med.	Mortality Experience of Employees Exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCCD)
428	Bond; et al.	Annals of N.Y. Acad. of Sci.	Case-Control Study of Brain Tumor Deaths Among Employees at a Chemical Manufacturing Plant
429	Egnatz; et al.	J. Occup. Med.	DBCP and Testicular Effects in Chemical Workers: An Epidemiological Survey in Midland, Michigan
430	Van Raalte; Grasso	Regulatory Toxicology and Pharmacology	Hematological, Myelotoxic, Clastogenic, Carcinogenic, and Leukemogenic Effects of Benzene
431	Kraybill, H.	Preventive Medicine	Evaluation of Public Health Aspects of Carcinogenic/Mutagenic Biorefractories in Drinking Water
432	Kraybill, H.	Jour. of Amer. Water Works Association	Carcinogenesis of Synthetic Organic Chemicals in Drinking Water
433	Yager; Hines Spear	Science	Exposure to Ethylene Oxide at Work Increases Sister Chromatid Exchanges in Human Peripheral Lymphocytes
434	Janerich; et al.	Science	Cancer Incidence in the Love Canal Area

Journal Articles (Cont'd.)

File no.	Author(s)	Journal	Title
435	Glotfelty; Taylor; Zoller	Science	Atmospheric Dispersion of Vapors: Are Molecular properties unimportant?
436	Hanis, N.; et al.	J. Occup. Med.	Cancer Mortality in Oil Refinery Workers
437	Riley; Bromberger-Barnea	Arch. Environ. Health	Monitoring Exposure of Brewery Workers to CO_2: A Study of Cellar Workers and Controls
438	Hanis, N.; et al.	J. Occup. Med.	Epidemiologic Study of Refinery and Chemical Plant Workers
439	Lynch, J.; et al.	J. Occup. Med.	An Association of Upper Respiratory Cancer With Exposure to Diethyl Sulfate
440 a&b	Austin; Schnatter	J. Occup. Med.	A Cohort Mortality Study of Petrochemical Workers (pre-publ. copy and reprint)
441	Austin; Schnatter	J. Occup. Med.	A Case-Control Study of Chemical Exposures and Brain Tumors in Petrochemical Workers
442	Kraybill, H.	(article in book): Water Chlorination ... Vol 1	Origin, Classification and Distribution of Chemicals in Drinking Water with an Assessment of their Carcinogenic Potential
443	Helmes, C; et al.	J. Environ. Sci. Health	Evaluation and Classification of the Potential Carcinogenicity of Organic Air Pollutants
444	Hohenemser; Kates; Slovic	Science	The Nature of Technological Hazard
445	Marsh, G.M.	Brit. Journal Indus. Medicine	Proportional mortality pattersn among chemical plant workers exposed to formaldehyde.
446	Marsh, G.M.	J. Occup. Med.	Mortality among workers from a plastic-producing plant
447	Zack, J.A.; Suskind, R.R.	J. Occup. Med.	The mortality experience of workers exposed to TCDD in a TCP process accident
448	Hanis, N.; et al.	Annals of N.Y. Acad. of Science	Brain Cancer: Issues and Dilemmas
449	Heath, C.W.	Environ. Health Perspectives	Field Epidemiologic Studies of Populations Exposed to Waste Dumps
450	Kreiss, K.; et al.	J. American Med. Assoc.	Cross-Sectional Study of a Community with Exceptional Exposure to DDT
451	Kreiss, K.; et al.	J. American Med. Assoc.	Association of Blood Pressure and Polychlorinated Biphenyl Levels
452	Clark, C.S., et al.	Arch. Environ. Health	An Environmental Health Survey of Drinking Water . . . in Hardeman County, Tennessee

CMA Literature File

500 - 549

Reports

File no.	Prepared by	Item
500	Corn, M., et al., Johns Hopkins Univ.	Corn Report
501	MITRE Corporation	A Ranking System for Hazardous Substances Releases
502	PEDCo Environmental	Analysis of Eckhardt Committee Survey, for CMA
503	National Research Council	Risk Assessment in the Federal Government
504	Office of Technology Assessment, U.S. Congress	Technologies and Management Strategies for Hazardous Waste Control
505	Thomas, L., Chairman, Governor's Panel, N.Y.	Love Canal Report
506	Heath, C., et al., CDC	Use of Long-term Registries for Health Followup of Populations Exposed to Toxic Hazards
507	Holmes, T.	Ph.D. Thesis Draft: A Mortality Study of Butyl-chlorobutyl rubber workers.
508	EPA	Ambient Water Quality Criteria for DDT.
509	Austin, D., California Dept. of Health Services	Contra Costa County Study on Cancer Incidence
510	Same as #509	Summary of Contra Costa County Study
511	Eckhardt Subcommittee, U.S.H.R.	Waste Disposal Site Survey Report
512	EPA	Hazardous Substances: National Contingency Plan
513	EPA	Proposed Superfund Priorities List of Sites
514(a)&(b)	JRB Associates	Review of Literature on Herbicides, Vol. 1 & 2 (Prepared for the Veterans Administration)
515(a)-(c)	California Department of Health Services	Carcinogen Identification Policy
516(1)-(10)	EPA	Notification of Hazardous Waste Sites, Regions 1-10 10 volumes.
517	Scientific Document Service, Seveso, Italy	CEFIC Health Protection Committee Documents. (Studies on accidental release of TCDD in Seveso
518	National Research Council	Long-range Environmental Outlook

Reports (Cont'd.)

File no.	Prepared by	Item
519	Shell Oil ?	A Chronological Summary of the McColl Site, Fullerton, CA (1942-1983).
520	California Dept. of Health Svcs. (DOHS)	Press release summarizing preliminary results of the McColl Site Technical Study, Phase I
521	Radian Corporation for: Calif. DOHS	McColl Phase 2: Environmental Setting of the McColl Site, Fullerton, CA
522	Radian Corporation for: Calif. DOHS	McColl Phase 2: Physical and Chemical Characterization and Distribution of the Waste at McColl
523	Radian Corporation for: Calif. DOHS	McColl Phase 2: Summary of the Nature and Extent of the Problem at the McColl Site
524	Radian Corporation for: Calif. DOHS	McColl Phase 2: Modeling of the Air Emissions from the McColl Site Investigation.
525	Calif. DOHS	Executive Summary of the McColl Site Health Survey
526	NCI; Mitre Corp.; et al. For: NIH	Biorefractories in water: carcinogens, mutagens promoters
527	Kraybill, H.F.	Animal Models and Systems for Risk Evaluation of Low Level Carcinogenic Contaminants in Water
528	Kraybill, H.F.	Outline on Contaminants, Type, Origin and Toxicity of Water Contaminants
529	Zack, J.A.; Gaffey, W. (Monsanto)	A mortality study of workers employed at the Monsanto Company Plant in Nitro, West Virginia
530	CDC	A System for Prevention, Assessment, and Control of Exposures and Health Effects from Hazardous Sites
531	NCI; SRI International	Organic Air Pollutants: Carcinogens, Mutagens, Promoters (52-page report)
532(a)&(b)	Levine, R.; Chitwood, D.; University of Miami; for: State of Florida	Analysis of Existing Data Pertaining to Potential Public Health Problems from Hazardous Wastes in Florida, and a 3-page summary
533	Fred C. Hart Associates for: EPA	Assessment of Hazardous Waste Mismanagement: Damage Case Histories
534	CDC (Heath, et al)	A Study of Cytogenetic Patterns in Persons Living Near the Love Canal
535	Task Force on Environ. Cancer and Heart and Lung Disease	Summary of the Workshop on Exposure to Environmenta Agents, Their Metabolism, and Mechanisms of Toxicity: Research Needs
536	National Research Council	Management of Hazardous Industrial Wastes: Research and Development Needs

Reports (Cont'd.)

File no.	Prepared by	Item
537	Office of Technology Assessment, U.S. Congress	Role of Genetic Testing in the Prevention of Occupational Disease

CMA Literature File

550 - 599

Studies Underway

File no.	Prepared by	Item
550(a)&(b)	Hanis, N., et al. (Exxon)	A Retrospective Mortality Study of Workers in Three Major U.S. Refineries and chemical plants 1970-1977. Parts 1 & 2

CMA Literature File

600 - 699

Abstracts & Summaries

File no.	Prepared by	Item
600	Ott, M., et al, (Dow)	Health Evaluation of Employees Occupationally Exposed to Methylene Chloride (Abstracts of general study and its subdivisions)
601	Bond, G., et al, (Dow)	A Case-Control Study of Brain Tumor Mortality at a Texas Chemical Plant
602	Ott, M., Langner (Dow)	A Mortality Survey of Men Engaged in the Manufacture of Organic Dyes
603	Cook, R., et al, (Dow)	A Cross-Sectional Study of Ethylene Glycol Monomethyl Ether Process Employees
604	Bond, G., et al, (Dow)	Medical and Morbidity Surveillance Findings Among Dow, Michigan Division Employees Potentially Exposed to TCDD.
605	Reeve, G., et al.	An Investigation of Brain Tumors Among Chemical Plant Employees Using a Sample-Based Cohort Method
606	Carlo, G., et al (Dow)	Assessment of Fertility Potential Among Male Employees
607	Carlo, G.	Clean Air Legislation and Its Scientific Initiatives
608	Currier, M., et al, (Dow)	A Cross-Sectional Study of Employees with potential Workplace Exposure to Ethylene Oxide.
609	Braun, D., Carlysle, S. (Union Carbide)	An Epidemiology Study of Silanes Workers
610	FASEB & NLM	Bibliography and Abstracts on Chemical Waste Disposal
611	Enterline, P., (Shell)	Shell Study: ECH
612	Whorton, D., et al, (Shell)	Shell Study: ECH Fertility Survey
613	Enterline, P. (Shell)	Shell Study: IPA
614	Enterline, P. (Shell)	Shell Study: Lubes Dewax
615	Whorton, D. et al, (Shell)	Shell Study: PTBBA Fertility Survey
616	Rushton, et al, (Am. Petr. Inst.)	Oil Distribution Centers -- Great Britain

Abstracts & Summaries (Cont'd.)

File no.	Prepared by	Item
617	Tabershaw-Cooper (Am. Petr. Inst.)	American Petroleum Institute Study: Refinery Study - U.S.A.
618	Am. Petr. Inst.	API: Epidemiology Studies (in progress)
619	Wen, C, et al.	Paper on Gulf Refinery Study
620	du Pont	General Mortality and Mortality Due to Cancer Among Workers in the Chemical Industry
621	Lipshultz, L.; et al.	Dibromochloropropane and its Effect on Testicular Function in Man from Journal of Urology 124(4)
622	Theriault; Goulet	A Mortality Study of Oil Refinery Workers from Journal of Occupational Medicine 21(5)
623	Hanis; Stavraky; Fowler	Cancer Mortality in Oil Refinery Workers from Journal of Occupational Medicine 21(3)
624	Austin; Schnatter	A Case-Control Study of Chemical Exposures and Brain Tumors in Petrochemical Workers, from Journal of Occup. Med. In press.
625	Hanis, N.; et al.	Study of Exxon Refinery and Chemical Plant from Journal of Occupational Medicine 24(3)
626	Rushton; Alderson	Study of British Oil Refineries from British Journal of Industrial Medicine 38
627	FASEB & NLM	Bibliography and abstracts on The Effects of Environmental Chemicals on the Immune System
628	FASEB & NLM	Bibliography and abstracts on Health Effects of Environmental Chemicals on the Adult Human Reproductive System
629	NCI	Asbestos and Health: An Annotated Bibliography of Public and Professional Education Materials

CMA Literature File

700 - 749

News Releases & Bulletins

File no.	Date	Item	Subject
700	2/7/83	CMA News Release	CMA's Joining in Suit Against DHHS and EPA
701	3/17/83	CMA News Release	Contract Award to UAREP for Feasibility Study
702	5/17/83	HHS News	CDC study of cytogenetic patterns in residents of Love Canal area (see file #534)

750 - 799

TSCA Bulletins (Toxic Substances Control Act) -- EPA Publication

File no.	Date
750	12/81
751	8/82
752	10/82
753	3/83

CMA Literature File

800 - 899

News Articles

File no.	Date	Source	Title
800	1/23/83	New York Times	Dioxin's Peril to Humans: Proof Is Elusive
801	2/13/83	Washington Post	Superfund "Fast Track" Mired in Procedure
802	3/18/83	Frederick Post	Dioxin Exposure Tests Uncover Only Skin Rashes
803	3/11/83	Science	House Reviews EPA's Record on Pesticides
804	1/22/83	S.F. Examiner	Smog Absolved in East Bay Cancer
805	1/22/83	Oakland Tribune	Higher Cancer Rate in Contra Costa Blamed on Smoking
806	unknown	New York Times	Panel Discounts Two Studies on Love Canal Problem
807	3/29/83	Washington Post	Efforts of Terrified Neighbors Brought Belated End to Dump Site
808	3/29/83	Washington Post	Shuffling EPA Inadequate, Environmentalists Say
809	3/25/83	Science	EPA's Troubles Reach a Crescendo
810	3/25/83	Science	Congress Investigates Malfeasance at EPA
811	unknown	Spotlight	A Framework for Environmental Health Research: the Harvard-EPA Cooperative Agreement
812	11/29/82	Chem. & Eng. News	California Setting New Hazardous Waste Rules
813	11/29/82	Chem. & Eng. News	Former EPA Official Addresses Waste Issues
814	4/23/83	Science News	Second Superfund Buyout Proposed: A Missouri Dioxin Site
815	4/23/83	Science News	Times Beach: The Long Road to Recovery
816	1983	Human Ecology and Health	Perceptions of Environmental Contamination and their Implications for Health
817	6/13/80	Science	Love Canal: False Alarm Caused by Botched Study
818	6/13/80	Science	Love Canal Residents Under Stress
819	May 1983	The Chemist	Surviving a State of Siege: Love Canal Revisited
820	5/28/83	Washington Post	Chemical Health Panel Ordered: To Be New HHS Agency
821	6/3/83	Science	Waste Management

CMA Literature File

900 - 999

Requests for Proposals

File no.	Issuer	Date	Title
900	EPA	3/11/83	Technical Handbook on Decontamination of Buildings, Structures, and Construction Equipment at Superfund Sites
901	NIEHS	3/10/83	Toxicology Research and Testing Program Health and Safety Support

CMA Literature File

1000 - 1099

Miscellaneous

File no.	General Description	Title or Subject	Prepared by
1000	Symposium Proceedings, Rockefeller Univ.	Assessment of Health Effects at Chemical Disposal Sites	Lowrance, W.
1001	Agenda & Registration Form	Fourth Annual Symposium on Environmental Epidemiology, Univ. of Pittsburgh, May '83	
1002	2 Tables	Data Development Work Group: Tables on Availability of Data	CMA
1003	Summary	Technical and Epidemiological Studies of the McColl Site (Fullerton, Cal.) 3/14/83	CMA ?
1004	12 Tables	Average Annual Age-Specific Death Rates in the du Pont Company Among Active and Pensioned Employees, 1957-71, and Among United States White Persons, 1964	du Pont
1005	Table	Mortality by Occupation and Industry Among Men 20-64 Years of Age: United States, 1950.	Guralnick, DHEW
1006	Draft for CMA Bd. of Dir. Meeting Book	Public Compensation (for harm by hazardous substances)	O'Leary, T. CMA
1007	Legal Brief with 3 Attachments	Comments of FMC Corporation on EPA's Proposed National Priorities List. Attachments: Analysis of NPA's Hazard Ranking System -- Potential Impacts :... at FMC/NOD Site -- Annotated National Priorities List	FMC Corp. Arthur D. Little, Inc
1008	Letter with Attached Application	Cooperative Agreement for Project on Impact of Toxic Substances Regulatory Alternatives On Industry, dated March 21, 1983	EPA
1009	Table	Top 50 Chemicals: Production was lower in 1981 for the Second Year Straight. From: Chemical & Engin. News, June 14, 1982	
1010	Table	Production of Chemicals and Allied Products. From: Survey of Current Business, Jan. 1983	
1011	Tables and List of Chem. Manufacturers	U.S. Production of Selected Synthetic Organic Chemicals	U.S. Int'l. Trade Com.
1012	1-page Chemical Profiles	Profiles of About 150 Chemicals From: Chemical Marketing Reporter, Issues dated between 1980 and 1982	

Miscellaneous (Cont'd.)

File no.	General Description	Title or Subject	Prepared by
1013	CFR, Title 21 parts 189-193	Food and Drugs - banned substances	Ofc. of Fed. Reg.
1014	CFR, Title 16 parts 1303-1500.17	Commercial Practices - banned substances	Ofc. of Fed. Reg.
1015	EPA Booklet	Suspended and Cancelled Pesticides	EPA
1016	Public Law 96-510	Comprehensive Environmental Response, Compensation, and Liability Act of 1980	US Congress
1017	1-page summary	Triana, Alabama (situation of waste site)	B. McKusick
1018	EPA Document List	Lists documents on various chemicals, prepared by Environ. Criteria and Assessment Office	EPA
1019	Table and Graph	Relative Carcinogenic Potencies Among 53 Chemicals Evaluated by the Carcinogen Assessment Group	EPA
1020	Section from a chapter of draft	Quantitative Estimation. From Chap. 13, Health Assessment Document for Acrylonitrile. By: Carcinogen Assessment Group	EPA
1021	Cover material for Scientists' Forum	Scientists' Forum on an Assessment of the Scientific Knowledge Base Related to Victim Compansation, Jan. 27-29, '82, Wash. DC	National Science Foundation
1022	Paper presented at above Forum	Identification ... of Health Hazards in Population: Epidemiological Assessment	R. Hoover
1023	"	Tracing the Movement of Pollutants ...	V. Freed
1024	"	Common law tort remedies for pollutant-induced disease	J.W. Wade
1025	"	Clinical Environ. & Occupational Medicine	Carnow; Conibear
1026	"	History of Victim Compensation Science	Silcox
1027	"	Model of compensation for pollutant-induced disease: the case of 2,4,5,T	Jasanoff
1028	"	Model ...: case of nitrilotriacetic acid	S. Albert
1029	"	Model of compensation for DES	L. Ritts
1030	"	Model of compensation for asbestos	S. Soble
1031	Technical Information	Tox Box^{TM}, a hazardous waste container, made by Lab Safety Supply, Wisconsin	Lab Safety Supply
1032	Equipment Catalogue	Safety Equipment for Science & Industry, Lab Safety Supply, Wisconsin	"

Miscellaneous (Cont'd.)

File no.	General Description	Title or Subject	Prepared by
1033	Equipment Catalogue	Air Sampling Catalog: Equipment and Supplies, Gilian Instrument Corp., Wayne, New Jersey	Gilian Corp.
1034 (a)&(b)	Toxline computer printout	Titles of articles on hazardous waste disposal	Toxline
1035 (a)&(b)	Enviroline computer printout	Titles of articles on hazardous waste disposal	Enviroline
1036	Report to Congress	Toxic Substances Control Act (TSCA) Report to Congress for Fiscal Year 1982	EPA
1037	List of chemicals	110 Chemicals found in Superfund waste sites, with specific sites listed for each chemical	CDC
1038	List of chemicals	15 chemicals occurring most frequently in Superfund waste sites	NIEHS
1039	List of chemicals	15 chemicals nominated by EPA for toxicologial testing	NPA
1040	Letter and attachment	Sun Information Services Company's computerized SunHealth Occupational Health Information Management System, dated 4/27/83	Sun Information Svcs. Co.
1041 a,b,c	Letter and two brochures	Diamond Shamrock Corp.'s Computerized Occupational Health & Environmental Surveillance System (COHESS), dated 4/29/83	Diamond Shamrock
1042	Scientific equipment catalogue	Industrial Hygiene News: 1983/84 Catalog and Buyer's Guide	Ind. Hyg. News
1043	News Letter	Occupational Health & Safety Letter, 4/8/83 (article on UAREP's study for CMA on page 4)	Environews, Inc.
1044	Ad literature and subscription form	*Chemical Regulation Reporter*, a weekly publ. of The Bureau of National Affairs, Inc.	Bureau of Nat. Af.
1045	Comments from CAST (Council for Agricul. Science & Technology)	The Missouri Dioxin Controversy: Scientific Overview	CAST

APPENDIX III

Key Reference Material

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APPENDIX IV

Partial List of Senior Scientific Experts Proposed for Executive Scientific Panel and Task Forces

Susan G. Austin, Sc.D.
Union Carbide Corporation

* Norman E. Breslow, Ph.D.
University of Washington

* Patricia A. Buffler, Ph.D.
University of Texas

* John Cairns, M.D.
Harvard University

Glyn G. Caldwell, M.D.
Centers for Disease Control

* Philip Cole, M.D.
University of Alabama

* Morton Corn, Ph.D.
Johns Hopkins University

Terri Damstra, Ph.D.
National Toxicology Program

* John Davies, M.D., M.P.H.
University of Miami

* W. Richard Doll, M.D., D.Sc.
Imperial Cancer Research Unit
Green College, Oxford University

Philip E. Enterline, Ph.D.
University of Pittsburgh

Joseph F. Fraumeni, Jr., M.D.
National Cancer Institute

William R. Gaffey, Ph.D.
Monsanto Company

Murray B. Gardner, M.D.
University of Southern California

Perry J. Gehring, Ph.D.
Dow Chemical, U.S.A.

James E. Gibson, Ph.D.
Chemical Industry Institute
of Toxicology

Joe W. Grisham, M.D., Ph.D.
University of North Carolina

Clark Heath, M.D.
Centers for Disease Control

Brian E. Henderson, M.D.
University of Southern California

Howard H. Hiatt, M.D.
Harvard University

Barbara Hulka, M.D., M.P.H.
University of North Carolina

* Donald Kennedy, Ph.D.
President, Stanford University

Herman F. Kraybill, Ph.D.
National Cancer Institute

Joshua Lederberg, Ph.D.
President, Rockefeller University

Brian MacMahon, M.D., Ph.D.
Harvard University

Tom S. Miya, Ph.D.
University of North Carolina

John A. Moore, D.V.M.
National Toxicology Program

Robert A. Neal, Ph.D.
President, Chemical Industry
Institute of Toxicology

* Frank J. Rauscher, Jr., Ph.D.
American Cancer Society

* Herbert S. Rosenkranz, Ph.D.
Case Western Research University

Carl Shy, M.D.
University of North Carolina

Ellen K. Silbergeld, Ph.D.
Environmental Defense Fund

* Edward A. Smuckler, M.D., Ph.D.
University of California

Benjamin F. Trump., M.D.
University of Maryland

Arthur C. Upton, M.D.
New York University

* Possible candidate for Executive Panel

Special Communication

Angiosarcoma of Liver in the Manufacture of Polyvinyl Chloride

Three reported cases of angiosarcoma of the liver serve as an alert to the probability that this condition may, in some instances, be causally related to employment in the manufacture of polyvinyl chloride resins.

J. L. Creech, Jr., M.D. and M. N. Johnson, M.D.

On January 22-23, 1974, a manufacturer of polyvinyl chloride and copolymers notified its employees, the National Institute of Occupational Safety and Health, the Kentucky State Department of Labor, and the public that three workers had died of angiosarcomas of the liver. These cases had as a common denominator employment in the manufacture of polyvinyl chloride resins.

A patient of one of the authors (J.L.C., Jr.) died on September 27, 1971 from an angiosarcoma of the liver. At that time the potential causal relation to the manufacture of polyvinyl chloride resins was not realized. Although other clinical manifestations of exposure to VCM were known (acroosteolysis), only one experimental paper[1] indicated that vinyl chloride may be a carcinogen. The tumors in rats that he described occurred primarily in the skin and were epidermoid in origin.

Eighteen months later on March 3, 1973, a second former employee in the Louisville plant died. Since he was not under our care, no connection was made that the two cases may have had a common origin. When a third patient died on December 19, 1973, the pathologist on gross examination diagnosed an angiosarcoma of the liver. Since all three patients were treated by different physicians, no relationship to exposure to polyvinyl chloride was surmised until the authors, recognizing the rarity of the tumor, and learning that all three had worked in the PVC plant, brought the matter to the attention of the company. Further search indicates that a fourth death from angiosarcoma may have occurred five years earlier (1968), but the death certificate shows primary liver tumor as the cause of death. (Clinical, epidemiologic, toxicological and occupational investigations are being vigorously pursued with regard to other employees who may have been similarly exposed.)

A brief resume of the first case under our care is presented. The history, clinical course, and pathologic findings are consistent with the others who died of angiosarcoma. These cases will be presented in a group and in more detail at a future date.

Case Report

Patient one, a 36-year-old white male, was hospitalized January 5, 1970 because of tarry stools.

Occupational History. — The patient had been employed from November 1955 until his illness, except for two lay-offs of nine months in 1958 and six months in 1959, as a chemical helper and operator in the Louisville plant of B. F. Goodrich Chemical Company in the manufacture of polyvinyl chloride resins.

Present Illness. — At the time of admission the patient had no complaints except passage of tarry stools. Past history was negative except for an operation for hemorrhoids four years earlier. Physical examination showed pallor, and black stool on rectal examination. Liver and spleen were not palpable. Although upper G.I. series was interpreted as normal, a tentative diagnosis was made of bleeding duodenal ulcer.

On diet and medication, the patient had no recurrence of

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overt bleeding until May 1, 1970, four months later, when he was readmitted for tarry stools. Again he was found to have marked pallor, and tarry stool on rectal examination. At this time the liver was demonstrably enlarged and the spleen was palpable. Blood chemistry studies (SMA 12) were not significantly abnormal: total bilirubin, alkaline phosphatase, LDH and SGOT were only slightly elevated. G.I. series showed only anterior displacement of the stomach, but barium swallow suggested esophageal varices. Intravenous pyelogram was normal; barium enema was normal except for displacement of the splenic flexure by an enlarged spleen. Liver scan on May 4, 1970 was interpreted as being compatible with a large lesion in the left lobe of the liver, extending into the right lobe.

Operative Procedure and Findings. — On May 7, 1970 an exploratory laparotomy was carried out. Under anesthesia the abdomen was entered through a vertical incision. Marked enlargement of the liver and spleen were noted, and the liver was adherent to the anterior surface of the stomach. The liver capsule was quite thickened and tough. There was a gelatinous-like material at the junction of the liver and the stomach. Retroperitoneal nodes were very soft, enlarged, and succulent in appearance. A biopsy of the liver was performed. There was marked degree of hemorrhage following this which was controlled with large sutures through the liver.

Report of Pathologic Findings

Gross. — Specimen is a portion of soft gray and light brown tissue that is 2.2 x 3.9 x 1 cms. at the present state. In some areas it appears to be composed of liver and in others light beige, moderately firm tissue with areas of sponginess and softness.

Microscopic. — Most of the tissue pattern of liver is replaced by luxuriantly growing cells forming irregular spaces. These spaces display many trabeculations in their lumina and are lined by pleomorphic malignant endothelial cells. Here and there these cells grow in nearly solid sheets. Elsewhere they line capillaries. There is a newly formed stroma with various degrees of hyalinization here and there.

Diagnosis. — "Liver, biopsy of, angiosarcoma (malignant hemangioendothelioma)."

Clinical Course

Following the laparotomy and biopsy the patient was given 4000 R cobalt and several intra-arterial infusions of 5-FU. During this time there was marked distortion of his blood chemistry with severe diminution of albumin and marked increase in total bilirubin, alkaline phosphatase, LDH and SGOT. After a stormy course and several months of convalescence, including several abdominal paracenteses for recurrent ascites, he recovered sufficiently to return to work for a short time. He succumbed to the liver tumor fourteen months post-operatively and died on September 27, 1971.

Reference

1. Viola PL, Bigotti A and Caputo A. Oncogenic response of rat skin, lungs, and bones to vinyl chloride. *Cancer Research* 31: 516-519, 1971.

Special Communication

Editor's Note

Professor Cesare Maltoni, Director of the Institute of Oncology of the University of Bologna, Italy, presented the results of his unpublished experiments with vinyl chloride on February 15, 1974 at the U.S. Dept. of Labor hearing in Washington, D.C. on "Possible Hazards of Vinyl Chloride Manufacture and Use". His work, which is obviously original and innovative, demonstrated angiosarcoma of the liver and other tumors in experimental animals at air concentrations of vinyl chloride which are known to be present at times in workroom atmospheres. Professor Maltoni describes his methodology as predictive of the carcinogenic potential of airborne chemical contaminants. Professor Maltoni is apparently the first to show the relationship between exposure to vinyl chloride and the appearance of angiosarcoma in mammals.

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Mortality Study of Workers in the Manufacture of Vinyl Chloride and its Polymers*

Irving R. Tabershaw, M.D. and William R. Gaffey, Ph.D.

Animal studies have shown that inhalation of vinyl chloride produces in rats angiosarcoma of the liver as well as cancers of the lung, kidney, skin and other sites. Although workers in occupations involving exposure to vinyl chloride have been found to have an increased risk of hemangiosarcoma, no excess of other cancers has so far been reported.

This historical prospective mortality study of 8384 men who had at least one year of occupational exposure to vinyl chloride before December 31, 1972, demonstrated that cancers of the digestive system (primarily angiosarcoma), respiratory system, brain, and cancers of unknown site, as well as lymphomas, occurred more often than expected in those members of the study population with the greatest estimated exposure. The mortality from other cancers was lower than that of the general male population, with the exception of cancers of the buccal cavity and pharynx. There was an excess of these cancers, which however was inversely related to estimated exposure. The explanation for the latter finding is not apparent.

The other major findings of the study are: (1) The overall mortality of the study population was approximately 75% of what would be expected in a comparable population of U.S. males. (2) No cause of death showed a statistically significant excess over what would be expected in a comparable U.S. male population; and (3) No deaths identified as angiosarcoma of the liver were found other than those previously identified.

This is the first epidemiological study which suggests that in humans vinyl chloride may also be associated with cancer of multiple sites.

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*The research on which this report is based was supported by a group of companies engaged in the synthesis and/or the polymerization of vinyl chloride and administered in their behalf by the Manufacturing Chemists Association.

Vinyl chloride in its manufacture and polymerization has been identified as a narcotizing agent[1] as a liver toxin,[2,3] and as a vasospastic agent producing a specific occupational disease, acroosteolysis.[4] Recently, vinyl chloride has been incriminated as a carcinogen producing in a group of workers engaged in the manufacture of polyvinyl chloride a rare fatal liver tumor, hemangiosarcoma.[5] Large doses of the chemical in rats reportedly produced cancer of the skin, lung and other organs.[6] Unpublished but public information[7] indicates that inhalation experiments with rats in doses easily reached in manufacturing operations produces in addition to angiosarcoma of the liver, skin, kidney and other malignant lesions.

The present study, however, was not restricted to the conditions and sites suggested by the above investigations, but concerned itself with the entire spectrum of causes of death, to the extent permitted by the size of the study group. The objectives of the study were: (1) To compare the mortality of individuals who have worked in vinyl chloride plants with that of the general population, (2) To compare mortality patterns within the population of vinyl chloride workers, based upon estimated occupational exposure, and (3) To compare mortality among vinyl chloride workers with the mortality of other occupational groups.

The study population consisted of individuals from 33 plants who had worked for at least one year in a job involving exposure to vinyl chloride before December 31, 1972, and included retired and terminated as well as active workers. For each such worker the date of birth and an employment history were obtained, and the vital status of the worker as of December 31, 1972, was ascertained. For those found to have died, those death certificates that were available were obtained and the cause of death determined. The observed mortality was compared with that of the United States male population.

Data Collection

In each plant, data were collected for each worker stated by the plant management to have been employed for at least one year in a job involving exposure to vinyl chloride. In some plants, particularly those producing the monomer, this determination could be made on the basis of job titles. Usually, however, exposure was a function of both job title and the location of the job in the plant, so that the assessment of exposure had to be made on a case-by-case basis by plant officials.

Data were collected for as far back in time as complete records were kept. In most cases this covered the entire history of the plant. In others, records were kept for a fixed period such as a decade. In a few, records were kept for different periods, depending on whether the worker had died on the job or had left employment.

In most plants it was impossible to quantify exposure. However, industrial hygiene and safety personnel in each plant were able to identify certain jobs and locations as involving the highest exposures in the plant, and to classify other exposures as medium or low relative to the "high" represented by the jobs with the greatest exposure. Consequently, each exposed job in a worker's history was scored 1, 2 or 3 to indicate low, medium or high estimated exposure.

This gross classification has two major failings as a result of the subjective nature of the estimates. The first is that the scores represent estimated relative exposure within a given plant. It is therefore possible that in objective terms, a "high" score in one plant corresponds to a "medium" or even "low" score in another. The second is that the scores usually do not take into account changes in exposure over time. A worker with long service may therefore have had jobs in the remote past which involved "low" exposure relative to other jobs at that time, but which might be "high" in comparison with current exposures in the same job. This subjective classification is therefore of questionable validity in characterizing the exposure of a given worker. For epidemiological purposes, however, those who have high scores can reasonably be expected, on the average, to have had the greatest exposure, while those with low scores will have had the least, even though the true exposure in each group may vary considerably from person to person.

The estimated exposure history of each worker was summarized by calculating an Exposure Index (EI). This was done by multiplying the number of months on each job by the exposure score, totalling these over all exposed jobs, and dividing by the total number of months of exposure.

Follow-up of Study Population

A follow-up procedure was instituted for those who had left employment and whose vital status could not be determined at the local plant, using direct mail follow-up and retail credit bureau investigations. Table 1 shows the vital status of the population as of December 31, 1972. Follow-up is 85% complete. Those who were not found were born (and began their exposure) about ten years before the group on which follow-up was complete, and had about half the duration of employment in exposed jobs with a slightly higher EI. Although there appears to be nothing very unusual about this group in terms of work history and exposure, it is nevertheless true that their exposures took place further back in time than that of the group successfully traced. It is therefore possible that their mortality, after a substantial latent period, might show a somewhat different pattern from that of the traced group.

All of the subsequent analysis is concerned with the 7128 workers on whom follow-up was complete. Table 2 shows their distribution by duration of exposed employment and the year in which that employment began. Although almost half the study group first entered exposed employment in 1960 or later, there are nevertheless 854 workers with 20 years or more exposure, and 1640 with 15 years or more. Table 3 shows the relationship between duration of exposure and EI. There does not appear to be a close relationship between the EI

Table 1. — Follow-up Status of 8384 Vinyl Chloride Workers.

	Total	Alive	Dead	Unknown	Death Certificates	
					Rec'd	Not Rec'd
Number	8384	6776	352	1256	328	24
Percent	100.0	80.8	4.2	15.0	92.7	7.3

and the duration of exposure, that is workers with a higher EI do not differ substantially in duration of exposure from those with a lower EI. One implication is that in assessing the relationship between mortality and exposure both duration and level of exposure can be examined separately as well as in combination.

Calculation of Risk of Death

The risk of death is expressed as a Standardized Mortality Ratio (SMR), which is the ratio of the number of observed deaths in the study population to the number of deaths to be expected in a comparable population of U.S. males. SMR's were calculated for overall mortality and for 33 major cause groups.

Table 4 shows observed and expected deaths, and the SMR, for each of these causes for the total study group. In calculating the SMR's for specific causes, the 24 deaths for which no certificates were found were assumed to have the same cause distribution as those for which certificates were available.

In the standard population each SMR would be equal to 100. Therefore the statistical significance of the deviation of each SMR in the study population from the expected value of 100 was tested.[8] A single dagger indicates those SMR's which differed significantly from 100 at the 5% level, that is, which had a probability of .05 or less of occurring by chance. A double dagger indicates those which were significant at the 1% level. SMR's based on fewer than five observed cases were not tested for significance.

Table 5 shows the same SMR's for workers with an Exposure Index below 1.5 versus those at 1.5 or above. The dividing point of 1.5 represents a level halfway between "low" and "medium."

Table 6 shows similar results for workers with less than five years exposure versus those with five years or more.

In order to examine the possible interaction between duration and level of exposure, the study population was divided into four groups on the basis of both EI (low vs high) and duration of exposure (short vs long) using the same dichotomization as Tables 5 and 6.

Table 7 shows the results for short versus long exposure in the low EI group, and Table 8 shows the same comparison in the high EI group.

In each of the above tables, deaths for which certificates had not been received were assumed to be distributed as a uniform percentage of all causes. The cause specific SMR's were therefore adjusted upward by a percentage which varied in each subgroup.

Results of Analysis

The overall mortality of the study population is statistically significantly lower than that of the U.S. male population. There were 352 observed deaths compared with 467 expected, for an SMR of 75.

Table 4 shows that no specific cause of death was statistically significantly greater than expected. Several, particularly heart disease, accidents and "other diseases" not detailed in the tables, were significantly below their expected values.

When the study population is divided according to intensity and duration of exposure (Tables 5 and 6) and combinations of these measurements (Tables 7 and 8) three major patterns emerge.

For malignant neoplasms as a whole, the SMR increases with increasing exposure, whether measured by level, duration, or both. In the high exposure group with 5 years or more exposure (Table 8) there are 36 observed cases and 26.11 expected.

For cardiovascular — renal diseases as a group, there are also increases in the SMR with increasing exposure, but the numbers of observed cases remain less than expected, the differences being statistically significant in all groups except the high exposure, long duration group in Table 8.

For all other causes, there are no consistent relationships with exposure.

Within the malignant neoplasms, the largest (although not statistically) significant SMR is in cancers of the buccal cavity and pharynx, with five observed, 2.84 expected, and an SMR of 189. However, Tables 5 to 8 show that all these cases have Exposure Indexes below 1.5 and four out of the five have less than five years exposure. Table 10 is a listing of these deaths with age at death, duration of exposure, and cause as stated on the death certificate.

Cancer of the digestive system shows

Table 2. — Distribution of Months in Exposed Employment by Year in Which Exposure Began, for 7128 Vinyl Chloride Workers with Completed Follow-up

Year Exp Started	Total	Months of Exposure <60	60-119	120-179	180-239	240-299	300-359	360-419	Unknown
1930-39	35	2	4	1	4	6	13	5	
1940-49	1048	135	93	119	151	277	237	34	2
1950-59	1962	389	257	383	631	282			20
1960-69	3368	1714	1442	195					17
1970-71	715	715							
Total	7128	2955	1796	698	786	565	250	39	39

Table 3. — No. and % of 7128 Vinyl Chloride Workers by Months of Exposed Employment and Exposure Index.

Exposure Index	Total No. %	Months of Exposure <60 No. %	60-119 No. %	120-179 No. %	180-239 No. %	240-299 No. %	300-359 No. %	360-419 No. %	Unknown
1.0-1.4	4032 (100)	1715 (43)	1125 (28)	402 (10)	406 (10)	274 (7)	92 (2)	18 (0)	
1.5+	3057 (100)	1240 (41)	671 (22)	295 (10)	380 (12)	291 (10)	159 (5)	21 (1)	
Unknown	39								39
Total	7128 (100)	2955 (41)	1796 (25)	697 (10)	786 (11)	565 (8)	251 (4)	39 (1)	39 (1)

Table 4 — Observed Deaths/Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers.

Cause of Death with I.C.D. Number	Obs/Exp	SMR*
All causes	352/467.28	75 ‡
Tuberculosis (001-019)	1/5.71	19
Tuberculosis of respiratory system (001-008)	1/5.30	20
Malignant neoplasms (140-205)	79/77.16	110
Malignant neoplasms: buccal cavity and pharynx (140-148)	5/2.84	189
Malignant neoplasms: digestive organs and peritoneum (150-159)	19/21.67	94
Malignant neoplasms: respiratory system (160-164)	25/23.93	112
Malignant neoplasms: genital organs (170-179)	3/3.55	91
Malignant neoplasms: urinary organs (180-181)	1/3.60	30
Malignant neoplasms: other and unspecified sites (190-199)	17/11.75	155
Leukemia and aleukemia (204)	3/3.77	85
Lymphomas (200-203, 205)	6/6.06	106
Diabetes mellitus (260)	7/4.41	170
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	155/207.46	80 ‡
Vascular lesions affecting CNS (330-334)	13/24.48	57 ‡
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	5/6.87	78
Arteriosclerotic heart disease (420)	121/137.33	95
Nonrheumatic endocarditis (421, 422)	1/6.58	16
Hypertensive heart disease (440-443)	3/9.33	35
Other hypertensive disease (444-447)	3/1.60	178
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/4.27	0
Influenza and pneumonia (480-493)	5/9.96	54 †
Ulcer of stomach and duodenum (540, 541)	2/3.83	56
Appendicitis (550-553)	0/0.66	0
Hernia and intestinal obstruction (560, 561, 570)	1/1.51	71
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/1.31	87
Cirrhosis of liver (581)	3/15.60	21
Hyperplasia of prostate (610)	0/0.30	0
Symptoms, senility and ill-defined conditions (780-795)	1/7.34	15
All other diseases (residual)	21/45.78	49 ‡
Motor vehicle accidents (810-835)	17/32.70	56 ‡
Other accidents (800-802, 840-962)	18/30.64	63 ‡
Suicide (963, 970-979)	16/16.05	107
Homicide (964, 980-985)	1/11.98	9
No. of workers	7128	
Person-years	77846	

* SMR's adjusted for deaths with cause unknown
† Significant at 5% level
‡ Significant at 1% level

no excess in the study population as a whole. However, in those workers with an EI of 1.5 or higher, there are 12 observed cases where 9.14 are expected (Table 5). In the subgroup of the above workers with five years or more exposure, there are 11 observed cases and 7.47 expected.

Respiratory cancer shows a slight excess in the total group, and a similar pattern for different exposure categories, with 13 observed versus 10.28 expected when the Exposure Index is 1.5 or higher, and 12 observed versus 8.50 expected when, in addition, the duration of exposure is five years or more.

Malignant neoplasms of other and unspecified sites show an excess in the total group, and an increase with both level and duration of exposure (Tables 5 and 6). The relationship with exposure is more pronounced, since those with exposures of less than five years have fewer cases than expected.

The lymphomas, although occurring at about the expected rate when the whole group is considered, are concentrated almost entirely in the high exposure, long duration group. In that category there are four cases observed and 1.84 expected.

Cancers of the genital and urinary organs, and leukemia, have fewer cases than expected. The number of cases is too small to examine any trends.

Discussion

The favorable overall mortality of the study population is a phenomenon commonly observed in working populations. Standardized Mortality Ratios in the low 80's and below have been found.[9,10] Even in occupations with well defined hazards which cause an increased risk from a specific cause, the overall mortality may still be favorable because of the low risk from other major causes of death, frequently in the cardiovascular — renal category.[11]

Table 5. — Observed Deaths/Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers, by Estimated Level of Exposure.

Cause of Death with I.C.D. No.	D < 1.5		D ≥ 1.5	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All Causes	188/270.33	79 ‡	157/195.68	80 ‡
Tuberculosis (001-019)	0/3.18	0	0/2.33	0
Tuberculosis of respiratory system (001-008)	0/3.16	0	0/2.18	0
Malignant neoplasms (140-205)	37/44.28	90	41/32.67	134
Malignant neoplasms, buccal cavity and pharynx (140-148)	5/1.62	330	0/1.21	0
Malignant neoplasms, digestive organs and peritoneum (150-159)	7/12.50	60 †	12/9.14	141
Malignant neoplasms, respiratory system (160-164)	11/13.56	86	13/10.28	135
Malignant neoplasms, genital organs (170-179)	2/2.30	93	1/1.43	75
Malignant neoplasms, urinary organs (180-181)	1/2.07	51	0/1.52	0
Malignant neoplasms, other and unspecified sites (190-199)	9/6.57	146	8/4.52	190
Leukemia and aleukemia (204)	1/2.18	49	2/1.57	136
Lymphomas (200-203, 205)	1/3.48	31	5/2.54	212
Diabetes mellitus (260)	5/3.65	146	2/2.65	81
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	84/120.11	75 ‡	69/86.99	85 †
Vascular lesions affecting CNS (330-334)	7/14.42	52 ‡	6/10.06	64
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	3/3.98	80	2/2.85	75
Arteriosclerotic heart disease (420)	68/78.94	92	51/58.05	95
Nonrheumatic endocarditis (421, 422)	0/4.70	0	1/2.89	38
Hypertensive heart disease (440-443)	1/5.48	19	2/3.86	56
Other hypertensive disease (444-447)	1/1.52	70	2/1.07	201
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/2.50	0	0/1.77	0
Influenza and pneumonia (480-491)	5/5.80	92	0/4.13	0
Ulcer of stomach and duodenum (540, 541)	1/2.21	48	1/1.60	68
Appendicitis (550-553)	0/0.39	0	0/0.27	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.80	0	1/0.63	171 †
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.76	0	1/0.55	196
Cirrhosis of liver (581)	2/8.90	23	1/6.64	16
Hyperplasia of prostate (610)	0/0.25	0	0/0.14	0
Symptoms, senility and ill-defined conditions (780-795)	0/4.22	0	1/3.09	34
All other diseases (residual)	14/21.90	68 †	6/15.89	41 ‡
Motor vehicle accidents (810-835)	8/19.08	45 ‡	9/13.46	72
Other accidents (800-802, 840-962)	11/17.83	66 †	6/12.67	50 ‡
Suicide (963, 970-979)	9/9.73	99	7/7.07	107
Homicide (964, 980-985)	0/6.90	0	1/4.94	21
No. of workers	4032		3957	
Person-years	45354		32108	

*SMR's adjusted for deaths with cause unknown

†Significant at 5% level

‡Significant at 1% level

In view of these facts, SMR's which are higher than expected may be worthy of attention even if they are not statistically significant. This is especially true in the present study since the number of deaths from many causes is quite small, and even a relatively high SMR may not reach statistical significance.

If, in addition, a particular cause shows a consistent pattern of increase with exposure or estimated exposure, the findings are particularly interesting.

By these criteria, mortality from digestive cancer, respiratory cancer, cancer of other and unspecified sites, and lymphomas, appear to be related to exposure as defined in this study.

In view of the association between vinyl chloride exposure and angiosarcoma of the liver, the digestive cancers were examined further to see what contribution angiosarcoma made to the observed mortality pattern.

Of the 19 digestive cancers, seven were liver cancers, of which two were angiosarcomas according to the death certificate. However, among angiosarcoma deaths in vinyl chloride workers identified by other investigators, there were six which occurred in the present study population during the study period. They were all found in the course of the study. Table 11 shows these cases with the cause of death as given on the death certificate. Note that one case was certified as cirrhosis, and was so considered throughout this study, since the validity of comparisons with population data required that cause of death be determined only from information on the death certificate. The other five were correctly classified as

Table 6 — Observed Deaths/Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers by Duration of Exposed Employment

Cause of Death with I.C.D. No.	< 60 months		≥ 60 months	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All causes	94/140.53	67‡	251/329.30	76‡
Tuberculosis (001-019)	0/2.27	0	0/3.55	0
Tuberculosis of respiratory system (001-008)	0/2.07	0	0/3.33	0
Malignant neoplasms (140-205)	13/19.96	76	65/57.61	116
Malignant neoplasms buccal cavity and pharynx (140-148)	4/0.70	688	1/2.16	47
Malignant neoplasms digestive organs and peritoneum (150-159)	2/5.76	46	17/16.56	106
Malignant neoplasms respiratory system (160-164)	3/5.57	65	21/18.51	116
Malignant neoplasms genital organs (170-179)	0/0.99	0	3/2.76	117
Malignant neoplasms urinary organs (180-181)	0/0.83	0	1/2.79	37
Malignant neoplasms other and unspecified sites (190-199)	2/3.40	71	15/8.23	187
Leukemia and aleukemia (204)	1/1.25	96	2/2.53	81
Lymphomas (200-203, 205)	1/2.01	60	5/4.07	126
Diabetes mellitus (260)	2/1.80	118	5/4.54	113
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	28/51.45	65‡	125/157.39	81‡
Vascular lesions affecting CNS (330-334)	4/5.97	81	9/18.71	49‡
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/2.39	101	3/4.53	68
Arteriosclerotic heart disease (420)	21/32.54	78	98/105.39	96
Nonrheumatic endocarditis (421, 422)	0/1.79	0	1/1.35	70
Hypertensive heart disease (440-443)	1/2.42	49	2/7.01	30
Other hypertensive disease (444-447)	0/0.79	0	3/1.87	170
Chronic & unspecified nephritis & renal sclerosis (592, 594)	0/1.55	0	0/2.76	0
Influenza and pneumonia (480-493)	3/2.91	123	2/7.09	29
Ulcer of stomach and duodenum (540, 541)	1/1.07	112	1/2.78	37
Appendicitis (550-553)	0/0.74	0	0/0.43	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.42	0	1/1.10	93
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.40	301	0/0.92	0
Cirrhosis of liver (581)	1/4.45	26	2/11.18	18
Hyperplasia of prostate (610)	0/0.07	0	0/0.31	0
Symptoms, senility and ill-defined conditions (780-795)	1/2.78	53	0/5.05	0
All other diseases (residual)	4/11.97	40	16/24.34	63
Motor vehicle accidents (810-835)	10/16.14	75	7/16.65	41‡
Other accidents (800-802, 840-962)	7/12.94	65†	10/17.82	58
Suicide (963, 970-979)	6/5.34	114	10/10.28	100
Homicide (964, 980-985)	1/5.80	20	0/6.70	0
No. of Workers	2955		4134	
Person-years	34701		43240	

*SMR's adjusted for deaths with cause unknown

†Significant at 5% level

‡Significant at 1% level

Table 7. — Observed Deaths/Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers with Exposure Indices Below 1.5, by Duration of Exposed Employment

Cause of Death with I.C.D. No.	< 60 Months Exposure		≥ 60 Months Exposure	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All Causes	56/89.23	63+	132/181.28	73+
Tuberculosis (001-019)	0/1.41	0	0/1.97	0
Tuberculosis of respiratory system (001-008)	0/1.31	0	0/1.85	0
Malignant neoplasms (140-205)	8/12.86	73	29/31.46	95
Malignant neoplasms, buccal cavity and pharynx (140-148)	4/0.45	1036	1/1.17	88
Malignant neoplasms, digestive organs and peritoneum (150-159)	1/3.43	34	6/9.08	68
Malignant neoplasms, respiratory system (160-164)	2/3.58	65	9/10.00	93
Malignant neoplasms, genital organs (170-179)	0/0.64	0	2/1.62	127
Malignant neoplasms, urinary organs (180-181)	0/0.55	0	1/1.53	67
Malignant neoplasms, other and unspecified sites (190-199)	1/0.97	120	8/4.43	187
Leukemia and aleukemia (204)	0/0.79	0	1/1.40	73
Lymphomas (200-203, 205)	0/1.76	0	1/2.23	46
Diabetes mellitus (260)	2/1.15	201	3/2.50	124
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	21/31.18	73 *	63/86.82	75 *
Vascular lesions affecting CNS (330-334)	2/3.93	59	5/10.51	49 †
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/1.30	155	1/2.49	41
Arteriosclerotic heart disease (420)	16/21.14	84	57/57.87	93
Nonrheumatic endocarditis (421, 422)	0/1.18	0	0/3.02	0
Hypertensive heart disease (440-443)	1/1.58	74	0/3.90	0
Other hypertensive disease (444-447)	0/0.50	0	1/1.02	101
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/0.97	0	0/1.53	0
Influenza and pneumonia (480-493)	3/1.86	188	2/3.95	53
Ulcer of stomach and duodenum (540-541)	0/0.68	0	1/1.53	67
Appendicitis (550-553)	0/0.15	0	0/0.24	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.27	0	0/0.61	0
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.26	0	0/0.51	0
Cirrhosis of liver (581)	1/2.80	42	1/6.09	16
Hyperplasia of prostate (610)	0/0.05	0	0/0.20	0
Symptoms, senility and ill-defined conditions (780-795)	0/1.43	0	0/2.79	0
All other diseases (residual)	4/7.45	63	10/12.92	79
Motor vehicle accidents (810-835)	3/9.87	35	5/9.22	56
Other accidents (800-802, 840-962)	3/7.98	44	8/9.65	83
Suicide (963, 970-979)	3/4.68	86	6/5.66	109
Homicide (964, 980-985)	0/3.55	0	0/3.43	0
No. of Workers	1715		2317	
Person years	21418		23920	

* SMR's adjusted for deaths with cause unknown

† Significant at 5% level

+ Significant at 1% level

Table 8. — Observed Deaths/Expected Deaths and Standardized Mortality Ratios in Vinyl Chloride Workers with Exposure Indices of 3.5 or Greater, by Duration of Exposed Employment.

Cause of Death with I.C.D. No.	< 60 Months Exposure		≥ 60 Months Exposure	
	Obs/Exp	SMR*	Obs/Exp	SMR*
All Causes	38/47.93	79	119/147.81	81
Tuberculosis (001-019)	0/0.76	0	0/1.57	0
Tuberculosis of respiratory system (001-008)	0/0.71	0	0/1.48	0
Malignant neoplasms (140-205)	5/6.57	96	36/26.11	141
Malignant neoplasms: buccal cavity and pharynx (140-148)	0/0.73	0	0/0.99	0
Malignant neoplasms: digestive organs and peritoneum (150-159)	1/1.67	76	11/7.47	151
Malignant neoplasms: respiratory system (160-164)	1/1.79	71	12/8.50	144
Malignant neoplasms: genital organs (170-179)	0/0.29	0	1/1.41	73
Malignant neoplasms: urinary organs (180-181)	0/0.26	0	0/1.26	0
Malignant neoplasms: other and unspecified sites (190-199)	1/1.18	107	7/3.51	204
Leukemia and aleukemia (204)	1/0.44	288	1/1.13	90
Lymphomas (200-203, 205)	1/0.71	178	4/1.84	222
Diabetes mellitus (260)	0/0.61	0	2/2.04	100
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	7/16.54	54‡	62/70.46	90
Vascular lesions affecting CNS (330-334)	2/1.87	135	4/8.19	50
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	0/0.82	0	2/2.04	100
Arteriosclerotic heart disease (420)	5/10.41	61†	46/47.65	98
Nonrheumatic endocarditis (421-422)	0/0.57	0	1/2.32	44
Hypertensive heart disease (440-443)	0/0.76	0	2/3.10	66
Other hypertensive disease (444-447)	0/0.27	0	2/0.81	253
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/0.50	0	0/1.23	0
Influenza and pneumonia (480-493)	0/0.99	0	0/3.13	0
Ulcer of stomach and duodenum (540-541)	1/0.35	362	0/1.25	0
Appendicitis (550-553)	0/0.08	0	0/0.19	0
Hernia and intestinal obstruction (560-561, 570)	0/0.14	0	1/0.49	209
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.14	904	0/0.41	0
Cirrhosis of liver (581)	0/1.56	0	3/5.08	70
Hyperplasia of prostate (610)	0/0.01	0	0/0.13	0
Symptoms, senility and ill-defined conditions (780-795)	1/0.80	158	0/2.30	0
All other diseases (residual)	0/4.03	0	6/11.82	51†
Motor vehicle accidents (810-835)	7/6.05	146	2/7.43	28
Other accidents (800-807, 840-962)	4/4.73	107	2/7.96	26
Suicide (963, 970-979)	3/2.40	158	4/4.67	88
Homicide (964, 980-985)	1/2.18	58	0/2.76	0
No. of Workers	1748		1817	
Person-Years	17878		19305	

* SMR's adjusted for deaths with cause unknown

† Significant at 5% level

‡ Significant at 1% level

Table 9. — Other Malignancies (190-199, I.C.D.) in VDM Epidemiology Study Population.

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
2744	38	186	Widespread metastatic melanoma (1 yr.) Malignant melanoma of back	yes
3700	46	50	Malignant melanoma with widespread metastases	no
7096	67	69	Brain tumor (carcinoma) (8 mos.)	
3846	43	64	Meningitis and pneumonitis (5 wks.) Ependymoma, fourth ventricle brain (2 mos. post op craniotomy)	no
4433	58	81	Astrocytoma, malignant left cerebral hemisphere (1 yr.)	yes
4800	61	51	Carcinoma of the brain Arteriosclerosis Pneumonia due to static congestion	no
7279	57	271	Brain tumor, glioblastoma multiforme (18 mos.)	
7300	48	211	Brain tumor, malignant (2 mos.)	
7371	58	289	Brain tumor, malignant	no
7306	53	716	Thyroid carcinoma with metastases	no
5769	57	239	Carcinomatosis Carcinoma vertebral body	yes
5746	67	201	Liposarcoma with metastasis (3 mos.)	yes
2850	64	193	Carcinomatosis, primary region not known (Past history: rheumatoid arthritis, myocardial infarction)	no
4778	54	300	Cardiac arrest (1 hr.) Respiratory arrest and cerebral hypoxia (2 days) Widespread metastatic carcinoma (2 mos.)	
4786	61	318	Generalized metastasis undifferentiated (7 mos.) Squamous cell carcinoma, primary site undetermined	yes
5783	69	166	Metastatic carcinoma of abdomen Original site undetermined (Pulmonary emphysema)	
7301	55	133	Carcinomatosis (4 mos.) Primary site undetermined	no

liver cancer but only two were specified as angiosarcoma.

If there had been no angiosarcomas, the number of deaths classified as cirrhosis in this study would have decreased by one and the number classified as digestive cancer would have decreased by five. The pattern of duration and intensity of exposure in these five cases was such that if they had not been present there would have been no relationship between exposure and digestive cancer. The mortality pattern in this cause group is therefore attributable to angiosarcomas of the liver.

The other cause group worth further investigation is cancer of other and unspecified sites, both because it is a heterogeneous category and because it seems, unlike the other cancers, to be more related to duration than to level of exposures.

Table 9 shows a list of the specific causes included in this category, which is essentially brain cancer and generalized cancer with primary site unknown. About 40% of the observed deaths were due to brain cancer. In the general male population, about 22% of this category is due to brain cancer, so that not only is the mortality from cancer of other and unspecified sites excessive, but brain cancer is overrepresented within the category.

The possibility exists based on the lack of specificity of some of the listed causes that some of the brain cancers were not primary, but metastases from another unidentified site such as the lung.

The cancers of the buccal cavity and pharynx are difficult to explain because of their occurrence in the low exposure, short exposure group. It is possible that this is a chance occurrence, that exposures to other substances were involved, or that the mouth and pharynx may be peculiarly susceptible because of the gaseous nature of the chemical.

Possible Biases in the Calculation of Risk

There are two major potential sources of bias in the study. The first is that the follow-up rate is lower than is desirable. The second is that observations of workers with long exposures followed by a long latent period are not adequately represented so that the power of the study to detect causes of death associated with long exposure and long latency is impaired. Populations with such characteristics exist and should be investigated.

Table 10 — Malignant Neoplasms of Buccal Cavity and Pharynx (140-148, I.C.D.) in VCM Epidemiology Study Population

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
300	31	49	Carcinoma of lip with metastasis to lung and neck (5 yrs.)	no
3431	56	30	Pulmonary edema (50 min.) Pulmonary metastases (Carcinoma, epidermoid, tongue and mandible)	no
3001	57	71	Metastatic squamous cell ca. primary lesion palate	yes
7365	54	40	Atelectasis due to metastasis to mediastinum (4 mos.) Multiple malignant metastases to brain, liver & mediastinum (1 yr.) Adenocarcinoma of nasal pharynx (2 yrs.)	yes
3354	63	26	Massive hemorrhage into tracheo-tree Status post laryngo-pharyngectomy left radial neck dissection Well differentiated keratinizing squamous cell carcinoma of left pyriform sinus (mos.)	yes

Table 11 — Angiosarcoma Deaths in VCM Epidemiology Study Population

Study No.	Age at Death	Mos. Exposed	Cause as Given	Autopsy
4750	54	203	Cirrhosis of liver (sev. weeks)	yes
4755	60	281	Bleeding from hepatoma (3 days) (Laennecs cirrhosis)	yes
5765	65	167	Angiosarcoma rt. lobe of liver (10 mos.)	yes
7303	38	174	Liver failure (1 mo.) Cancer of liver, primary (15 mos.)	
7376	52	238	Cardiac tamponade and massive left hemothorax (mins.) Widely metastatic angiosarcoma of liver (4 mos.)	
7385	43	214	Hepatic failure Primary carcinoma liver	yes

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Respiratory cancer in relation to occupational exposures among retired asbestos workers[1]

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Enterline, P., de Coufle, P., and Henderson, V. (1973). *British Journal of Industrial Medicine*, **30**, 162-166. **Respiratory cancer in relation to occupational exposures among retired asbestos workers.** A cohort of 1 348 men who completed their working lifetime in the asbestos industry and retired with an industry pension during the period 1941-67 was observed through 1969 for deaths. The average length of employment in the asbestos industry for these men was 25 years and all had exposures to asbestos dust. In some instances these exposures were very high and continued for many years. Mortality for this cohort of men after age 65 was 14·7% higher than for the entire population of United States white men living at the same ages and time periods. This excess was due almost entirely to cancer and respiratory disease. The cancer excess was chiefly due to respiratory cancer where mortality was 2·7 times the expected. The respiratory disease excess was entirely due to asbestosis.

A time-weighted measure of asbestos dust exposure at the time of retirement was calculated for each man. This was made up of the summed products of dust levels for each job (expressed in mppcf) and years at each level. This measure was directly related to the respiratory cancer excess at ages 65 and over, ranging from 1·7 times expected for men with less than 125 mppcf-years exposure to 5·6 times expected for men with 750 or more mppcf-years exposure. There appeared to be no direct relationship between asbestos dust exposure and respiratory cancer below 125 mppcf-years. Important increments in respiratory cancer mortality apparently occurred somewhere between 100 and 200 mppcf-years exposure.

Separation of the effects of time from the effects of average dust level on respiratory cancer mortality showed that the contribution of each was about the same and that a time-weighted measure of asbestos dust appears to be an appropriate method for predicting respiratory cancer effects.

There are relatively few studies on the dose-response relationship between asbestos dust and respiratory cancer. In a recent article McDonald *et al* (1971) reported on the incidence of respiratory cancer deaths among Canadian asbestos miners in relation to their cumulative asbestos dust exposure. That study suggests that a definite dose-response relationship exists and that below certain measurable levels of asbestos dust there appears to be little or no carcinogenic response. Three earlier studies also suggested a dose-response relationship. Knox, Holmes, Doll, and Hill (1968) found, in a preliminary report, that after dust controls were introduced in a British asbestos textile plant during the 1930s, the incidence of respiratory cancer fell from a 10-fold excess to a rate similar to that in the general population. Newhouse

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[1] A revision of a paper read on 9 February 1972 at the Annual Meeting of the American Academy of Occupational Medicine, Pittsburgh, Pennsylvania, U.S.A.

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(1969) reported that workers with heavy asbestos dust exposure had an excess of respiratory cancer, whereas those exposed to moderate or light doses had no excess. In a study which compared three segments of the American asbestos industry, Enterline and Kendrick (1967) reported that the estimated levels of asbestos dust related directly to variations in respiratory cancer deaths and to asbestosis deaths.

This is a report on the mortality experiences at ages 65 and over of a group of men who completed their working lifetime as production or maintenance employees in the asbestos industry, who retired with an industry pension, and who were all exposed to asbestos dust in varying degrees in the course of their employment. The average duration of employment in the asbestos industry was 25 years with a range from 3 to 51 years. Historic dust levels were sometimes in excess of 50 million particles per cubic foot of air. For each of these retirees it was possible, by combining personnel records with asbestos dust level estimates for each job and time period, to reconstruct their asbestos dust exposure history and, for each man, to assign a measure of his cumulative asbestos dust exposure at the time of retirement.

The cohort upon which this study is based consisted of all men retiring during the years 1941-67 and deaths occurring among these men through 1969. Such a study could be conducted almost entirely using industry records, except that where deaths occurred official copies of death certificates needed to be obtained. Included in this study were three kinds of retirees — those who retired normally at age 65; those who retired before age 65 for personal reasons but lived to age 65; and men who retired before age 65 because of a disability but who lived to be 65. The observed mortality among these men was compared with an expected mortality based upon the experience of the entire US white male population living in the time-age intervals that characterized the retired population. Inclusion of disabled retirees improves comparability with the US white male population as the latter also includes men unable to work for health reasons. The modified life table method was used for computing the expected number of deaths. This method is widely used and has been described in detail in an earlier report (Enterline, 1965).

There was a total of 1 376 men in this study who reached age 65. Of these, complete exposure and job histories were available for 1 348. Dust levels at each job site and time period were expressed as million particles per cubic foot of air (mppcf). It was not possible to estimate very precisely the dust level for each job, particularly for past periods, and dust levels could be only roughly classified. Jobs were placed in one of six classes or ranges, and for calculating total exposure mid-points of the range were used. Classes used were: no exposure (0); under 5 mppcf (2·5); 5-10 mppcf (7·5); 10-30 mppcf (20·0); 30-50 mppcf (40·0); 50 mppcf or more (62·5).

To compute total dust exposure for each man the dust level at each job and time period was multiplied by years at that job and summed across all jobs. This total cumulative exposure can be thought of as mppcf-years. Thus, if a man with a 30-year work history initially worked 10 years at a job with a dust level at that time of 20·0 mppcf, 10 years at a dust level of 2·5 mppcf, and 10 years at a dust level of 7·5 mppcf his total exposure would be: (10 × 20) + (10 × 2·5) + (10 × 7·5) or 300 mppcf-years. His average exposure level would be 10 mppcf.

Results

Table 1 shows observed and expected deaths and standardized mortality ratios (SMR) for selected causes of death for the entire cohort of 1 348 men for whom complete job and exposure data could be obtained. The expected value of the SMR, where observed and expected deaths is the same, is 100, and deviations from 100 are percentage deviations from expected mortality. As noted above, expected deaths were based upon the mortality experience of the entire US white male population living at the same ages and time periods as the cohort under study. Of the 754 deaths observed, death certificates could be located for 733. It is not known how much SMRs for individual causes of death might be changed were the remaining 21 death certificates available. It is unlikely, however, that there would be much of an increase for any single cause and no attempt has been made to allocate these 21 deaths to cause of death groupings. All death certificates were coded according to the Seventh Revision of the International

TABLE 1

OBSERVED AND EXPECTED DEATHS, AND SMR FOR SELECTED CAUSES OF DEATH, 1348 MALES RETIRING FROM THE ASBESTOS INDUSTRY 1941-67 AND FOLLOWED THROUGH 1969

Cause of death	*Observed*	*Expected*	*SMR*
All causes	754	657·2	114·7
Cancer (140-205)	167	109·4	152·6
Digestive (150-159)	53	41·8	126·8
Respiratory (162-163)	58	21·7	267·3
All other cancers	56	45·9	122·0
Strokes (330-334)	61	77·1	79·1
Heart disease (400-443)	326	306·6	106·3
Resp. disease (470-527)	67	36·8	182·1
Pneumoconiosis and pulmonary fibrosis (523-525)	29	—	—
All other causes	112	125·3	89·4
Death certificates not located	21	—	—

Classification of Diseases by a regular coder from the US National Center for Health Statistics. Numbers in parentheses in Table 1 correspond to the International Statistical Classification rubrics.

Table 1 shows that those men who retired from an industry with asbestos dust exposure had an overall mortality rate 14·7% higher than all US males. This excess is due almost entirely to cancer and disease of the respiratory system. For cancer, the greatest excess is in cancer of the respiratory system (ISC 162, 163) but with some excess also in cancer of the digestive system and other cancers. For respiratory disease the excess is due entirely to pneumoconiosis and pulmonary fibrosis. Eighteen of these deaths were due to asbestosis (ISC 523.2).

Data shown in Table 1 are very similar to data from other studies of asbestos workers which included men at ages under 65 (McDonald *et al.*, 1971; Enterline and Kendrick, 1967; Selikoff, Churg and Hammond, 1964; Mancuso and Coulter, 1963; Doll, 1955; Elwood, Cochrane, Benjamin, and Seys-Prosser, 1964). The excess in cancer, and particularly respiratory cancer and digestive cancer, and respiratory disease reported in other studies shows up quite clearly in this population of retired males. This is important since it could be argued that by age 65 men susceptible to an occupational hazard may already have been selected out of a cohort by death, and any effects of occupational exposure could not be detected.

In the analysis which follows only the relationship between asbestos dust exposure and respiratory cancer will be shown. Parallel analyses could, of course, be made for other causes of death.

Table 2 shows the relationship between total dose expressed as mppcf-years and mortality from respiratory cancer. For each dose interval actual means are shown in parentheses. It is quite clear that total dust exposure is highly related to respiratory cancer. If these data are plotted on arithmetic paper this relationship appears to be linear except at the highest dose level.

Of particular interest is the SMR of 166·7 for the 533 men with a total exposure of less than 125 mppcf-years. For a man with 25 years work in the asbestos industry this represents work, on average, at or below the old threshold limit value (TLV) of 5 mppcf. Table 3 divides the 533 men with a total mppcf-years under 125 into three total dust groupings to see if any relationship between dust and respiratory cancer exists within the under 125 mppcf-years' category. As in Table 2, actual means for each dose interval are shown in parentheses. No direct relationship between dust and respiratory cancer excess appears. In fact the relationship appears to be slightly inverse. Whether the SMR for

TABLE 2

Observed and Expected Deaths and SMR for Respiratory Cancer (ISC 162-163) by Total Dust Exposure, 1348 Men Retiring from the Asbestos Industry 1941-67 and Followed Through 1969

Total dust exposure (mppcf-yr)	*No. of men*	*Respiratory cancer deaths*		
		Observed	*Expected*	*SMR*
Under 125 (62·9)	533	15	9·0	166·7
125-249 (181·0)	305	12	4·8	250·0
250-499 (351·0)	328	17	5·2	326·9
500-749 (606·1)	126	9	1·8	500·0
750 and over (960·2)	56	5	0·9	555·6

TABLE 3

Observed and Expected Deaths and SMR for Respiratory Cancer (ISC 162-163), 533 Men with Total Dust Exposure below 125 mppcf-yr, Retiring from the Asbestos Industry 1941-67 and Followed Through 1969

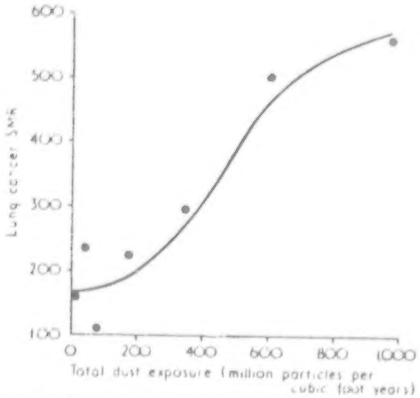
Total dust exposure (mppcf/yr)	*No. of men*	*Respiratory cancer deaths*		
		Observed	*Expected*	*SMR*
Under 25·0 (11·1)	75	2	1·3	153·8
25·0-62·4 (44·4)	181	8	3·1	258·1
62·5-124·9 (89·1)	277	5	4·6	108·7

espiratory cancer of 166·7 for men in the lowest xposure category is related to asbestos dust or to omething else cannot definitely be established from hese data. One of the factors that probably elevates omewhat this SMR is the fact that the men in this tudy were mainly urban dwellers and respiratory ancer death rates are normally higher in urban than n non-urban areas. Thus, since the expected number f deaths is based on the entire United States it may e too low. Correction for this for men with total sbestos dust exposures under 125 mppcf-years educes the SMR for this group of men to 146·2. ata published by Haenszel, Loveland and Sirken 1962) for the year 1958 were used to make this orrection.

Some additional help in evaluating the SMR of 166·7 for men with total dust exposures of under 125 mppcf-years also comes from a completely arallel study of 324 male retirees who worked as roduction or maintenance employees in the manuacture of a variety of building materials and were xposed to some dust, none of which contained sbestos or any substance suspected as being carcinoenic. Nine of these men died of respiratory cancer ISC 162, 163) as compared with 5·3 expected, giving n SMR of 169·8.

The Figure combines data from Tables 2 and 3 and stimates the relationship between total asbestos dust xposure and respiratory cancer. Superimposed on he Figure is a freehand drawing of cumulative ormal distribution—a distribution frequently used s a model for dose-response relationships.

If an SMR for cancer of the respiratory system of round 150 can be considered not excessive for men ho spent most of their working life as production and maintenance workers in an occupation with a fair amount of dust, then it would appear that the TLV for respiratory cancer expressed in terms of total mppcf-years might be somewhere between 100 and 200 mppcf-years.



IGURE. Total asbestos dust exposure and lung cancer mortality.

If the total dust estimate (mppcf-years) is separated into its two components, duration of exposure and average dust level, it is possible to test the assumption that time and dose should receive equal weight and are additive. This was done by constructing a 3 × 3 table with duration of exposure on one axis and average dust level on the other. For men in each of the nine cells in this table mean exposure years, mean exposure levels, and SMRs for respiratory cancer were calculated. From this it was possible to derive a linear multiple regression equation in which the SMR for respiratory cancer is the predicted or dependent variable. It is also possible to calculate correlation coefficients to see how well the combination of years worked and dust level predicts respiratory cancer excess, and how well each of these predicts respiratory cancer with the other held constant. These correlation coefficients are shown in Table 4.

TABLE 4

DEGREE OF RELATIONSHIP BETWEEN TIME, ASBESTOS DUST LEVEL, AND SMR FOR RESPIRATORY CANCER (ASSUMING A LINEAR MODEL)

Predicting variables	*Symbol*	*Correlation coefficient*	*Per cent variance explained (R^2 or r^2)*
Combination of time and level	$R_{1.23}$	0·850	0·722
Time at fixed level	$r_{12.3}$	0·738	0·545
Level at fixed time	$r_{13.2}$	0·772	0·596

Recalling that a correlation coefficient ranges from 0 for no predictability to 1 for perfect predictability, it is apparent, first, that a linear combination of duration and level of exposure is a good predictor of respiratory cancer excess and, second, that exposure time with dust level held constant predicts nearly as well as dust level with exposure time held constant. The meaning of exposure time in this context is uncertain. Whether the contribution of time to the incidence of respiratory cancer is primarily a reflection of increasing dose or a reflection of an increasing latent period would be difficult to establish, although it seems probable that both factors are involved.

An example of the use of the multiple regression equation is illustrated in Table 5. Here predicted respiratory cancer SMRs are shown for men working

TABLE 5

PREDICTED SMR FOR RESPIRATORY CANCER FOR MEN WORKING 25 YEARS AT SELECTED ASBESTOS DUST LEVELS (ASSUMING A LINEAR MODEL)

Dust level (mppcf)	*Predicted SMR (US expected)*	*Predicted SMR (US urban expected)*
5	195.3	171.3
10	257.8	226.2
15	320.4	281.0
20	383.0	335.9

25 years at various asbestos dust levels. SMRs are shown where the expected mortality is based on all US white males and where the expected is based only on white males living in urban areas.

Discussion

These data are similar to those reported by McDonald *et al.* (1971). In their study, both time and dust level appeared to make independent contributions to respiratory cancer increments. While direct comparisons of excess respiratory cancer are not possible, it is possible to compare the range of estimates in relation to the range of total dust exposure. In both studies, age-corrected respiratory cancer mortality rates at the highest level of total dust exposure are roughly three times those at the lowest.

The study presented here was relatively simple to conduct since all records needed, with the exception of death certificates, were available in industry. Estimates of dust levels for jobs as they existed during periods long past are obviously very crude and numbers were in many instances very small. Nevertheless the results appear to be coherent.

Had larger numbers been available it might hav been possible to identify more precisely a TLV f respiratory cancer in this type of population. Th available data suggest, however, that large incre ments in respiratory cancer appear at a cumulativ asbestos dust exposure of somewhere between 10 and 200 mppcf-years.

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LUNG CANCER AFTER EMPLOYMENT IN SHIPYARDS DURING WORLD WAR II

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Abstract A case-control study, undertaken to identify reasons for the exceptionally high rate of lung cancer among male residents of coastal Georgia, revealed a significantly increased risk associated with employment in area shipyards during World War II. The summary relative-risk estimate, adjusted for smoking, other occupations, age, race and county of residence was 1.6 (95 per cent confidence limits = 1.1 to 2.3). A synergistic relation was found between shipyard employment and cigarette smoking. These findings suggest that asbestos and possibly other shipyard exposures during wartime employment account for part of the excess mortality from lung cancer in certain coastal areas of the United States. (N Engl J Med 299:620-624, 1978)

THE mapping of cancer mortality in the United States according to county, 1950-69, revealed an area along the southeast Atlantic coast where the rates for lung cancer were exceptionally high among white men.[1,2] To identify reasons for the elevated mortality, we conducted a case-control study of lung cancer diagnosed among residents of coastal Georgia.

Methods

Cases of primary lung cancer among male residents of an 11-county coastal area* were identified from three sources: diagnoses since 1970 at the single large hospital in Brunswick, diagnoses during 1975-76 at the three major hospitals in Savannah, and death certificates for the period 1970-74 among residents of Chatham (Savannah) and the neighboring rural coastal counties where death was attributed to lung cancer (eighth revision of *International Classification of Diseases* 162.1).

Controls were selected from admissions to the four hospitals and from death certificates for diagnoses other than lung cancer, bladder cancer or chronic lung disease. The hospital controls were nearest admissions at the same facilities of similar sex, race, age (±2 years), county of residence and current vital status. Two controls were chosen for each case in Brunswick (where the number of cases was smaller), and one for each case in Savannah. A single death-certificate control was selected for lung-cancer cases identified by death certificate according to similar matching criteria.

Personal interviews of the patients and controls, or their next of kin in the event that they had died, were conducted by locally hired public-health nurses and interviewers under the supervision of a professional survey organization. Each interviewer was trained in interviewing technics, including standardized methods of probing. Nearly all questions were in closed form to minimize inter-observer biases. Sample questions in 10 per cent of all interviews were later checked for verification by telephone.

Information was obtained on place, type and length of employment for all jobs held for six months or more. The respondents were asked to describe the duties performed for each job, as well as to list materials handled during the course of their work. The occupational section of each questionnaire was concluded by review with each respondent of a checklist of industries (including shipbuilding) and materials (including asbestos). Data obtained on cigarette-smoking characteristics for each respondent included, for those who had at some time smoked — "ever smokers" — the age they started, usual amount smoked, age (or ages) when they had stopped smoking and type of cigarette.

Estimates of the relative risk (odds ratios) of "ever" (six months or more) employment upon lung cancer were calculated for each of 16 industrial categories, including shipbuilding.[3] The relative risk estimates were determined for various strata categorized according to race (white or black), age at diagnosis or death (30-54, 55-64 and 65+), county of residence (Chatham, Glynn, and other), "usual" industry of employment (16 categories) and cigarette smoking (three categories). We calculated tests for interactions of the relative risks for shipbuilding across the various levels of the other factors, using a logistic representation of these retrospective data.[4] When effect modification was absent, summary relative risks were estimated and tested by this technic,[4] as well as by the Mantel-Haenszel method.[5]

To obtain an estimate of the possible effect of inaccuracies of next-of-kin responses, we conducted simultaneous interviews, usually of the husband and wife, for a small sample (24 pairs) of the study subjects and their next of kin.

Results

A total of 535 cases of primary lung cancer and 659 controls were identified from the Brunswick and Savannah hospitals and the Chatham and rural-county mortality series (Table 1). This distinction excludes 23 Savannah hospital cases in residents of smaller coastal counties, since appropriate hospital controls matched for county of residence for the group could not be easily obtained.

Interviews were completed for 89 per cent of the lung-cancer cases and 87 per cent of the controls. Of those not interviewed, about two thirds had moved out of the coastal area or could not be located; for the remainder, either the patient or next of kin (32 cases) or attending physician (14 cases) would not consent to interview. Approximately 4 per cent of the subjects interviewed had lived in coastal Georgia for less than five years and were excluded from the analysis. Thus, the study group consisted of 458 lung-cancer cases and 553 controls, most of whom were longtime residents (median of 41 years) of coastal Georgia.

The major findings of this study pertained to shipyard employment. Shipbuilding was the "usual" industry in eight cases (mostly workers in smaller shipyards) and one control (crude relative risk of 9.8). However, 95 patients and 80 controls (crude relative risk of 1.5) were employed at some time in the industry, most working for just a few years during World War II (only 21 per cent of the total worked in the in-

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*Included were Brantley, Bryan, Camden, Charlton, Chatham, Glynn, Liberty, Long, McIntosh, Pierce and Wayne counties. The participating hospitals were Glynn-Brunswick Memorial Hospital, Candler General Hospital, St. Joseph's Hospital and Memorial Medical Center.

dustry for more than five years). As shown in Table 2, the risk ratios were greater than 1 regardless of the method of ascertainment for the series comprising this investigation.

The higher risk associated with shipbuilding was not accounted for by cigarette smoking. Table 3 shows elevated relative risks for shipbuilding in each of three cigarette-smoking categories. The first smoking category consisted of those who had never smoked, those who usually smoked less than half a package per day (whose risk relative to that of never smokers was 0.4) and workers who had stopped smoking at least 10 years before diagnosis (risk of 1.8), the second group included smokers who usually smoked half a package to 1½ packages per day (risk of 4.5), in addition to those (13 per cent of the total) whose usual amount smoked was unknown (risk of 6.2), and third, those who usually smoked two or more packages per day (risk of 8.6). The summary relative risk for shipbuilding, adjusted for cigarette smoking by the Mantel-Haenszel method, was 1.6 (P = 0.006).

Table 1. Numbers of Lung-Cancer Cases and Controls Identified for Study by Method of Ascertainment

Source of Identification	Period	Cases	Controls
Brunswick Hospital	1970-76	170	319
Savannah hospitals	1975-76	149	153
Death certificate	1970-74	216	187
Totals		535	659

The higher estimates of relative risk for shipbuilding were seen in both whites and blacks, in all age categories and in Glynn (Brunswick), Chatham (Savannah) and the rural coastal counties as well (Table 4). The higher risks for shipbuilding were also not readily explicable by other occupations. Table 5 shows that the distribution of the "usual" industries of employment of those who had worked in shipyards included a higher proportion of construction and farming jobs and a lower proportion in the paper and pulp industry than the distribution among those who had never worked in the shipbuilding industry. The relative risks for farming and the paper industry, adjusted for smoking, were 0.7 and 1.0, so that the differential representation in these two industries does not account for the increase associated with shipbuilding. There was a smoking-adjusted increased risk of 1.3 associated with construction, but the risk with shipbuilding was high among both construction and nonconstruction workers (Table 6).

When smoking, residence, age, race and usual industry were simultaneously taken into account in the logistic model, the adjusted summary relative risk was 1.6 (P = 0.01), with 95 per cent confidence limits of 1.1 to 2.3. Although the relative risks for shipbuilding increased with heavier consumption of tobacco, the differences in these risks among the three smoking categories were not statistically significant (P>0.10).

Table 2. Relative Risks for Shipbuilding According to Method of Ascertainment and County of Residence, Cases and Controls

Series	County of Residence	Case or Control	Ever Employed in Shipbuilding		Relative Risk
			yes	no	
Brunswick Hospital	Glynn	Case	19	62	1.7
		Control	23	130	
	Other rural	Case	10	45	1.3
		Control	18	101	
Savannah Hospital	Chatham	Case	23	111	1.4
		Control	17	116	
Mortality	Chatham	Case	32	91	2.0
		Control	14	79	
	Other rural	Case	11	54	1.2
		Control	8	47	

Nevertheless, the excess risk increased sharply with smoking (Fig. 1), indicative of a strong synergism between these two risk factors.

Table 7 shows relative-risk estimates according to types of shipyard jobs. An excess risk was not seen for welders or riggers, but was apparent for other groups, including those where direct exposure to asbestos would not be likely. Each interview requested information on whether asbestos was among the "materials handled at work." Among the 175 respondents in our study who had worked in shipbuilding, contact with asbestos was reported by only 10 (four cases and six controls).

Information obtained on the specific histologic types of primary lung cancer among the hospital series revealed no differences among those ever employed in the shipbuilding industry as compared to those never so employed.

The small sample evaluation of comparability of patient and next-of-kin responses revealed little difference in "usual" occupation according to type of respondent. The patients, however, tended to report a greater number of jobs ever-held (particularly in farming, although not in shipbuilding) than their next

Table 3. Relative Risks for Shipbuilding According to Cigarette-Smoking Category

Smoking Category*	Case or Control	Ever Employed in Shipbuilding		Crude Relative Risk
		yes	no	
Nonsmoker, light smoker or stopped smoking	Case	11	50	1.3
	Control	35	203	
Moderate	Case	70	217	1.7
	Control	42	220	
Heavy	Case	14	96	2.4
	Control	3	50	

*See text for definition of categories.

of kin. Concordance for smoking category was 83 per cent, with the discrepancies split between higher and lower consumption reported by the next of kin, in each instance with the classification by the study subject and next of kin being in adjacent categories. Most (76 per cent) of the interviews for the entire sample of 458 cases and 553 controls were with next of kin. When the total data were stratified according to type of respondent, the risk ratios for shipbuilding were 1.5 for self-respondents and 1.6 for the next-of-kin respondents.

DISCUSSION

Because of excellent community participation (less than 3 per cent of those approached refused interview), it was possible to cover nearly all the identified subjects in this case-control investigation. However, since lung cancer is often rapidly fatal — nationally the median survival is five months[9] — most of the interviews were conducted with the next of kin of deceased persons. Our sample check for comparability revealed generally good agreement between self and next-of-kin responses, but some inaccuracies may have resulted from second-party reporting, or from the difficulties in recalling events taking place 30 or more years before the interview. However, these possibilities seem unlikely to have produced any major differences between cases and controls.

Table 4. Relative Risks for Shipbuilding According to Race, Age and Residence

Indicator	Case or Control	Ever Employed in Shipbuilding		Crude Relative Risk	Smoking-Adjusted Relative Risk
		Yes	No		
Race					
White	Case	82	315	1.5	1.5
	Control	67	381		
Black	Case	13	48	1.9	2.0
	Control	13	92		
Age (yr)					
<55	Case	13	73	1.8	1.8
	Control	10	102		
55-64	Case	34	124	1.4	1.5
	Control	29	150		
65+	Case	48	166	1.6	1.6
	Control	41	221		
Residence					
Chatham	Case	55	202	1.7	1.6
	Control	31	195		
Glynn	Case	19	62	1.7	2.1
	Control	23	130		
Other	Case	21	99	1.2	1.5
	Control	26	148		

A significantly higher risk of lung cancer was found in Georgia residents who worked in shipyards during World War II. The higher risk, which was observed in each of five independent series of lung-cancer cases and controls in this investigation, was not readily explicable by smoking habits or other occupational exposure. The excess was seen in white and black men in both Savannah and Brunswick, where two large shipyards began operations in 1942. By late 1943 about 22,000 were employed in the Savannah shipyard, and about 16,000 in Brunswick.[7,8] Both yards closed shortly after the war's end. Twenty-one per cent of the patients with lung cancer in our study reported working in the industry. Among the small number of men who continued to work in shipbuilding and repair at other locations, the risk of lung cancer was exceptionally high. It seems likely, therefore, that shipyard exposures account for at least part of the excess mortality from lung cancer in coastal Georgia. Assuming that 14 per cent (the proportion among controls) of the general population in the area had worked in the shipyards and were at 1.6 times the "usual" risk of lung cancer, and assuming that the remaining 86 per cent were at usual risk, the coastal lung-cancer rate would be expected to exceed the usual rate by about 8 per cent. Provisional estimates of mortality rates from lung cancer for 1970-75 among white men show that the rates in the area

Table 5. Percentage Distribution of Usual Industry of Employment among Those Ever Employed in Shipbuilding as Compared to Those Never Employed in This Industry

Usual Industry	Ever Employed in Shipbuilding	
	Yes	No
Agriculture	14.3	9.2
Fishing	0	2.2
Forestry	1.1	1.8
Construction	16.6	8.4
Lumber-wood	2.3	4.4
Metal machinery	2.9	2.8
Shipbuilding	5.1	0
Paper pulp	1.7	7.8
Chemicals or other nondurable-goods manufacture	2.9	5.5
Transportation	18.3	15.7
Wholesale & retail	9.1	11.7
Personal service	4.0	2.9
Professional & government	8.6	11.1
Military	0.6	3.3
Not elsewhere classified	4.6	5.3
Unknown	8.0	8.0
Totals	100.1	100.1

Table 6. Relative Risks for Ever Employment in the Shipbuilding Industry According to Usual Employment in the Construction Industry

Usual Industry Construction	Case or Control	Ever Employed in Shipbuilding		Crude Relative Risk	Smoking-Adjusted Relative Risk
		Yes	No		
No	Case	77	331	1.5	1.6
	Control	69	435		
Yes	Case	18	32	1.9	2.0
	Control	11	38		

exceed the national rate by about 30 per cent, suggesting that the coastal excess may be only partly attributable to wartime shipyard exposures. However, it is possible that the maximum effect of exposures during World War II occurred before the 1970-76 period of investigation, so that the risks presented in this study may be underestimates.

Ship construction and repair involves contact with asbestos,[9,10] which is known to induce lung cancer and mesothelioma, but few data on this exposure are available from our survey. Only 5 per cent of those ever employed in the shipyards reported handling asbestos during shipyard duties, with only one classified as an insulator. The lung-cancer risk did not seem limited to any particular shipyard trade, in agreement with reports from Europe of mesothelioma among various classes of shipyard workers.[10-16] Asbestos-related lung disease has been seen in pipecoverers in shipyards in the United States[17] and also among other workers regardless of their occupation within the industry.[18] Furthermore, the multiplicative effect between shipyard employment and cigarette smoking in our study is consistent with other reports of a synergism between exposure to asbestos and smoking on lung-cancer risk.[19]

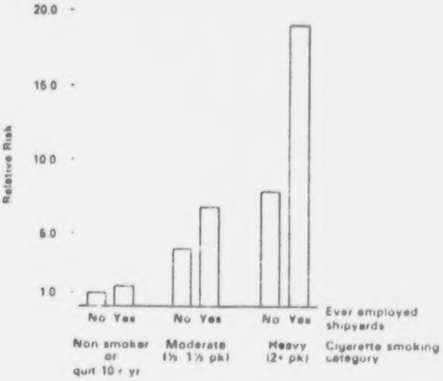

Figure 1. Relative Risk of Lung Cancer According to Usual Cigarette Smoking Category and Employment in Shipyards during World War II.

Despite the increased mortality from lung cancer in coastal Georgia, we found only five cases with mesothelioma and two with asbestosis through a search of medical records and pathology files since 1960 in the participating hospitals. This number of cases of mesothelioma was not higher than the national incidence estimated at about two cases per million per year,[20] but diagnostic ascertainment in the area may be incomplete. On the other hand, since mesotheliomas have been reported to occur even after relatively light exposures to asbestos,[21,22] the failure to see greater numbers of this rare tumor among coastal residents raises the possibility of shipyard hazards in addition to asbestos.

Table 7. Relative Risks for Ever Employment in Major Shipbuilding Job Subcategories

SUBCATEGORY	CASES*	CONTROLS*	CRUDE RELATIVE RISK
Insulator, boiler maker	2	2	1.2
Pipefitter	10	4	3.1
Shipfitter, steamfitter	6	5	1.5
Welder, burner	11	20	0.7
Rigger, leaderman	6	9	0.8
Machinist, machine operator, metal worker	13	11	1.4
Laborer, construction	24	20	1.5
Electrician	5	3	2.0
Clerk, accountant, draftsman, guard	7	3	2.8
Not specified	15	5	3.7

*Total no. slightly exceeds no. ever employed in the shipbuilding industry since persons with jobs in ≥2 categories were counted in each.

Further studies of shipyard workers should help quantify the risk of lung cancer, mesothelioma and other tumors seen after exposure to asbestos.[23] The magnitude of the problem may be greater than has previously been thought, since counties throughout the United States with wartime shipyards tend to have elevated mortality not only from lung cancer but also from cancers of the larynx, oropharynx and upper gastrointestinal tract.[24]

We are indebted to the study members, their physicians and the staff of the Glynn-Brunswick Memorial Hospital, Candler General Hospital, St. Joseph's Hospital, and Memorial Medical Center for co-operation; to Drs. W. A. Rasmussen and W. D. Lundquist for advice and support; Dr. G. A. Dever and Mr. T. W. McKinley for access to death certificates; and Mr. J. Escatell, Ms. K. Hall, Dr. B. J. Stone, Ms. L. Morris and Mr. J. Housworth for computer assistance.

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EFFICACY OF CHEST PHYSIOTHERAPY AND INTERMITTENT POSITIVE-PRESSURE BREATHING IN THE RESOLUTION OF PNEUMONIA

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Abstract We undertook a randomized clinical trial to evaluate the efficacy of chest physiotherapy and intermittent positive-pressure breathing in the treatment of pneumonia. The diagnosis of pneumonia required a compatible clinical history and x-ray confirmation. A total of 54 patients were assigned to treatment and control groups and were similar in age, smoking history, underlying lung disease and prior antibiotic treatment. Antibiotic therapy, guided by Gram stain and sputum and blood cultures, was similar in both groups. Chest physiotherapy, consisting of postural drainage, percussion and vibration, was given concurrently with intermittent positive-pressure breathing with use of racemic epinephrine every four hours. There was no statistically significant difference in duration of fever, extent of radiographic clearing, duration of hospital stay and mortality between the control and treated groups. Chest physiotherapy and intermittent positive-pressure breathing do not hasten the resolution of pneumonia. (N Engl J Med 299:624-627, 1978)

A RECENT symposium[1] held under the auspices of the American Thoracic Society evaluated the scientific basis of respiratory therapy, including chest physiotherapy and intermittent positive-pressure breathing. These therapies, now extensively used in diverse bronchopulmonary disorders, have not often been validated by randomized clinical studies, or such studies have failed to show clinical benefit.[2-5] One application not previously studied is their efficacy in the treatment of pneumonia. Despite the lack of data concerning effectiveness in this common clinical situation, an in-house retrospective study of 100 consecutive patients hospitalized at the Medical Center Hospital of Vermont with pneumonia revealed that chest physiotherapy and intermittent positive-pressure breathing were used as therapeutic adjuncts in two thirds of the cases. Furthermore, a survey carried out on the treatment of pneumonia in four regional hospitals in Vermont showed that as many as 64 per cent of patients received intermittent positive-pressure breathing, and 44 per cent received chest physiotherapy — a frequency of use estimated to occur nationwide (Baker JP: personal communication). To assess the effects of this form of therapy in hastening the resolution of pneumonia, we conducted a randomized prospective clinical trial.

Methods

Patients admitted to a pulmonary unit with a radiographic diagnosis of pneumonia and a compatible clinical history of fever and increased cough were eligible for randomization. Informed consent was required. Patients were excluded from the study by any of the following complicating factors: lung abscess, established underlying bronchiectasis, tuberculous pneumonia or cystic fibrosis, respiratory failure (partial pressure of carbon dioxide over 50 mm Hg) or a need for assisted ventilation, presence of renal failure (blood urea nitrogen over 40 mg per deciliter), immunosuppressive therapy (receiving more than 40 mg per day or equivalent of prednisone, azathioprine or antineoplastic agents), stroke within six weeks, and underlying cancers.

Patients with other underlying cardiopulmonary disorders such as heart disease or chronic obstructive lung disease were included, as were those with medical conditions such as alcoholism and diabetes. Postoperative patients were not studied.

The randomization procedure pre-stratified patients on the basis of age (under or over 55 years), smoking history (smoker vs. nonsmoker), sex and prior antibiotic therapy. The house officer looking after the patient then selected a pre-randomized sealed envelope containing a control or treatment group label, which assigned the patient.

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Dust exposure and mortality in chrysotile mining, 1910-75

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ABSTRACT We report a further follow-up of a birth cohort of 11 379 workers exposed to chrysotile. The cohort consisted of all 10 939 men and 440 women, born 1891-1920, who had worked for at least a month in the mines and mills of Asbestos and Thetford Mines in Quebec. For all subjects, length of service and estimates of accumulated dust exposure were obtained, with a smoking history for the vast majority. Three methods of analysis, two based on the "man-years" method, the other a "case-and multiple-controls" approach, gave results consistent with one another and with previous analyses.

By the end of 1975, 4463 men and 84 women had died. Among men, the overall excess mortality, 1926-75, was 2% at Asbestos and 10% at Thetford Mines, much the dustier region. The women, mostly employed at Asbestos, had a standardised mortality ratio (SMR) (all causes, 1936-75) of 0·90. Analysis of deaths 20 years or more after first employment showed that in men with short service (less than five years) there was no discernible correlation with dust exposure. Among men employed at least 20 years, there were clear excesses in those exposed to the heaviest dust concentrations. Reanalysis in terms of exposure to age 45 showed definite and consistent trends for SMRs for total mortality, for lung cancer, and for pneumoconiosis to be higher the heavier the exposure. The response to increasing dose was effectively linear for lung cancer and for pneumoconiosis. Lung cancer deaths occurred in non-smokers, and showed a greater increase of incidence with increasing exposure than did lung cancer in smokers, but there was insufficient evidence to distinguish between multiplicative and additive risk models. There were no excess deaths from laryngeal cancer, but a clear association with smoking. Ten men and one woman died from pleural mesothelioma.

If the only subjects studied had been the 1904 men with at least 20 years' employment in the lower dust concentrations, averaging 6·6 million particles per cubic foot (or about 20 fibres/cc), excess mortality would not have been considered statistically significant, except for pneumoconiosis. The inability of such a large epidemiological survey to detect increased risk at what, today, are considered unacceptable dust concentrations, and the consequent importance of exposure response models are therefore emphasised.

Mining, milling, and processing of the asbestos group of fibrous minerals, long known for their remarkable strength and fire resistance, began on a commercial scale at the end of the nineteenth century. In the Eastern Townships region of Quebec deposits of chrysotile were noted in the 1847 Canadian Geological Survey. Exploitation began at Thetford in 1878 and four years later some 60 miles away near Danville. Within 30 years the region was producing most of the world's asbestos. The proportion fell as Russian, South African, and Italian mines came into operation, but Quebec still produces about 25% of the world's supply, now estimated at about six million tons a year; indeed, production in Quebec and elsewhere has continued to expand.

In and around Thetford many companies started mining, and the town was renamed Thetford Mines.

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Amalgamations and closures over the years reduced the number of companies to six. One large company has operated near Danville, and the new town of Asbestos was built; this company has also manufactured asbestos products in a small local factory. There was formerly a small mine a few miles away, owned by another company, which is included with the companies of the Thetford area for the purposes of this paper.

Attention drawn at the New York Academy of Sciences in 1964 to the serious threats to health posed by airborne asbestos dust led immediately to recommendations[1] that epidemiological inquiries were urgently needed in populations exposed to single types of fibres. At the invitation of the Canadian Government, and with encouragement from the Government of Quebec, a research programme was established in 1966 at McGill University to study the health effects of work in the Quebec chrysotile-producing industry. Results of the first three years' work, which included the reading of 16 000 chest radiographs, comprehensive tests on a sample of current workers, and studies of mortality to the end of 1966 were summarised by McDonald *et al* in 1974.[2]

Our knowledge of the mortality experienced by chrysotile workers is derived from observations on a birth cohort (1891-1920) of 10 939 men and 440 women followed from first employment (the earliest in 1904) to the end of 1975, by which time 4463 men and 84 women were known to have died. The first death was due to an accident in 1910, and another 112 men died before the end of 1925, mainly through accidents or respiratory tuberculosis. During the five decades, 1926-75, 4350 men died compared with 4107 expected on the basis of Quebec age- and year-specific death rates, a standardised mortality ratio (SMR) of 1·06, but the evolution of mortality in the cohort differed substantially in the two mining areas.[3] Table 1 shows that there were almost equal numbers of employees in the two mining areas, Asbestos and Thetford Mines. The numbers in each of the six quinquennia of birth were similar, varying only between 1509 and 1969. The first excesses which were during the war years, were about equal in the two areas, and may have been attributable, at least in part, to the exclusion, until 1944, from the Quebec vital statistics of deaths in military personnel outside Canada. In the decade 1946-55 there were 42 fewer deaths than expected at Asbestos but 16 more than expected at Thetford Mines. From 1956 to 1970 there were excesses in both areas, but considerably less at Asbestos (78) than at Thetford Mines (173). In the final period, 1971-5, there were 18 fewer deaths than expected at Asbestos, but still an excess of 12 at Thetford Mines. During the five decades, 1926-75, there had been a net excess of 33·9 deaths at Asbestos (1·6% of the 2074·1 expected) and of 208·8 at Thetford Mines (10·3% of 2033·2) – that is, SMRs of 1·02 and 1·10 respectively.

The primary aim of the mortality survey was "to define as accurately as possible the quantitative relationship between exposure to chrysotile asbestos and the incidence of lung cancer."[4] The present paper reports the results of the follow-up until the end of 1975. Mortality from lung cancer is associated with smoking, and the concern must be to attempt to separate the hazards. It is also important to consider total mortality, if only to confirm that an excess of deaths from lung cancer does not blind us to a shortfall of greater magnitude from other causes, as has sometimes occurred in other surveys. Additional aims were to measure exposure-response relations for other malignant diseases, particularly cancers of the gastrointestinal tract and larynx, and for pneumoconiosis and other respiratory diseases.

Material and methods

In November 1966 a register was compiled of everyone, nearly 30 000, ever employed in the Quebec chrysotile production industry. A small proportion, who worked at the factory in Asbestos, were also included because of the movement of employees between it and the mine or mills. Everyone born between 1891 and 1920 inclusive and employed for one month or more was identified from the register. These criteria for admission to the cohort were chosen to ensure a wide range in levels of exposure and durations of employment, while those born before 1891 would have been particularly difficult to trace and those born after 1920 have yet to reach the age of high mortality. All available means were adopted in attempts to trace every person in the cohort[3]; there was no appropriate social security system to facilitate this. For all deaths, exact dates and places were sought so that copies of death certificates could be obtained. The cause of death was coded according to the International Classification of Diseases (7th revision) by a person who regularly performed this task for the Quebec Department of Vital Statistics.

Tracing has been carried out to four points in time: 31 October 1966,[3] to the end of 1969,[2,5] to the end of 1973,[6] and now to the end of 1975. The proportion traced has slightly improved over the years; most of the 9·9% still lost to view were men with short service many years ago; less than 2% were untraced after 1935. The total cohort now numbers 11 379 (10 939 men and 440 women) whereas it was originally estimated[1] as 11 788. The reduction has been due to improving information which permitted (a) more stringent application of admission criteria and (b) better recognition of cases in which the same employee had been separately registered with different companies or under different names. In the nine years since our first report the number of deaths has increased from 2457 to 4547, now comprising 44·3% of those traced. By the end of 1975 the youngest people alive in the cohort were 55 years old, the oldest 84.

Much work was put into determining the dust exposure for each subject, as described by Gibbs and Lachance.[7] In essence, some 5783 jobs were identified from detailed work histories, and for each one, for each applicable year, an average dust concentration was estimated in millions of particles per cubic foot (mpcf) from available dust measurements and approximations. Estimates were based on well over 4000 midget impinger dust counts measured, between 1949 and 1966, by Maurice Lachance, ingenieur, in annual surveys in all companies. Before 1949 there had been few measurements, so estimates were made by Gibbs and Lachance after interviews with long-service employees and comparisons with more recent conditions.

For each person in the cohort, we defined his or her

Gross service—The period from start of first job to termination of last job (or to November 1966, if earlier).

Net service—The total of periods of actual employment.

Service gap—(Gross service less net service)/(gross service), as per cent.

Accumulated dust exposure—The total of annual dust exposures, summed over defined periods; the units are mpcf multiplied by years (mpcf y).

Smoking habit was assessed by a questionnaire, answered by 99·6% of those alive in 1970 and by the relatives or friends of 93% of those who died after 1950; for earlier deaths, numbering 816, it was thought that smoking histories would often not be obtainable or sufficiently reliable. Each man has been classified according to his reported smoking habit at the time of report as non-smoker, moderate smoker, or heavy smoker—that is, one who smoked 26-50 cigarettes a day.

The data to 1966 and those to 1969 were analysed primarily by a method which was recognised[4] as having two weaknesses: (1) that deaths accumulated over many years were used without regard for age at death, and (2) that length of exposure was related to length of survival. These problems were covered in part by subsidiary analyses included in our first two reports[1,3] and in a more satisfactory but preliminary way in a third more comprehensive paper.[2] Although it was appreciated that an analysis based on man-years of exposure with comparison against an external reference population[8] was the orthodox approach to the problem, this too had its weaknesses. After a period of exploration and consultation,[9] the 1973 mortality data were used for an appraisal of methods of cohort analysis and presented for discussion at a general meeting of the Royal Statistical Society.[9] The merits and practical usefulness of the man-years method (which reasons a priori—that is, from cause to effect) were confirmed, but, clearly, an approach using a small fixed number of controls for each death,[10] and arguing a posteriori (from effect to cause), had other advantages. In particular it is efficient for examining interaction between several stimulus variables—for example, smoking, dust concentration, and duration of employment—and it does not depend on the availability of death rates for a reference population. Although rates for Quebec have been available since 1926, the appropriateness of this or any other reference population has always to be questioned. In this latest report we have used both methods; in addition we have made use of certain findings from the detailed analyses of deaths to the end of 1973 by Liddell *et al.*[11]

A PRIORI ANALYSES

For the man-years method, and to avoid possibly misleading non-standard terminology, we have continued to use the following definitions.

Subcohort—A division of the cohort, according to some factor(s) such as years of exposure or smoking habit.

Study interval—The interval over which each subject is studied in a particular facet of the study—which is not necessarily the same as the period over which the subject has been in view.

There are several aims, mainly conflicting, in the definition of subcohorts: they should be mutually exclusive and comprehensive, fairly equal in size, and large enough to provide reliable measures of mortality yet small enough to be homogeneous, preferably with at last three classes of each factor of interest in the relations of dose to response. There are usually several such factors, however, and their interactions may be important, so that many subcohorts are necessary. The compromises adopted are described below.

For a first series of analyses, the male cohort was subdivided initially by length of gross service, the boundaries being taken at one year, five years, and 20 years; thereafter, each gross service group was divided into four more-or-less equal classes according to accumulated dust exposure. For those with very short service (less than one year), the three margins between the dust groups were drawn at 1, 3, and 10 mpcf y; for those with short service (one to five years), the margins were at 6, 30, and 100 mpcf y; and for medium service (five to 20 years), at 30, 100, and 300 mpcf y. For the long-service group (at least 20 years), the classification, with boundaries at 100, 300, and 600 mpcf y, was made in terms of dust exposure accumulated in the first 20 years from start of service. The study interval for this first series of analyses started, in all four service groups, at 20 years after first employment in the industry, thus excluding only 21 deaths due to asbestos-related disease that had occurred within the first 20 years.

Relative risks of lung cancer were considered in detail by Liddell *et al*,[6] and it appeared that there was little to suggest that the way in which dust exposure had been accumulated played any part in determining the risk. It therefore seemed appropriate to base a second series of analyses on dust exposure accumulated to a certain age, arbitrarily taken as 45 years, at which age most men had completed their service. After the cohort had been divided by exposure to age 45, two further, but separate, subdivisions were made by mining area (Asbestos and Thetford Mines) and by smoking habit; those whose smoking habit was unknown being added to the largest group—that is, moderate smokers. The study interval started at age 45.

From data for Quebec obtained from Statistics Canada, we assembled mid-year populations and deaths from all causes in five-year age-groups (15-84) for men for the years 1926 through 1974, and for women for the years from 1936, the earliest practicable year, to 1974; the material for the last year was duplicated to provide estimates for 1975. Statistics Canada also provided magnetic tapes containing age, sex, and cause of all Quebec deaths, 1951-73, coded to the International Classification of Diseases for the 12 specific groups of causes defined in table 2. The numbers of deaths for "other known causes" were obtained by subtraction. Annual death rates for the reference population were calculated for five-year age-groups, 15-84, from the mid-year populations, 1951-75; those for 1973 were replicated twice to give estimates for 1974 and 1975.

A POSTERIORI ANALYSIS

The Miettinen method[10] uses a fixed number of controls for each case of interest. Our selection of internal controls was strictly at random from among those miners known to have survived to a greater age than that at which the case died and to have been born in (or as close as possible to) the same year as the case. Each control came from the same mining area as the case; for some purposes, one control was matched for smoking habit with the case. Those of unknown or doubtful habit were classified as "undifferentiated" and three groups of definite smokers were considered in the matching—that is, those smoking 15-25, 26-35, and 36-50 cigarettes a day. An attempt was made to match precisely into the subgroups, but for definite smokers relaxation was not infrequently necessary, and indeed in a few cases the control had to be found from the "undifferentiated" class.

The causes of death studied in this way, with the numbers of controls selected with and without regard for smoking were as follows: pneumoconiosis (1, 3); lung cancer (1, 3); abdominal cancers (0, 2); laryngeal cancers (1, 3); and mesothelioma (1, 4). For these analyses, the dust exposure was calculated up to nine years before the death of each case and to the same age for his controls.

Results

MEN

Just under half the men from each area were known to be alive in 1976, but one-tenth had been lost to view (table 1). Most of these losses were before 1936; in a review of the situation when tracing had been completed to the end of 1973 it was found that of 9723 men known to have been alive in 1936, 5797 had been traced alive, 3750 were known to have died, and only 176 were untraced (59.6%, 38.6%, and 1.8%, respectively).

Table 1 *Constitution of male cohort at end of 1975 (Percentages in parentheses)*

	Asbestos	*Thetford Mines*	*Composite cohort*
Alive	2608 (49.2)	2779 (49.3)	5387 (49.2)
Dead	2179 (41.1)	2284 (40.5)	4463 (40.8)
Lost to view	515 (9.7)	574 (10.2)	1089 (10.0)
Total	5302 (100)	5637 (100)	10939 (100)

The 4463 male deaths are shown in table 2 by year, age, and certified cause. Deaths from pneumoconiosis and lung cancer were generally at ages over 45 and from 1956 onwards. Respiratory tuberculosis was a major cause of death until the early 1950s, while accidents (including, but not dominated by, accidents at work) have been a common cause of death throughout, particularly in those under 45. In a total of 125 deaths (2.8% of all deaths), cause could not be ascertained, partly because death registration was not universal in Quebec until 1926, and partly because some death certificates were not found.

The number of death certificates on which it was stated that a necropsy had been carried out was 789

Table 2 *Deaths of men, by year, age, and certified cause of death*

Cause of death (ICD code)*	*Age at death*	*Year of death* *Before 1946*	*1946-55*	*1956-65*	*1966-75*	*Total*	
All causes	<45	564	136	54		754	4463
	45-64	111	418	842	702	2093	
	≥65	—		389	1227	1616	
Pneumoconiosis (523-524)	<45	0	0	1		1	46
	45-64	1	6	10	11	30	
	≥65	—	—	7	8	15	
Malignant neoplasms:							
Lung (162-164)	<45	2	2	2		6	250
	45-64	0	12	51	72	135	
	≥65	—		20	89	109	
Oesophagus and stomach (150-151)	<45	5	2	1		8	154
	45-64	4	22	34	17	77	
	≥65	—		12	57	69	
Colon and rectum (152-154)	<45	4	1	0		5	88
	45-64	1	8	20	18	47	
	≥65	—		6	30	36	
Other abdominal (155-159)	<45	5	2	1		8	80
	45-64	1	6	15	14	36	
	≥65	—		6	30	36	
Larynx (161)	<45	0	0	0		0	21
	45-64	2	5	6	5	18	
	≥65	—		1	2	3	
Other (140-148, 160, 165-205)	<45	12	4	1		17	276
	45-64	2	28	52	48	130	
	≥65			28	101	129	
Heart disease (400-443)	<45	28	25	18		71	1543
	45-64	25	154	355	285	819	
	≥65			166	487	653	
Respiratory tuberculosis (001-008)	<45	118	30	1	—	149	248
	45-64	20	11	27	7	65	
	≥65			5	9	14	
Other respiratory (470-522, 525-527)	<45	60	3	0	—	63	234
	45-64	5	12	28	37	82	
	≥65			17	72	89	
Cerebrovascular (330-334)	<45	6	2	3		11	268
	45-64	4	12	42	38	96	
	≥65			39	122	161	
Accidents (800-999)	<45	170	41	17		228	461
	45-64	18	44	71	51	184	
	≥65	—		9	40	49	
All other known causes	<45	112	23	1		136	659
	45-64	25	82	112	82	301	
	≥65			67	155	222	
Cause not known	<45	40	1	0		41	125
	45-64	1	16	19	15	53	
	≥65			6	25	31	

*Code in the 7th revision of the International Classification of Diseases

(17·7% of all deaths), and necropsy reports were obtained for the majority. These showed nine extra cases of lung cancer, but to maintain comparability with the general population only the coded causes of death were used throughout. Ten male deaths due to pleural mesothelioma were identified, nine from death certificates and one at necropsy. Six of these had been coded to lung cancer (ICD 162 or 163).

Table 3 describes the cohort by duration of employment; generally shorter at Asbestos than at Thetford Mines. In table 4 the four service groups have been further subdivided, as described in the

Table 3 *Gross service of men, by mining area. (Percentages in parentheses)*

Length of gross service (yr)	*Asbestos*	*Thetford Mines*	*Complete cohort*
Very short (< 1)	1783 (33.6)	1224 (21.7)	3007 (27.5)
Short (1-< 5)	1215 (22.9)	1109 (19.7)	2324 (21.2)
Medium (5-<20)	1147 (21.6)	1356 (24.1)	2503 (22.9)
Long (≥ 20)	1157 (21.8)	1948 (34.6)	3105 (28.4)
Total	5302 (100)	5637 (100)	10939 (100)

Table 4 *Age at start and duration of employment, with dust exposure. (Numbers of men, with average age at start of employment, average gross and net service, in years, and average dust "concentration")*

Length of gross service (yr)		*Accumulated dust exposure (see text)*			
		Low	*Medium*	*High*	*Very high*
Very short (< 1)	No	1022	838	571	576
	Age at start	26.3	26.1	24.9	22.9
	Gross service	0.2	0.4	0.4	0.5
	Net service	0.2	0.4	0.4	0.5
	*Concentration**	*2.6*	*4.3*	*14.4*	*78.0*
Short (1-< 5)	No	593	867	484	380
	Age at start	28.6	26.1	23.5	20.8
	Gross service	1.7	2.5	2.6	3.0
	Net service	1.3	2.2	2.5	2.8
	*Concentration**	*2.5*	*6.2*	*23.6*	*82.6*
Medium (5-< 20)	No	714	758	544	487
	Age at start	28.6	27.7	24.5	22.3
	Gross service	9.5	12.4	12.2	12.4
	Net service	6.4	10.4	10.5	11.3
	*Concentration**	*2.5*	*5.6*	*17.0*	*62.3*
Long (≥ 20)	No	1037	867	565	636
	Age at start	24.1	23.0	22.3	21.6
	Gross service	31.3	31.6	31.6	33.4
	Net service	24.9	27.8	28.6	30.8
	*Concentration**	*4.2*	*9.4*	*19.2*	*46.8*

*Found as average of dust exposure accumulated over gross service divided by average net service and expressed as million particles per cubic foot (mpcf)

previous section. In making these subdivisions priority was given to keeping subcohorts of adequate size for analysis, rather than to maintaining the same boundaries between dust concentration categories. Table 4 shows, nevertheless, that the mean *concentrations* (mpcf) in each set of four did not range very widely: low: 2.5 to 4.2; medium: 4.3 to 9.4; high: 14.4 to 23.6; very high: 46.8 to 82.6.

Dust concentrations have always been lower at Asbestos than at Thetford Mines. Thus to qualify for high dust exposure men at Asbestos had to have started work earlier or to have had longer service, particularly net service; gaps (as recorded) also were shorter, proportionately, at Asbestos. For the second form of a priori analysis, the men were classified by their dust exposure accumulated to age 45 (table 5). There is recent evidence that a very few employment gaps, some due to war service, did not appear in the work histories at Asbestos, and so exposures may be slightly overstated there. Nevertheless, table 5 shows a substantially lower proportion of men at Asbestos with exposures greater than 300 mpcf y.

Table 5 *Dust exposure accumulated to age 45. (Percentages in parentheses)*

Dust exposure (mpcf y) accumulated to age 45 years	*Asbestos*	*Thetford Mines*	*Complete cohort*
≤ 30	2954 (55.7)	2532 (44.9)	5486 (50.2)
30-< 300	1686 (31.8)	2027 (36.0)	3713 (33.9)
≥ 300	662 (12.5)	1078 (19.1)	1740 (15.9)
Total	5302 (100)	5637 (100)	10939 (100)

Despite some difficulties in the classification of smoking histories (mainly because these had not been obtained for most of those lost to view nor for about 800 men who had died before 1950; but also because of certain coding errors[6] (which we did not consider important enough to correct) there appear to have been only small differences between the two mining areas, and no major association between smoking habit and dust exposure to age 45. There were, however, clear differences in the reported smoking habits of the men born in the six quinquennia: the proportion of non-smokers was 24.9% among those born 1891-5 and lower in each succeeding quinquennium, being only 9.3% among those born 1916-20; correspondingly, the proportions of heavy smokers rose in each quinquennium, from 2.9% to 12.3%.

Tables 6 and 7 analyse the 3291 deaths, 20 or more years after first employment, occurring from 1951 to 75. Comparison with table 2 shows that, although 26.3% of all observed deaths were thus excluded from the analysis because they occurred before 1951 or within 20 years of first employment, over 90% of deaths from pneumoconiosis and from lung cancer were included, and percentages were also high for malignant neoplasms of other sites (except the larynx) and stroke. Only small proportions, however, were included of deaths from respiratory tuberculosis (23.0%) and from accidents (39.7%), with other specific groups of causes intermediate.

When account is taken only of length of service (table 6), trends of risk, as measured by the ratios of observed to expected deaths—that is, SMRs in which the standardisation was by both age and era—

Table 6 *Deaths, by cause, in relation to duration of service*

Cause of death (see table 2)	*Length of gross service (yr)*									
	Very short (< 1)		*Short (1 – 5)*		*Medium (5 – 20)*		*Long (≥ 20)*		*Complete cohort*	
	O	*SMR*	*O*	*SMR*	*O*	*SMR*	*O*	*SMR*	*O*	*SMR*
All causes	885	1 07	629	1 09	679	1 15	1098	1 07	3291	1 09
Pneumoconiosis	1	1 15	3	5 00	2	3 39	36	34 62	42	13 55
Malignant neoplasms										
Lung	47	0 97	29	0 83	50	1 37	104	1 61	230	1 25
Oesophagus and stomach	37	1 30	25	1 27	18	0 91	50	1 47	130	1 27
Colon and rectum	22	0 78	13	0 67	23	1 16	21	0 62	79	0 78
Other abdominal	20	1 08	12	0 92	14	1 04	21	0 90	67	0 98
Larynx	6	1 48	5	1 75	1	0 34	4	0 78	16	1 07
Other	67	1 12	43	1 04	48	1 13	79	1 08	237	1 09
Heart disease	370	1 06	251	1 02	287	1 15	424	0 97	1332	1 04
Respiratory tuberculosis	7	0 62	7	0 89	21	2 68	22	1 56	57	1 39
Other respiratory	29	0 66	46	1 52	22	0 71	59	1 12	156	0 99
Cerebrovascular	62	0 95	49	1 12	50	1 13	82	1 11	243	1 07
Accidents	52	1 36	38	1 32	37	1 18	56	0 96	183	1 17
All other known causes	130	1 03	94	1 07	94	1 05	132	0 85	450	0 98
Cause not known	35	—	14		12		8		69	

Columns headed O give the numbers of deaths of men 20 years or more after first employment, occurring during 1951-75; figures under headings SMR are ratios of deaths observed to those expected on basis of male mortality in Quebec.

were generally without clear trends, probably reflecting differences in selection and other factors already discussed. Exceptions were deaths attributed to pneumoconiosis and to accidents: of the 42 deaths from pneumoconiosis 36 were in men with at least 20 years' service; the trend with accidents was in the reverse direction, but it must be emphasised that, as the study interval started only 20 years after first employment, the trend is not a reflection of early work accidents in the asbestos industry shortening service prematurely.

Among those in the very short and short service groups—that is, those with gross service of less than five years (tables 7(a) and (b))—careful study of differences between groups according to severity of exposure showed no consistent pattern. Table 7(c) deals with men with gross service between five and 20 years; their service had also been completed before the start of the study interval. There were fairly consistent trends for higher SMRs the greater the dust exposure for total mortality, for pneumoconiosis (although based on only two deaths), heart disease, and stroke. In addition SMRs were highest in the group with the most severe exposure, for lung cancer and "other" respiratory diseases. All these findings are understandable as pulmonary fibrosis could well contribute directly to cardiopulmonary disease and, in addition, might adversely affect the probability of survival in any life-threatening condition. Table 7(d) concerns 3105 men

Table 7 *Deaths, by cause, in relation to dust concentration*

(a) Gross service less than one year

Cause of death (see table 2)	*Accumulated dust exposure (see table 4)*							
	Low		*Medium*		*High*		*Very high*	
	O	*SMR*	*O*	*SMR*	*O*	*SMR*	*O*	*SMR*
All causes	311	1 12	260	1 13	162	0 95	152	1 03
Pneumoconiosis	0	0	0	0	1	5 66	0	0
Malignant neoplasms								
Lung	19	1 17	12	0 91	9	0 88	7	0 80
Oesophagus and stomach	12	1 24	12	1 50	9	1 54	4	0 81
Colon and rectum	5	0 52	7	0 88	6	1 03	4	0 81
Other abdominal	3	0 48	6	1 17	4	1 04	7	2 12
Larynx	2	1 45	2	1 77	1	1 19	1	1 40
Other	20	0 99	23	1 38	13	1 05	11	1 04
Heart disease	136	1 15	112	1 15	63	0 87	59	0 94
Respiratory tuberculosis	4	1 05	1	0 32	1	0 44	1	0 48
Other respiratory	11	0 74	10	0 82	3	0 33	5	0 66
Cerebrovascular	25	1 14	18	0 98	9	0 67	10	0 90
Accidents	16	1 30	19	1 86	10	1 27	7	0 90
All other known causes	45	1 06	29	0 82	26	1 00	30	1 33
Cause not known	13	—	9	—	7	—	6	—

See footnote to table 6.

7(b) *Gross service: one year, less than five years*

Cause of death (see table 2)	*Accumulated dust exposure (see table 4)*							
	Low		*Medium*		*High*		*Very high*	
	O	*SMR*	*O*	*SMR*	*O*	*SMR*	*O*	*SMR*
All causes	131	1.12	246	1.09	130	1.12	112	1.04
Pneumoconiosis	0	0	3	12.80	0	0	0	0
Malignant neoplasms								
Lung	5	0.66	13	0.95	6	0.82	5	0.78
Oesophagus and stomach	8	1.83	7	0.90	4	1.03	6	1.64
Colon and rectum	2	0.46	4	0.52	4	1.04	3	0.82
Other abdominal	2	0.70	7	1.37	2	0.75	1	0.41
Larynx	2	3.17	1	0.89	1	1.71	1	1.90
Other	14	1.53	16	0.96	9	1.08	4	0.52
Heart disease	51	0.95	99	1.03	59	1.19	42	0.92
Respiratory tuberculosis	0	0	5	1.64	1	0.61	1	0.65
Other respiratory	10	1.49	16	1.34	10	1.66	10	1.78
Cerebrovascular	18	1.83	17	0.98	10	1.19	4	0.49
Accidents	11	1.89	12	1.10	3	0.47	12	2.14
All other known causes	16	0.83	40	1.16	16	0.91	22	1.33
Cause not known	2		6		5	–	1	–

See footnote to table 6

7(c) *Gross service: five years, less than 20 years*

Cause of death (see table 2)	*Accumulated dust exposure (see table 4)*							
	Low		*Medium*		*High*		*Very high*	
	O	*SMR*	*O*	*SMR*	*O*	*SMR*	*O*	*SMR*
All causes	161	1.10	194	1.07	170	1.22	154	1.26
Pneumoconiosis	0	0	0	0	1	7.36	1	8.42
Malignant neoplasms								
Lung	13	1.41	14	1.22	7	0.83	16	2.17
Oesophagus and stomach	6	1.21	6	0.99	5	1.07	1	0.25
Colon and rectum	4	0.81	7	1.14	9	1.92	3	0.74
Other abdominal	6	1.78	3	0.72	3	0.95	2	0.73
Larynx	0	0	0	0	1	1.44	0	0
Other	9	0.85	19	1.44	11	1.10	9	1.03
Heart disease	66	1.06	81	1.05	72	1.22	68	1.31
Respiratory tuberculosis	1	1.55	9	3.94	5	2.64	4	2.28
Other respiratory	5	0.64	5	0.51	5	0.69	7	1.12
Cerebrovascular	8	0.73	13	0.94	14	1.34	15	1.67
Accidents	8	1.07	10	1.08	10	1.33	9	1.28
All other known causes	29	1.30	21	0.77	25	1.17	19	1.01
Cause not known	4		6		2		0	

See footnote to table 6

7(d) *Gross service: 20 or more years*

Cause of death (see table 2)	*Accumulated dust exposure (see table 4)*							
	Low		*Medium*		*High*		*Very high*	
	O	*SMR*	*O*	*SMR*	*O*	*SMR*	*O*	*SMR*
All causes	367	0.98	253	0.89	183	1.07	295	1.50
Pneumoconiosis	4	10.49	7	23.75	5	30.10	20	101.52
Malignant neoplasms								
Lung	28	1.21	20	1.08	24	2.20	32	2.65
Oesophagus and stomach	17	1.36	6	0.64	8	1.44	19	2.89
Colon and rectum	7	0.56	4	0.43	1	0.18	9	1.39
Other abdominal	10	1.18	3	0.46	2	0.51	6	1.35
Larynx	2	1.07	1	0.69	0	0	1	1.03
Other	33	1.23	16	0.79	11	0.90	19	1.36
Heart disease	138	0.87	115	0.95	77	1.06	94	1.12
Respiratory tuberculosis	5	1.01	5	1.31	3	1.27	9	3.06
Other respiratory	18	0.92	10	0.68	14	1.62	17	1.74
Cerebrovascular	32	1.15	18	0.89	10	0.84	22	1.58
Accidents	16	0.82	19	1.16	9	0.85	12	1.01
All other known causes	52	0.92	29	0.68	18	0.70	33	1.10
Cause not known	5		0		1	–	2	–

See footnote to table 6

Table 8 *Deaths, by cause, in relation to dust exposure accumulated to age 45*

Cause of death (see table 5)	Dust exposure (mpcf y) accumulated to age 45					
	< 30		30 – < 300		300	
	O	SMR	O	SMR	O	SMR
All causes	1668	1.02	1138	1.04	642	1.30
Pneumoconiosis	5	2.98	12	10.81	27	54.00
Malignant neoplasms						
Lung	91	0.93	81	1.18	70	2.25
Oesophagus and stomach	68	1.25	42	1.14	26	1.58
Colon and rectum	54	0.62	28	0.77	18	1.11
Other abdominal	33	1.00	21	0.84	10	0.88
Larynx	9	1.11	6	1.08	2	0.81
Other	129	1.10	83	1.06	38	1.08
Heart disease	696	1.00	463	0.99	240	1.14
Respiratory tuberculosis	21	0.94	25	1.67	15	2.20
Other respiratory	71	0.84	55	0.98	40	1.62
Cerebrovascular	119	0.96	56	1.08	46	1.32
Accidents	104	1.28	60	1.00	33	1.16
All other known causes	237	0.95	154	0.92	74	0.99
Cause not known	47		22		3	

See footnote to table 6

with at least 20 years' service, and an average of almost 32 years of employment (table 4). Here, the most severely exposed had the highest SMR not only for total mortality but for all listed causes other than laryngeal cancer and accidents. Further, the tendency for increased risk with each augmentation in exposure was completely consistent for pneumoconiosis and for heart disease, and positive, although rather less consistent, for total mortality, lung cancer, respiratory tuberculosis, and other respiratory diseases.

The other form of a priori analysis, with exposure calculated to age 45 at which age the study interval started, is summarised in table 8. The total number of deaths observed in this analysis was 3448 (77.3% of all deaths), with SMR = 1.07, very close to that for all causes in the complete cohort as given in table 6. Indeed, for each cause of death, SMRs from both methods of analysis were always close.

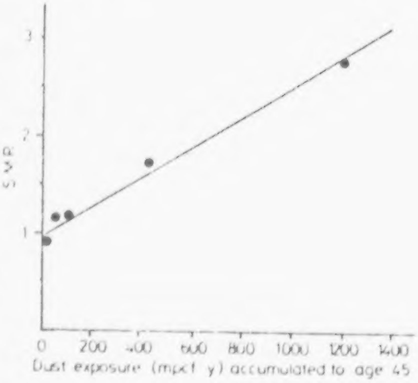
Clear trends were found for SMRs to be higher the heavier the exposure, for total mortality, pneumoconiosis, lung cancer, cancer of colon and rectum, respiratory tuberculosis, other respiratory diseases, and stroke. The trends were most clear-cut in pneumoconiosis and lung cancer, appearing in both Asbestos and Thetford Mines. The lung cancer trend was essentially linear as shown in the figure, where exposures of 30 mpcf y or more have been broken down further, into four classes. The trend for respiratory tuberculosis was also consistent in the two areas, but not those for the other causes listed here. The risk of death from cancer of the oesophagus or stomach was highest in the most severely exposed, but only at Thetford Mines; so was the risk of death due to heart disease, but only at Asbestos. Other abdominal cancers and laryngeal cancers were

Table 9 *Deaths from lung cancer in relation to dust exposure and smoking habit*

Smoking habit*	Dust exposure (mpcf y) accumulated to age 45							
	< 30		30 – < 300		300		All	
	O	SMR	O	SMR	O	SMR	O	SMR
Non-smokers	5	0.48	6	0.36	8	1.24	19	0.58
Moderate smokers	73	1.14	64	1.35	52	2.31	189	1.41
Heavy smokers	13	2.12	11	2.39	10	4.80	34	2.63
All smoking habits	91	0.93	81	1.18	70	2.25	242	1.23

See footnote to table 6

*See text for definitions



Lung cancer SMRs in relation to dust exposure accumulated to age 45. The line has been fitted by a modified least squares technique inversely related to exposure, and no other patterns could be elucidated.

The last line of table 9 is repeated from the previous table, but the right-hand margin shows a clear and substantial effect of cigarette smoking. For each category of smoker, the risk of lung cancer was lowest among those with low exposure to age 45 and highest among those with exposure of at least 300 mpcf y by that age. The interaction between the factors of smoking and dust exposure is examined later (table 11).

Corresponding detail for laryngeal cancer is confusing because of the very small number of deaths from this cause. As already seen (table 8), the dust effect was, if anything, inverse; however, the smoking effect was quite strong: non-smokers, two deaths, SMR = 0.46; moderate smokers, 10 deaths, SMR = 0.93; heavy smokers, five deaths, SMR = 4.85.

Table 10 summarises the findings from the Miettinen approach, that is, more than one control for each case, excluding those for smoking habit; the numbers of deaths are as in table 2 (but see footnote ‡ in table 10) because there were no restrictions on the start of the study interval. Four groups of dust exposure are distinguished, and the data are presented without regard to the matching. Matching was taken into account in the full analysis, however, which generally confirmed the tendencies shown in the two a priori approaches (Thomas, DC, unpublished), and relative risks were fairly similar at Asbestos and Thetford Mines.

Table 10 *Dust exposure in deaths from pneumoconiosis and from malignant disease and in controls*

	Dust exposure (mpcf y) accumulated up to [illegible] years before death of case				
	<30	30–<300	300–<1000	≥1000	All
Pneumoconiosis					
Deaths	7	9	13	17	46
Controls (3)*	63	49	21	5	138
Relative risk†	1	1.65	5.57	30.60	
Lung cancer					
Deaths‡	89	73	56	27	245
Controls (3)	333	243	127	32	735
Relative risk	1	1.12	1.65	3.16	
Cancer of oesophagus and stomach					
Deaths	74	41	22	17	154
Controls (2)	143	105	53	7	308
Relative risk	1	0.75	0.80	4.69	
Cancer of colon and rectum					
Deaths	[illegible]	[illegible]	11	7	88
Controls (2)	88	70	15	3	176
Relative risk	1	0.93	1.96	8.26	
Other abdominal cancers					
Deaths	41	25	7	8	80
Controls (2)	81	46	26	5	160
Relative risk	1	1.07	0.52	1.93	
Cancer of larynx					
Deaths	13	6	2	0	21
Controls (3)	36	21	5	1	63
Relative risk	1	0.79	1.11	0.00	

*Figures in brackets are numbers of controls for each death. Method of selecting controls is described in text; those reported here were not matched for smoking habit.

†Risk calculated by method of Doll in relation to those with exposure less than 30 mpcf y.

‡Excluding five deaths coded to 162-164 but found to be due to malignant mesothelioma.

Table 11 *Dust exposure in deaths from lung cancer and in controls matched for smoking habit*

	Dust exposure (mpcf y) accumulated up to [illegible] years before death of case				
	<30	30–<300	300–<1000	≥1000	All
Non-smokers					
Deaths	5	5	5	5	20
Controls*	10	6	3	1	20
Relative risk†	1	1.67	3.33	10.00	
Smoking habit undifferentiated					
Deaths	43	23	22	12	100
Controls	49	33	17	1	100
Relative risk	1	0.79	1.47	13.67	
Definite cigarette smokers					
Deaths	41	45	29	10	125
Controls	49	48	22	6	125
Relative risk	1	1.12	1.58	1.99	
All smoking habits					
Deaths‡	89	73	56	27	245
Controls	108	87	42	8	245
Relative risk	1	1.02	1.62	4.10	

*One control for each death.

†Risk calculated by method of Doll in relation to those with exposure less than 30 mpcf y.

‡Excluding five deaths coded to 162-164 but found to be due to malignant mesothelioma.

The interaction between dust exposure and smoking in lung cancer mortality is explored in table 11, where the distribution of dust exposure of the single control matched so far as possible for smoking is compared with that of the cases in each smoking category separately. The final section of this table differs from the corresponding part of the previous table because of the different selection of controls, but the relative risks are similar. Further, the relative risks within smoking categories are in reasonable accord with those obtainable from table 9, despite major differences in the methods of determining them.

Clearly lung cancer can occur in non-smokers, and indeed it might seem from our data that the relative risk due to asbestos dust exposure was, in fact, higher for non-smokers than for smokers. Linear dose-response relations have been fitted (Berry, G, unpublished), so far without regard for smoking: using the data on which table 10 is based, but taking into account the matching of controls for each case in terms of date of birth and place of employment, the fitted line was

$$\text{Relative risk} = 1 + 0{\cdot}0014\ (\text{mpcf y})$$

the standard error of the estimate of the slope being 0·0005. The linear fit accounted for χ^2, with one degree of freedom, of 21·37, leaving only a very low value for deviations from linearity.

There were in all 11 deaths (including one of a woman) from malignant mesothelial tumours observed to the end of 1975. All were of the pleura and appeared to follow a clear exposure trend. The problems of interpretation are complex, however, and the subject will be examined in detail elsewhere.[11]

WOMEN

Of the 440 women in the cohort, only 31 were lost to view (7·0%), a better rate of tracing than for men. Of those traced, 325 were alive until 1976 (79·5%) and 84 had died (20·5%), the lower percentage mortality than in the men being largely explained by the younger age of the women. The women had been employed mainly at Asbestos (408 (92·7%)), and only 84 (19·1%) had been exposed to more than 30 mpcf y by age 45. In addition to the case of pleural mesothelioma mentioned above, there was one death from lung cancer, compared with 1·19 expected. Careful study of the remaining detail showed no other exposure-related effect.

Discussion

This survey, begun in 1966 and continued without interruption for 12 years, has sought to determine the quantitative relations between occupational exposure in the chrysotile production industry of Quebec and mortality. Since analyses conducted at four stages during the 10-year period, by increasingly varied and sophisticated statistical methods, have been closely consistent,[3] we conclude that our objective has been achieved. Essentially linear relations have been shown between indices of exposure, based on dust concentration (mpcf) multiplied by length of service, and lung cancer, pneumoconiosis, and total number of deaths. Using this measure of environmental quality, we are now able to assess, with reasonable confidence, the mortality risk associated with work at different levels of dust concentration and for social decisions to be made as to what level, if any, is "acceptable."[12] As almost half of the cohort is now dead, no further study would appear necessary were it not for the current trend toward setting hygiene standards in terms of fibre rather than dust counts — despite the almost complete lack of epidemiological data based on fibre measurements. There would be no serious problem were the conversion a simple one. The evidence of Gibbs and Lachance[13] shows that this is not the case and so we have now embarked on a laborious attempt to estimate past exposures in fibres/ml and will use these in further analyses of our mortality and morbidity findings. In the meantime we may note the results of an analysis of Gibbs's data by Dagbert,[14] which showed that, at the relevant dust levels, the conversion factors range from about 3 to 7 fibres/ml for each mpcf. Further data now available to us, however, point to a lower range, perhaps 1 to 5.

When considering what degree of environmental exposure can be regarded as "safe," a linear response to exposure is a great convenience.[3,15] It allows extrapolation to exposure levels far below those at which reliable direct measurement of risk is possible. Our findings by two methods of analysis conform well with the hypotheses of linearity, but they do not constitute proof. It is not unreasonable, however, to suppose that the probability of initiation of the malignant process by mutation may be dose-related and that defence mechanisms will not change this.

Similar uncertainty remains over the form of interaction between asbestos exposure and cigarette smoking. Our data seem as compatible with the "additive model" of relative risk (model 1) discussed by Saracci,[16] as with the "multiplicative model" — that is, with the simplest form of his model 2; they are quite incompatible with model 3, in which "asbestos can only increase lung cancer in the presence of smoking." Either model allows one to speculate that the dust concentrations in the Quebec mills in the early 1950s, before effective dust suppression was introduced, carried a lung cancer risk equivalent to heavy smoking whereas, at more recent concentrations of around 1 mpcf, the order of risk may now approximate to less than one cigarette a day. Despite the much greater sophistication of the "matched" case-control analyses of the data (Thomas, DC, unpublished), they gave essentially the same results as our "unmatched" analysis, and so far have not been able to increase confidence in the exact form of the interaction.

Several factors bearing on the validity of this survey warrant examination. A study of this kind is as good as the environmental and mortality records on which it is based. On the former we can add little: the very large number of environmental measurements made in this industry since about 1950 have been used to the full, but for the more important earlier years we have only our estimates to go on.

While this is frankly acknowledged, two points can be made: (1) the estimates were based on many years of extensive study of Quebec mines and mills with very varied hygiene standards; and (2) random errors in exposure estimates would have been expected to obscure rather than enhance correlation between exposure and response.

At least equal effort was put into the follow-up and ascertainment of registered causes of death. In terms of completeness we believe this satisfactory; almost all of the 10% of subjects lost to view were men with short service many years ago; over 98% were traced after 1935 and a cause was obtained for 97% of deaths.

Questions remain, however, on the validity of recorded cause of death and on possibilities of related bias. These questions apply only to cause of death and not to the fact of death—the more important issue. It must be faced that, in this, as in other occupational cohort studies, there is considerable scope for diagnostic bias. In writing a death certificate most physicians prefer probable to improbable causes; men who die of respiratory disease after long heavy exposure to dust could well have had pneumoconiosis, whereas this must be considered unlikely in men without such history. Selective necropsies will, if anything, increase this type of bias. Even a 100% necropsy rate in the study population would overcome the problem only if the same population is used as the basis for calculating expectations. The net result, discussed in our first report on this cohort,[4] is that there may be a bias towards the over diagnosis of dust-related disease in men with heavy exposure. The effect will be intensified by a greater frequency of necropsies in deaths where a prima facie case for compensation exists. There was evidence of this in the high necropsy rate (52%) in deaths ascribed to pneumoconiosis. The effect of this trend might well overflow into those causes of death—especially cancer of the lung and other organs—which are under-diagnosed without necropsy. None of this, however, affects the validity of the exposure-response relation for deaths (all causes), although conceivably it contributes to the linearity and excess for pneumoconiosis and lung cancer.

A related problem in cohort studies is the so-called "healthy worker effect" (see, for example, Fox and Collier[17]) resulting from non-random selective entry and exit from various industries and for jobs within the industries. It is an unfortunate term because these selective forces, dependent on circumstances, may result in healthy or unhealthy worker effects. These effects matter because no observational study in epidemiology can correct for them; this is especially so in a priori analyses of the man-years type using an external reference population. Tables 6, 7(a), and 7(b) show some of the problems of interpretation: men with gross service less than five years (*a*) had about the same SMRs for all causes (1·07, 1·09) as those with long service (1·07); and (*b*) had SMRs that were higher at the lower exposure levels. Part of the explanation may be that the less fit went to the less heavy or less dusty jobs and often did not stay long in the industry. Another part may be that those with the shortest employment were the most difficult to trace, and among them death was probably more completely ascertained than survival, but this effect must be small because of the high trace rate actually achieved—that is, more than 99% after 1950.

This survey, and related research, in the chrysotile production industry were undertaken because of the critically important need, expressed in 1964,[1] for studies to compare the pathogenicity of the main asbestos fibre-types. A survey[18] of the much smaller Italian chrysotile mining industry has yielded findings very similar to our own; unfortunately, no comparable observations have been made on crocidolite or amosite production. Nor have there been any reports on mortality after exposure to single asbestos fibre-types in manufacturing or application, in which exposure was measured in more than duration. Two small factories working with chrysotile only were studied by Elwood and Cochrane[19] and Weiss,[20] a large plant with amosite only by Selikoff and Hammond,[21] and a small group with crocidolite only by McDonald and McDonald.[22] Because dust or fibre concentrations were not measured in these studies, comparisons of frequency of lung fibrosis and cancer cannot be made with confidence, although the evidence all points in the direction of more serious effects from the amphiboles than from chrysotile. The incidence of malignant mesothelial tumours, especially of the peritoneum, is so very much higher after exposure to amphiboles (and amphibole-rich mixtures) than after exposure to chrysotile alone, that differences in dust concentration are unlikely to explain it.[22–24]

As in earlier reports on the Quebec cohort, the patterns of gastrointestinal cancer are difficult to interpret. There was unquestionably a substantial excess in mortality from cancer of the upper gastrointestinal tract in men most heavily exposed. This excess was confined to Thetford Mines where most of the long and heavy exposures occurred. The exposure-response curves were far from regular, however, and it appears that some other important factors were also operating—perhaps selective, perhaps environmental. The SMR for cancer of the lower gastrointestinal tract for the complete cohort was 0·78 (table 6); nevertheless, there was some evidence of an exposure-response trend.

Our findings on laryngeal cancer are clear cut, although others' have not been so. There has been no excess in this industry, and there is no quantitative association with exposure; on the other hand, we find a direct relation with cigarette smoking.

Because an important current concern is the risk from concentrations of asbestos dust nearer to today's standards, we have re-examined our data for the 1904 men in the cohort employed for at least 20 years in the low and medium dust exposure groups. The concentrations to which these men were exposed (see table 4 and footnote*) averaged 6·6 mpcf, or perhaps 20 fibres/ml, and thus were much above anything permissible now. Nevertheless, as can be deduced from table 7(d), the total mortality in the study interval was 620 deaths, and the SMR was 0·94. This may well be a true healthy worker effect but not all cause-specific SMRs were below unity. Indeed, there were excesses for pneumoconiosis (10·3 excess deaths, leading to χ^2 on the usual basis, and with one degree of freedom, of 159·27), for lung cancer (6·4, $\chi^2 = 0·99$); cancer of oesophagus and stomach (1·1, $\chi^2 = 0·06$); "other" cancers (1·7, $\chi^2 = 0·06$); respiratory tuberculosis (1·3, $\chi^2 = 0·17$); and stroke (1·8, $\chi^2 = 0·07$). Apart from pneumoconiosis, these values of χ^2 are so low, even for lung cancer (where the associated p-value is 32·0%), that the observed excesses do not reach conventional levels of statistical significance. Moreover, the lung cancer SMR for the low dust exposure group (1·21) was higher than that (1·08) for the medium exposure group; only the greatly enhanced SMRs for those with high and very high exposure allow the conclusion that there was a response to exposure. Nevertheless, the lung cancer SMR for all 1904 men was 1·15, in close conformity with that which might be predicted from the figure (around 1·20), or the relative risk of 1·16 from the fitted line (Berry, G, unpublished). By their nature, epidemiological surveys, however large, cannot achieve greater precision than the one described here. Hence the clear importance of deciding whether a linear exposure-response model should be adopted, if only for practical reasons.

This survey was made possible by the contribution of many people and agencies, in private or official capacity. We would mention, in particular, Mmes M Beauchemin and F Proulx for their work in maintaining contact with ex-employees, Dr J M Fredette for help in finding death certificates, and Mmes M Gauthier and C Tessier for coding them. We had the active co-operation throughout of the mining companies and the several trades unions with members in the industry. Grants toward the cost of the research were made to McGill University and to the London School of Hygiene and Tropical Medicine by the Institute of Occupational and Environmental Health of the Quebec Asbestos Mining Association.

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MEDICAL PROGRESS

THE PATHOGENESIS OF ASBESTOS-ASSOCIATED DISEASES

John E. Craighead, M.D., and Brooke T. Mossman, Ph.D.

ASBESTOS is one of our most useful minerals. Over 3000 manufactured products of contemporary importance contain it. Asbestos is employed in construction materials because it is resistant to thermal and corrosive destruction and increases the tensile strength of the product. These properties are also the basis for the use of the mineral in friction equipment and in a wide variety of consumer items requiring a relatively inexpensive insulation material that is light and subject to molding. Since the turn of the century, about 3×10^7 tons of asbestos have been used in construction and in the fabrication of manufactured goods in the United States. At present, several million Americans are employed in industries that use asbestos products, and countless millions of American citizens are exposed to asbestos cryptically in the course of their daily lives.

Public concern over the effects of asbestos on health is mounting. Although a total ban on its use in this country has been proposed, most would agree that asbestos cannot be replaced expeditiously in many products. Litigation based on personal injury consequent to pulmonary fibrosis and cancer is an increasing problem for companies involved in the manufacture, use, and distribution of asbestos. About 12,000 suits have been brought against 260 companies by workers, their families, and members of the general public.[1,2] The spectrum of liability has now widened to involve the federal government for alleged negligence in establishing adequate environmental standards.

This review summarizes our current knowledge of the adverse effects of asbestos on health and provides a perspective on the pathogenetic mechanisms of the diseases associated with exposure. Since there are several different mineralogic types of asbestos, we will attempt to assess the extent to which findings with one type can be applied to another. Detailed analyses of the issues addressed in this paper have been published elsewhere.[1-6]

MINERALOGY

Asbestos is not one mineral but a family of fibrous hydrated silicates that are divided on the basis of mineralogic features into two groups: the serpentines and

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the amphiboles (Fig. 1). The term "asbestos" refers to the commercial product after mining and processing and is not a mineralogic designation. Although the length:width ratio of the mineral fiber known as asbestos is by definition ≥3:1, the individual fibers making up the materials used in commerce vary substantially in width and length (Fig. 2).

Chrysotile is the only serpentine of commercial importance. It is composed of pliable, curly fibers made up of fibrillar subunits. These fibrils are arranged in pseudohexagonal arrays composed of silicon oxide sheets formed into scroll-like structures. The magnesium ion, which imparts a strongly positive charge to the fiber, is an integral component of the lattice.

The amphiboles are straight, rodlike fibers consisting of double chains of tetrahedral groups having a basic silicon oxide composition and linked by one or more cations. The amphiboles differ from chrysotile in both physical and chemical makeup. There are several types of amphibole, but crocidolite and amosite are the two minerals of major importance.

Although an asbestos type is classified on the basis of its mineralogic characteristics, the products of different mines are not necessarily the same. Moreover, a commercial type of asbestos is not always mineralogically pure. For example, Canadian chrysotile contains small amounts of an amphibole fiber, tremolite. In addition, industrial grades of asbestos are contaminated with extraneous inorganic and organic substances that are acquired either naturally or during processing.

Deposits of serpentine and amphibole are ubiquitous in the crust of the earth. Outcrops are found in many geologic formations and probably account for the mineral fibers commonly found in surface water. Asbestos is also found with other minerals of commercial importance, such as the iron ore taconite and industrial-grade talc. Canada and South Africa are the major suppliers in the western world, although mines of limited commercial importance are found in many countries. In the United States serpentine and amphibole minerals are distributed widely in geologic strata, but only two relatively small mines in Vermont and California are active. The amount of asbestos produced in the Soviet Union and the People's Republic of China far exceeds that extracted in the West.

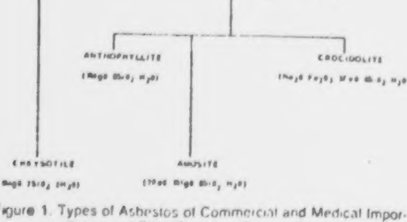

Figure 1. Types of Asbestos of Commercial and Medical Importance and Their Chemical Compositions

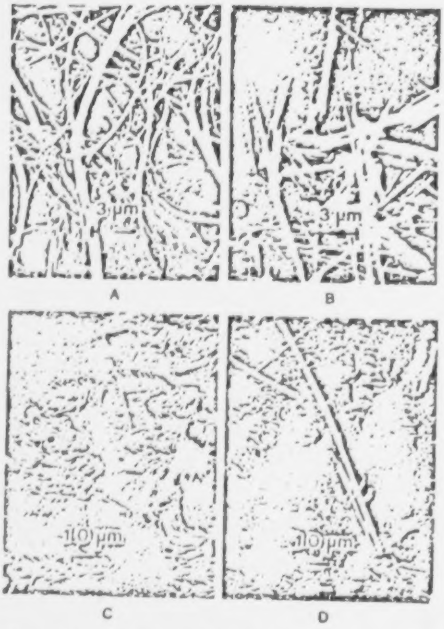

Figure 2. Differing Structural Features of Serpentine (Chrysotile) and Amphibole (Crocidolite) Asbestos.

These scanning electron micrographs of International Union against Cancer reference samples of chrysotile (Panel A) and crocidolite (Panel B) asbestos illustrate the heterogeneity of fibers in both length and diameter. Micrographs of the hamster tracheal epithelium after exposure in vitro to asbestos illustrate the curly, pliable nature of chrysotile (Panel C) and the straight, rod like form of crocidolite (Panel D). Note the dimensions of the fibers in comparison to the cilia. Photomicrographs were furnished by Mr. Craig Woodworth, Department of Pathology, University of Vermont College of Medicine.

Chrysotile currently accounts for over 90 per cent of the total asbestos marketed in this country and abroad. Crocidolite is the most widely used amphibole, but for reasons considered below, its commercial importance has decreased over the past several decades (Table 1).

Uses of Asbestos

The unique physical properties of asbestos dictate its continued use by industry, despite contemporary concerns about its effects on health. Although various man-made and naturally occurring substances have been developed as substitutes for asbestos, none

Table 1. Consumption of Different Types of Asbestos in the United States in 1978 *

Use	Type of Asbestos			Total Asbestos
	chrysotile	crocidolite	amosite	
		metric tons		
Asbestos cement pipes	119,700	24,100	200	144,000
Asbestos cement sheeting	7,900	—	—	7,900
Flooring products	90,000	—	200	90,200
Roofing products	26,500	—	—	26,500
Packing and gaskets	12,200	100	—	12,300
Insulation, thermal	6,000	—	—	6,000
Insulation, electrical	2,900	—	—	2,900
Friction products	43,700	—	—	43,700
Coatings and compounds	10,900	—	—	10,900
Plastics	1,200	100	—	1,300
Textiles	1,900	—	—	1,900
Paper	400	100	—	500
Other	9,000	—	1300	10,600
Total	332,300	24,400	1700	358,700

*Modified from the data of Fagan.[7]

matches asbestos in providing tensile strength and moldability as well as resistance to fire, heat, and corrosion. In addition, many of the manufactured substitutes are comparatively expensive.[8]

About 25 per cent of the asbestos consumed in the United States is incorporated into cement piping for water mains and sewage lines. Over 320,000 km of pipe, containing about 10 to 20 per cent asbestos, is believed to be in use in this country. Asbestos-containing cement is employed widely in corrugated and flat sheeting, panels, tiles, and moldings for the construction industry. The mineral is used extensively in roofing and paneling and as a filler in architectural dead spaces. In the past, suspensions of asbestos were sprayed onto the structural steel of buildings to provide insulation and fire protection.

Because of its thermal stability, asbestos is well suited for use in friction material and is applied to molded brake linings. Although substitutes are being increasingly employed in disk brakes, as in the aircraft industry, a drum-brake lining for passenger cars that does not contain asbestos is not available commercially.

Textiles and plastics of a variety of types and applications contain asbestos in various concentrations, since it imparts resistance to fire and corrosion as well as tensile strength without inordinately altering the properties of the product or increasing its weight.

The countless additional industrial uses of asbestos are of concern because they can be overlooked by the manufacturer and unrecognized by the consumer. Although asbestos was known to industry before the turn of the century, its use in the United States increased dramatically during the mobilization that accompanied World War II. Asbestos was employed liberally in the construction and reconditioning of ships and in such diverse war industries as the manufacture of aircraft engines, combat vehicles, and gas masks. Although worldwide production has continued to increase since the war, consumption in this country has dropped substantially during the past decade. This trend can be expected to continue. Since the latency period for the diseases associated with asbestos is usually 20 years or longer, most patients seen today were initially exposed in the 1940s and 1950s, when control measures were often not rigorous.

DISEASES OF THE RESPIRATORY TRACT AND THORAX

The major pathologic effects of asbestos result from the inhalation of fibers suspended in the ambient air. The occurrence of disease is influenced by the type of mineral and the dimensions of the fibers that constitute it, as well as by the concentration of fibers and the duration of exposure.

Deposition and Transport in the Lungs

Timbrell et al.[9] studied the deposition of fibers of asbestos in the respiratory tract, using a cast of the porcine tracheobronchial tree. The diameter of the individual fibers proved important, length was a less important determinant.[10,11] Fibers with a relatively broad diameter are deposited in the upper respiratory tract, whereas thin fibers are carried peripherally into the parenchyma of the lung, where they lodge in the terminal airways. Bifurcations are common sites for fiber impaction, since patterns of air flow are altered at these sites.

The shape of the fibers also has a role in transport. Aerodynamically, chrysotile has a relatively large theoretical cross-sectional diameter because of its curled configuration. Thus, fibers of this type tend to be deposited more proximally than the needle-like amphiboles, which are transported more readily to the periphery of the lung. These theoretical and experimental considerations have been verified by analyses of the lungs of rodents experimentally exposed to asbestos of different types.[12]

Three biologic mechanisms participate in the clearance of fibers from the lower respiratory tract. By far, the bulk of the dust is removed by the mucociliary escalator of the tracheal bronchial tree, and the material is either expectorated or swallowed.[13-15] In the peripheral airways, short fibers are ingested by macrophages, and at least some of these cells probably migrate across the wall of the bronchioles and acini.[16-18] Asbestos fibers are also taken up by the epithelial cells lining the airways and appear to move between cells of the mucosa.[18-20] This material accumulates in the interstitium and is carried to regional lymph nodes.[17] In general, short fibers are cleared more readily than long fibers,[17] which tend to be retained in the lumens of the respiratory bronchioles and the alveolar ducts.

About a third of the inhaled particles initially lodge in the distal airways. However, only about a quarter of this burden is retained in the respiratory tract one month later.[13] There are two phases of clearance through the tracheobronchial tree. About half the asbestos is removed within a few days. Subsequently, clearance continues for extended periods. The bulk of this material is excreted in the feces.[15]

A variety of extraneous influences such as cigarette smoke and air pollutants affect the clearance and intrapulmonary deposition of fibers. However, these factors are extraordinarily complex, in part because individuals appear to differ in their responses to inhaled dust.[21-29]

Asbestosis

Diffuse pulmonary fibrosis is the typical lesion associated with prolonged, heavy exposure to asbestos.[30] It develops slowly over a period of years and seems to progress in the absence of continued exposure to asbestos. Initially, fibrosis is found in and around the respiratory bronchioles and alveolar ducts, where relatively long fibers deposit. With time, the fibrotic lesion progresses in a seemingly centrifugal manner, so that increasing numbers of respiratory units are involved. Fibers of asbestos tend to accumulate preferentially in the lower lobes and adjacent to the visceral pleura. Fibrosis is usually prominent in these regions, and the pleural surfaces of these lobes are frequently thickened by a dense layer of fibrous tissue. In advanced asbestosis, the fibrotic pulmonary tissue contracts and is reorganized to form the new air space typical of the honeycomb lung.

Ferruginous bodies are the histologic hallmark of exposure to asbestos.[31-34] They consist of fibers coated by complexes of hemosiderin and glycoproteins and are believed to be formed by macrophages that have phagocytized the particles. Asbestosis can exist when ferruginous bodies are difficult to demonstrate in the lungs by light microscopy. On the other hand, ferruginous bodies can often be found in the absence of serious parenchymal disease.[35,36] Thus, their presence alone is probably not a stimulus for the proliferation of fibrous tissue. Although they have been shown to form from foreign inorganic and organic fibers of many different types,[37] ferruginous bodies in most human lungs have asbestos as a core.[36] For this reason, the structures are commonly known as asbestos bodies.

The number of uncoated fibers in the lung greatly exceeds the number of asbestos bodies in the tissue.[38,39] It is not known why some fibers are coated and form the typical asbestos bodies, whereas others are uncoated. Since uncoated fibers are usually difficult or impossible to demonstrate by light microscopy, lung tissue must be digested and the residue examined by either phase or electron microscopy in order to carry out qualitative and quantitative studies of the fibers. Whereas relatively long fibers (>5 μm) are found by light microscopical techniques, electron microscopy makes it possible to identify very small particles.[40,41] Thus far, attempts to correlate the extent of disease with either the number of asbestos bodies or the overall content of fibers in the lungs have been difficult, although fibrosis is usually evident when 10^6 fibers per gram of lung (wet weight) are present. Quantitative studies pose many problems and are only a crude measure of exposure, partly because many fibers are cleared from the lungs and others fragment to increasingly smaller particles with time.

Macrophages are a key element in the response of the host to asbestos. Whereas these cells phagocytize short fibers and remove them from the airways, they cannot encompass and transport the longer fibers. Although retention of these long fibers in the distal airways appears to be an important consideration in the causation of pulmonary fibrosis,[18,20] the pathogenesis of the lesion is not understood. Incomplete phagocytosis of asbestos fibers in the airways could result in spillage of lysosomal enzymes[42] and release of soluble fibrogenic factors from macrophages.[43] On the other hand, oxygen free radicals liberated by macrophages and other inflammatory cells might also injure lung tissue. This idea is supported by our observations that superoxide dismutase, an inhibitor of biologic oxidants, protects cultured respiratory epithelial cells from the cytotoxic effects of chrysotile (Mossman BT, Landesman JM: unpublished data). Chrysotile is cytotoxic in vitro presumably because the magnesium of the fibers interacts with the plasmalemma and damages it, along with lysosomal membranes of cells.[44,45] It is unclear whether this is an important mechanism of tissue injury, however, since pulmonary macrophages and epithelial cells in the lungs of animals exposed to aerosolized chrysotile fail to reveal ultrastructural evidence of injury.[18,19]

Other biologic phenomena may prove important in the causation of pulmonary fibrosis. Asbestos activates complement by the alternative pathway[46] — a reaction that may be expected to result in the accumulation of leukocytes in the tissue and the release of lysosomal enzymes. This observation is consistent with the finding of an acute inflammatory response in some early lesions.[30,47] Finally, consideration must be given to the possibility that asbestos stimulates the production of collagen by cells. When chrysotile is added to cultures of fibroblasts in vitro, the cells elaborate reticulin and collagen at an accelerated rate.[48,49]

Although the hypothetical mechanisms mentioned above could account for the deposition of fibrous tissue in the lungs, the pathogenesis of asbestosis in human beings remains to be established. The question may be moot, however, since modern environmental controls have dramatically reduced exposure in the work place. The dust concentrations permitted by current regulations will probably not induce substantial pulmonary fibrosis during the lifetime of an industrial worker.

Pleural Lesions

Plaques are curious lesions made up of hyalinized fibrous tissue located on the parietal pleura of the thorax, diaphragm, mediastinum, and pericardium.[50,51] They are usually but not invariably associated with exposure to asbestos.[30] Although the occurrence of plaques correlates with the duration and intensity of exposure, it is common to find lesions in the absence of obvious disease of the pulmonary parenchyma. Thus, relatively small amounts of dust can induce the development of plaques. These benign lesions do not appear to develop into malignant mesotheliomas.

Characteristically, plaques are located in the intercostal spaces on the anterior and posterior lateral aspects of the thorax and on the dome of the diaphragm at sites where the visceral and parietal pleuras approximate during respiratory excursions. The configuration of the plaques is highly variable. For example, on the chest wall they usually follow the contour of the rib, whereas on the diaphragm they are customarily either disk-shaped or geometrically shaped and have a nodular surface. Over time the lesions become calcified, permitting easy recognition on x-ray films. Although most of the available epidemiologic information is based on radiologic surveys,[52-55] it is not always clear in published reports whether plaques were differentiated from the fibrous lesions of the visceral pleura that accompany pulmonary asbestosis.

Since plaques are found most often in persons exposed occupationally to asbestos for extended periods,[54-56] their overall prevalence in the United States is low.[57] In Eastern Europe and Asia Minor the lesions are frequently found in older members of the general population who lack documented exposure to asbestos. The presence of fibrous minerals in soil and in local construction materials may account for the common occurrence of pleural plaques in these regions.[58-61]

Malignant mesotheliomas of the pleural and peritoneal cavities are considered pathognomonic of exposure to asbestos, although in many patients a history of contact with the mineral cannot be elicited.[62-64] These rare tumors are of particular concern from a public-health standpoint because they are thought to occur in persons who have had either transient or indirect exposure to asbestos.[65-67] The development of mesotheliomas as a consequence of casual exposure, however, is an uncommon event. On the other hand, the prevalence of the tumor in workers who have had heavy exposure over extended periods is about 2 to 3 per cent and has been reported to approach 10 per cent.[68,69] It is difficult to determine how often mesotheliomas actually occur, because the latency period is usually 20 years or longer and can often be as long as 40 to 50 years. Some suggest that an epidemic of mesotheliomas will appear in the late decades of this century, consequent to the exposure of large numbers of workers during World War II.

The pathogenesis of the pleural lesions associated with exposure to asbestos is not known, but it is a topic of considerable contemporary interest. Fibrosis of the visceral pleura, plaques of the parietal pleura, and mesothelioma probably develop by different mechanisms, although a rigorous defense of this conclusion would be difficult. As mentioned above, asbestos is deposited preferentially in the periphery of the lung after inhalation. It penetrates the visceral pleura and is carried in the pulmonary lymphatics to the pleural surface. One is tempted to attribute the fibrous lesions on the visceral pleura to irritation by the physical presence of fibers on or near the surface. This mechanism might also explain the occurrence of plaques in the parietal pleura. Alternatively, the lesions may represent an organized fibrinous exudate resulting from the physical movement of the lungs against the pleural surface of the thorax.

However, these hypotheses are not fully consistent with the pathological observations. For example, plaques are often found without fibrosis of the visceral pleura or adhesions between the pleural surfaces. In addition, the lesions are localized and do not occur in the apexes or in the costophrenic angles. The pathogenesis of the lesions cannot be defined at present, in part because plaques occur only in human beings and experimental models have not been developed.

Experimental studies by Stanton et al.[70,71] provide an intriguing basis for speculation about the pathogenesis of mesothelioma. The dimensions of the fiber, but not the chemical composition, were found to be the critical determinant affecting the development of tumors in rats. Long, thin fibers of a variety of types proved carcinogenic when introduced into the pleural space, whereas short fibers and those with a relatively broad diameter failed to induce mesotheliomas. These findings are consistent with epidemiologic observations documenting the relatively common occurrence of tumors in populations exposed to grades of crocidolite consisting predominantly of long, thin fibers and the rarity of tumors in persons exposed to the comparatively blunt, shorter fibers of amosite and anthophyllite.[65,72-74] A fibrous zeolite, erionite, has recently been associated with the occurrence of pleural fibrosis and mesothelioma in a rural area of Turkey where commercial mining of asbestos does not occur.[61] Since the fibers of this mineral do not possess the chemical properties of asbestos but are morphologically similar to crocidolite fibers, the observation is consistent with the experimental findings of Stanton and his associates.[70,71]

The basis for the development of mesotheliomas in the peritoneum is uncertain. Presumably, fibers of asbestos in the lungs are transported in lymphatics to the abdomen, where they have been recovered from lymph nodes and other organs.[75,76] Asbestos is also transported across the mucosa of the gut after ingestion.[77,78] Whatever the mechanism for entry of asbestos into the abdomen, it is assumed that the pathogenesis of the tumors in the peritoneal and pleural cavities is similar. Peritoneal mesotheliomas occur only in persons exposed to amphibole asbestos. The gradual disintegration of chrysotile in tissue may account for the relatively uncommon occurrence of mesotheliomas of both the pleural and peritoneal cavities in persons exposed exclusively to chrysotile.[79]

The mechanism of malignant transformation of mesothelial tissues is obscure. Surprisingly little experimental information has accumulated, although there is reason to believe that the lesions may be comparable to the foreign-body sarcomas induced subcutaneously in animals by sheets of plastic, glass, and metal. The cell of origin is not certain, since some tumors are made up of malignant serosal cells, whereas others have the histologic features of fibrosarcoma. Mesothelial cells phagocytize asbestos[80] and proliferate when exposed to asbestos in vitro,[81] but malignant transformation has not been demonstrated after exposure of cultured mesothelial cells to asbestos. Cocarcinogenic substances and cigarette smoke do not appear to be pathogenetic factors in vivo.

Bronchogenic Carcinoma

Epidemiologic studies have documented an association between bronchogenic carcinoma and occupational exposure to asbestos.[82-87] The prevalence of tumors is higher in persons working with the finished products (such as insulators) than in miners and millers. The severity of the pulmonary parenchymal fibrosis correlates with an increase in the number of neoplasms.[88,89] However, the incidence of tumors is also increased in asbestos workers who lack radiologic evidence of asbestosis.

Some controversy exists over the most common histologic type of tumor, but among persons with asbestosis, adenocarcinomas predominate.[90,91] The lesions tend to occur more frequently in the lower lobes in conjunction with severe degrees of fibrosis.[90] Atypical hyperplasia of bronchiolar epithelium and multifocal adenocarcinomas are often found in these sites.

A linear dose-response relation between the cumulative dosage of asbestos and the development of bronchogenic carcinoma has been reported in miners and millers of chrysotile in Canada[84] and factory workers in the United Kingdom.[85] In the former study, those at greatest risk were exposed to concentrations of asbestos in the air that were higher than the current regulations of the United States Occupational Safety and Health Administration permit. A higher carcinogenic potential for crocidolite than for chrysotile has been suggested by studies of occupational groups exposed to either type of asbestos or to the two in combination.[92] Mortality among chrysotile workers is increased 2.4-fold, whereas it is five times higher than normal among miners of both chrysotile and crocidolite.

Surveys of the smoking habits of insulators,[93] factory workers,[94,95] and miners and millers[96] have consistently shown that bronchogenic carcinoma is uncommon in those who do not smoke. Whereas there is only a slight increase in the prevalence of lung cancer among nonsmokers, heavy users of cigarettes (those smoking more than 20 per day) have an 80-fold to 90-fold greater predisposition to cancer of the lung.[93-96] Thus, the combined effects of asbestos and smoking appear to be multiplicative rather than additive.[97]

What is the mechanism of asbestos-induced carcinogenesis in the respiratory tract? A consideration of contemporary concepts of neoplastic transformation is appropriate in developing an answer to this question. As initially recognized by Berenblum, carcinogenesis is a sequence of events that can be divided into steps of initiation and promotion.[98] An initiator interacts with the DNA of the target cell — an event that can result in malignant change. The carcinogen either acts directly with the DNA of the cell or requires metabolic activation by cellular enzymes. A promoter is generally neither mutagenic nor carcinogenic, although it is required if the neoplasm is to develop. For example, if the skin of a mouse is painted with a small amount of a chemical carcinogen, such as a polycyclic aromatic hydrocarbon, tumors fail to develop unless a phorbol ester is subsequently applied to the site. Promoting substances cause cellular division and proliferation as well as biochemical changes in the cell that appear to be essential for neoplastic transformation.[99]

Although epidemiologic data link exposure to asbestos with bronchogenic carcinoma in human beings, the precise role of the mineral in the process has yet to be defined. Since asbestos is not a potent mutagen[100] and inconsistently causes chromosomal aberrations in cells,[101-103] a mode of action comparable to that of a classic chemical carcinogen is unlikely. It therefore seems more plausible to suggest that asbestos increases the susceptibility of epithelial cells of the bronchi and their branches to transformation by carcinogens in the environment.

What biologic mechanisms account for the synergistic carcinogenic effects of asbestos and cigarette smoke in the respiratory tract? A plausible hypothetical construct should be consistent with the apparent lack of a threshold in human beings and the occurrence of neoplasms in the absence of appreciable degrees of pulmonary asbestosis.

Asbestos has many of the properties of classic tumor promoters, such as the phorbol esters.[104] Proliferation and squamous metaplasia are induced in the respiratory mucosa of rodents in vitro.[105] Asbestos interacts with the membranes of cells[106,107] and induces the synthesis of the polyamines that accompany cell division.[108] Since cigarette smoke also contains a host of substances with promoter effects, the inhalants may act in either an additive or a synergistic fashion to enhance the susceptibility of the respiratory mucosa to carcinogens.

However, alternative mechanisms are worthy of consideration. Asbestos can be phagocytized by the bronchial epithelium and can be transported intracellularly both free in the cytoplasm and in phagolysosomes.[20] These fibers may serve as a physical carrier of the carcinogens in cigarette smoke to the basal cell, the presumptive progenitor of the neoplasms. Transfer of polycyclic aromatic hydrocarbons to and through biologic membranes occurs promptly and efficiently when the hydrocarbon is adsorbed to asbestos.[109] Thereafter, the hydrocarbons are converted by microsomal mixed-function oxidases to biologically active epoxides and diolepoxides, which can interact with the DNA of basal cells.[110] Another (but less attractive) hypothesis involves the alveolar macrophage, which phagocytizes asbestos in the airways and possesses the enzymatic capacity to convert polycyclic hydrocarbons to active metabolites.[111] At present, the mechanism of asbestos-associated carcinogenesis is unclear, although the mineral appears to act like a classic tumor promoter. The fibrous nature of asbestos is critical, since exposure to nonfibrous oxides of silicon (for example, quartz) and a variety of silicates is not associated with an increased risk of bronchogenic carcinoma in human beings.

Cancers of the Digestive System and Other Organs

Asbestos is implicated in the causation of cancer in the upper and lower gastrointestinal tract and the kidney.[112-113] Oropharyngeal and esophageal tumors occur more frequently in asbestos workers who smoke, whereas a direct relation between smoking and the development of carcinoma of the large intestine and the kidney has not been established.

Selikoff and Hammond[114] and Elmes and Simpson[115] have reported a statistically significant twofold to threefold increase in the prevalence of tumors of the digestive tract in insulators, factory workers, and shipyard employees. Other surveys have either demonstrated a smaller increase or failed to establish an association between exposure to asbestos and neoplasms in this system.[116] We believe that the evidence must be assessed cautiously because the associations thus far reported are relatively weak. Since death certificates are used to obtain data in most studies, it is possible that peritoneal mesotheliomas have been confused with metastatic carcinomas of gastrointestinal-tract origin.

The general population is exposed to small amounts of asbestos in drinking water, beverages, food, drugs, and agricultural products. Potable water often contains mineral fibers that are presumably derived from geologic deposits and refuse dumps. The finding of fibers of amphibole asbestos in the drinking water of Duluth, Minn., resulting from the disposal of taconite tailings into Lake Superior,[117] prompted investigations to determine the concentration and characteristics of mineral fibers in water supplies throughout the United States. Fibers with the properties of both serpentine and amphibole asbestos were found in over half the samples of water studied (Table 2). Thus, many Americans consume water containing asbestos-like minerals.

Table 2. Concentrations of Asbestos-Like Mineral Fibers in the Water Supplies of Selected but Representative Communities in the United States.*

City	Concentration
	no. of fibers >5 μm/liter
Atlanta	5.75
Boston	3.98
Duluth	1.72
Dallas	0
Kansas City, Mo.	0.07
New York	0
Philadelphia	16.95
San Francisco	0.60
Seattle	0.85

*Prepared from Table 1.2 in Levine.

Mineral fibers have been detected in the urine of residents of Duluth in numbers corresponding to the concentration of asbestos in drinking water.[118] Interestingly enough, fibers have been found in the glomeruli and tubules of rats exposed in inhalation chambers to synthetic fibers.[119] These observations suggest that asbestos migrates to the kidney after clearance from both the gastrointestinal and respiratory tracts. Whether this has an influence on the occurrence of tumors in the gastrointestinal tract is unknown.

When fed to laboratory animals, asbestos interacts with the mucosa of the gut.[78] Fibers enter cells of the mucosa and prove cytotoxic.[120] The experimental evidence strongly suggests that ingested asbestos is disseminated to abdominal organs by the lymphatics and blood vessels. This conclusion is supported by postmortem studies of occupationally exposed persons; these studies have demonstrated asbestos bodies and uncoated fibers in most major organs.[26]

How does the ingestion of asbestos induce gastrointestinal carcinomas in human beings? In efforts to address this question, rodents were fed large amounts of asbestos over extended periods. With one exception,[121] these studies failed to demonstrate an increase in the prevalence of tumors in the gut.[122-124] The possible synergistic effects of asbestos on the induction of intestinal neoplasms by chemical carcinogens has also been examined.[125] Intragastric administration of asbestos failed to augment tumor development in rodents fed azoxymethane, a recognized intestinal carcinogen.

The carcinogenic potential of asbestos in the gastrointestinal tract appears to be low. The pathogenetic basis for the purported increase in the prevalence of carcinomas in certain occupational groups remains to be established.

PATHOGENIC POTENTIAL OF ASBESTOS TYPES

As emphasized above, asbestos is not one but a family of fibrous minerals, each of which has distinctive physical and chemical characteristics. Minerals from various parts of the world and geologic formations often have dissimilar physical properties, even though they are classified under a specific mineralogic type. These differences are relevant to our understanding of the effects of asbestos on health, since the characteristics of the fiber have been fully defined in only a few epidemiologic and experimental studies. The problem of evaluating the effects of different types of asbestos on health is compounded by the common practice of custom blending of various minerals for specific industrial applications and the use of one type and then another, depending on availability and conditions of the market.

Since the serpentine chrysotile is used extensively in industry today, it is important to ask whether its pathogenic importance is comparable to that of the amphiboles crocidolite and amosite. These latter minerals are of historical importance, particularly since they were used widely during and immediately after World War II and are probably responsible for a substantial proportion of the disease occurring today.

Much current debate centers around the question of whether all types of asbestos possess the capacity to induce mesothelioma. Experiments in animals yield an affirmative answer, but the results of this work may not be applicable to human beings, since pathogenic potential and intrapulmonary transport of fibers are independent considerations. Of all the types, crocidolite is clearly the most strongly associated with the occurrence of the tumor. But there are interesting differences in prevalence, related to the physical character of this fiber type. For example, in Northwest Cape, South Africa, and western Australia, mesotheliomas occur commonly in persons with occupational or casual exposure to crocidolite.[65] The mineral mined in these regions is composed of relatively long, thin fibers. In contrast, mesotheliomas are rare in the Transvaal of South Africa, where the crocidolite fibers are much coarser.

Another amphibole, amosite, is associated sporadically with mesothelioma, whereas the tumor rarely if ever occurs in workers exposed to anthophyllite. Both these latter types are made up of relatively short, blunt fibers. A number of studies have been conducted in miners and millers in Quebec and Italy, where the serpentine chrysotile is extracted.[71,126] Although the results are debated, the bulk of the evidence indicates that chrysotile is not an important cause of mesothelioma in these workers.

However, the data from certain occupational groups, such as workers in the textile industry and insulators who are exposed predominantly but not exclusively to chrysotile, are not as definitive. The risk appears to increase as the mineral is processed or when dust concentrations cannot be evaluated critically. Unfortunately, most epidemiologic studies concerned with this important question are clouded by uncertainty because of the prolonged latency period of mesotheliomas. Considerable effort has focused on determining whether the various types of asbestos differ in their capacity to induce bronchogenic carcinoma and fibrosis of the lung. Unfortunately, there is no good answer to these questions at present, since dose-related differences in the prevalence of disease have not been established.

REGULATORY CONSIDERATIONS

No topic is more complex and subject to controversy than the establishment of criteria on which to base standards for air quality in the work place. Regulations are exceptionally difficult to develop, because it is necessary to use data on morbidity and mortality documenting disease retrospectively in members of occupational groups who have had heavy exposure either in the remote past or over a lifetime. The difficulties are compounded by the long latency period of asbestosis and the asbestos-associated cancers.

Although recommendations for levels of asbestos in the air of occupational settings in this country were formulated in the 1940s, it was not until 1970 that federal regulations were promulgated as a result of the passage of the Occupational Safety and Health Act and the Clean Air Act. The initial standard was based on the light microscopical count of fibers of a length of 5 μm, collected by mechanical means. A concentration of five fibers per cubic centimeter of air, averaged over an eight-hour period, was deemed permissible, with stipulations for transient excesses above that concentration. In 1976 the contemporary standard of two fibers per cubic centimeter was established, and more recently a level of 0.5 fiber per cubic centimeter has been proposed.

Is the current limit of two fibers per cubic centimeter sufficiently rigorous to prevent disease in the future? Is it appropriate to base regulation exclusively on determinations of fibers of >5 μm when the bulk of the dust in air consists of fibers of a shorter length? Because standards are based on extrapolations from data accumulated among workers exposed to relatively heavy concentrations of dust in the past, predictions must be based on analyses that assume that there are no thresholds below which the disease fails to occur. Within the ranges usually found in the occupational setting, there appears to be linearity in the dose-response relation, at least with regard to bronchogenic carcinoma. However, the likelihood that cancer will occur is influenced substantially by cigarette smoking, since the risk in the nonsmoker who has heavy exposure to asbestos is increased only a few fold. Thus, the risk for the nonsmoking asbestos worker is substantially lower than the risk for a member of the general population who smokes two or three packs of cigarettes each day.

The conclusion that asbestosis fails to develop below a certain threshold dosage is based on physical examinations and radiologic studies of workers and not on pathological examinations. By these criteria, it is probably impossible to be certain whether a fibrotic lesion in the lung is due to asbestos. With mesothelioma, the data are more controversial. Although a dose-response relation appears to exist, the threshold may be determined by the life span of the person exposed, because the latency period for these tumors is protracted. Since the problem cannot be answered with contemporary epidemiologic and experimental approaches, it must be resolved by practical rather than theoretical considerations.

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THE CITY OF NEW YORK

DEPARTMENT OF HEALTH

RECEIVED
AUG 10 1983
BRUCE W. KARRH

TEL. NO.

ROOM NO.

DATE August 4, 1983

TO Bruce Karrh, M.D.

FROM Stephen Schultz, M.D. SS

SUBJECT Brookfield Health Survey

Enclosed are the two preliminary reports which Dr. Sencer requested to be sent to you.

SS:jh
enc. (2)

70 A. Z. 175M (69)

THE CITY OF NEW YORK
DEPARTMENT OF HEALTH

BUREAU OF PREVENTABLE DISEASES
N.Y.C. DEPT. OF HEALTH
125 WORTH STREET
NEW YORK, N. Y. 10013
BOX 32

11/17/82

APPENDIX C

NEW YORK DEPARTMENT OF HEALTH ENVIRONMENTAL AND EPIDEMIOLOGICAL SURVEY OF THE BROOKFIELD LANDFILL AND SURROUNDING COMMUNITY - PRELIMINARY FINDINGS

In January 1982, sewer line construction through the inactive Brookfield landfill caused the neighboring community to complain of odors and increased health problems. Based upon a small series of non-random interviews, it was estimated that many people complained of nausea, wretching, vomiting, burning eyes, sore throat as well as respiratory complaints. At the same time there was public disclosure of past illegal disposal of toxic wastes at this landfill.

The Department of Health (DOH) initiated an environmental and epidemiological survey of the landfill and surrounding community. The environmental survey was designed to examine air, water and soil as possible routes of exposure and identify any chemicals which might be an immediate health hazard to the surrounding community. The epidemiological survey was designed to determine whether people living close to the landfill had more medical complaints in the period from January 1982 to July 1982 than people living further from the landfill and whether these medical complaints constituted diagnosed medical illness.

Epidemiological Survey

The area chosen for study was the census tract 146.01 and a comparable census tract 132.02 was selected as the control area (see map 1). The study area was divided into 3 zones. A stratified random samples was drawn from the study and control areas using the Coles reverse directory of listed telephone numbers. Those households with no telephone or no listed telephone were excluded from the survey. 30% of the households with listed telephones were sampled in Zone I while, 15% of those with listed telephones were sampled in Zones II and III. 10% of the households with listed telephones were sampled in the control area. For the control area, randomization was done without reference to census blocks. If any household refused or could not be interviewed, the next household listed was selected as an alternate in the control area and for the study area the next listed phone on that block was chosen.

The questionnaire used in the survey was developed from information gained from informal non-structured interviews with residents of the community as well as suggestions made by the Centers for Disease Control, U.S. Public Health Service. The questionnaire was not pretested nor were the individual questions validated. The questionnaire was administered by DOH nurses and supplemented by a physician and a nurse graduate student. It was planned to validate all health complaints of survey respondents by obtaining a release which would permit review of their medical records by the DOH.

The results of the sampling described yielded 561 households to be interviewed. When compared with census data, it appears that 75% and 78% of the households in the study and control area respectively had listed phones. The interviewing was started in June and completed in 8 weeks. A total of 555 were done, 99 in the control area and 456 in the study area.

	Area Zone	Households	Individuals
	I	153	569
Study	II	123	467
	III	180	641
	Total	456	1667
Control		99	317
	Total	555	1984

When compared to the census, this represents about 20% of the population in Zone I where 30% of the listed phones were sampled and 10% of the population in Zones II and III where 15% of the listed phones were sampled. In the control area where 10% of the listed phones were sampled, about 8% of the total population was sampled.

Of the 555 households interviewed, 285 (51%) were original choices while 270 (49%) were alternate households. These substitutions were made because of refusal to participate or because of inability to reach the original choice. When the original interviews are compared by location, there are increased numbers of non-respondents at greater distances from the landfill. 58% of those in Zone I volunteered when first contacted, while in Zones II and III in the study area, 56% and 49% responded initially. In the control area only 39% of the originally selected households were interviewed. Slightly more calls on the average were required in the control area (3.3) to gain an alternate than in the study area (3.0).

The study area and control area samples were similar to the census data for these census tracts in mean age, sex and race. There were, however, differences in age distribution. Less people in the 25-29 year age group were sampled than expected in the study area. In the 55-59 age group in the control group, more were sampled than expected from the census data.

The comparison of the study sample and the control sample to each other revealed similarities in sex and racial distribution but significant differences in age and family size. The control area residents tended to be older on the average and have a smaller family size. The household in the control area also lived at their current address for a longer time, 12 years versus 9 years for study area households.

In the 6 months period prior to the survey, the people living in the study area reported visiting a doctor or other health care professional more frequently than those living in the control area. 45% for study area residents versus 25% for control area residents gave such a report, a statistically significant difference. The reported visits were for the following complaints: chest or breathing problems, ear, nose or throat problems, stomach complaints, nerve problems including headache and emotional problems. The people in the study area and the control area, however, reported seeing medical professionals with the same frequency for kidney muscle, heart, blood, gynecological and skin complaints.

People living in the study also reported more symptoms since the beginning of 1982 for which they did not see a health care professional when compared to the control. These symptoms included frequent headache, sore throat and nausea.

Those living in the study area reported more allergies than persons living the control area. There was no difference between the two groups in terms of reported bronchitis, sinusitis or asthma.

Individuals in the study area reported more days absent from regular activities than those living in the control area. In the past six months, they reported an average of 2.3 days of absence while the control area residents reported 1.2 days of absence from normal activity.

Comparisons have been made only between the study area households as a whole and the control area households up until this point. If trends are examined within the study area itself, the tendency is for Zone I (nearest the landfill) to show the higher rates in the most of the categories reported. Zone I residents had rates greater then persons in Zones II and III in 13 out of 20 measures for complaints for which health professional visits were made. Out of 23 measures of symptoms experienced in the last 6 months for which health professions consultation was not sought, 16 of these rates were higher for Zone I residents than Zone II or III. If chance alone were at work, these medical events would be expected to be distributed with equal frequency throughout the study area. The trend observed, concentration of reported complaints in the area closest to the landfill is statistically significant.

Less than 20% of the survey respondents who reported their family members as seeing a health provider complied with the request for a medical release.

The responses to the questions addressing perceptions of the respondents in the study area and the control area revealed some significant differences. Those respondents living in the study area were less satisfied with living at their present location. They perceived odors more frequently and believed their health was poorer than those respondents living in the control area. More study group respondents felt their family members' health had changed for the worse since moving to this location in comparison to the control area respondents.

Environmental Sampling

Air at several locations on Brookfield landfill and in the surrounding community was sampled for volatile ogranic chemicals to examine the potential for toxic exposure directly from the landfill. Water infiltrating basements in the community, surface water and leachate seeps on the landfill were sampled for volatile organic chemicals and heavy metals to examine the potential for toxic exposure from leachate. All drinking water on Staten Island originates in the City's upstate reservoir system and therefore would not be a vector for materials from Brookfield landfill.

Private non-potable wells in the vicinity of Brookfield landfill were sampled for volatile organic chemicals and heavy metals to determine if a leachate plume could be identified.

All samples were analyzed by gas chromatography/mass spectrometry in the N.Y. City Department of Health laboratories.

RESULTS

1. Air Sampling

Air samples were taken using charcoal collecting tubes and distilled water impingers.

2. Landfill Air Sampling - A total of 5 air samples were taken at 2 locations in or next to the sewer trench under contruction. In addition, a total of 6 air samples were taken at the 2 sites identified by the N.Y. City Department of Sanitation as possible sites of illegal toxic waste disposal.

Volatile organic chemicals were detected in only 2 of the 5 air samples taken at the sewer trench. A sample at 1 of the trench locations contained chloroform, benzene, toluene, tetrachlorethylene and ethyl ether. Ethyl ether and benzene were the predominant constituents found; the concentrations were 6.5 and 4.5 parts per billion (ppb) in air, respectively. The other chemicals were found at less than 1 ppb. A sample at the other trench location yielded only chloroform (0.03 ppb).

Air samples at only 1 of the sites of possible toxic dumping contained volatile organics. There only was chloroform at 0.01 ppb detected. No volatile organic chemicals were detected at the other site.

3. Neighborhood Air Sampling - A total of 8 air samples were taken at 4 different locations spanning the residential area immediately bordering Brookfield landfill (see map 2). These samples also span Zone I of the epidemiological health survey.

Volatile organic chemicals were detected in the air at only 1 of the 4 locations. Benzene and chloroform were found at or less than 0.02 ppb.

4. Background Air Sampling - A total of 8 air samples were taken at 4 different sites at an average distance of 2.5 miles from the landfill, to detect ambient levels of volatile organic chemicals at locations removed from the landfill.

Volatile organic chemicals were detected at each of the 4 locations. Both chloroform and benzene were found at every location at an average of 0.04 ppb and 0.03 ppb, respectively. Toluene at 0.01 ppb was detected at only 1 location. In all cases, the levels found were greater than or equal to those seen in the neighborhood samples.

II. BASEMENT INFILTRATE SAMPLING

Basement water samples were taken from the sumps in the houses identified by the local residential association as bordering the landfill and having a history of water collecting in the basements. In total 5 houses were identified and sampled.

Only 1 sample had any detectable amount of volatile organic chemicals (2.6 ppb chloroform in water).

3 of the houses were in Zone I and 1 was in Zone III.

III. LANDFILL LEACHATE AND SURFACE WATER

3 water samples were taken in or near the sewer trench under construction through the landfill.

1 sample in the trench yielded xylene and n-propyl benzene at 228.8 and 156.1 ppb, respectively. Toluene and chloroform were also found at less than 3 ppb. One sample of surface water near the trench showed chloroform at less than 1 ppb.

A total of 4 samples were taken from leachate and surface water in the vicinity of the 2 sites of suspected toxic waste dumping.

1 sample contained 330.2 ppb of pentane. The other sites had less than 1 ppb total volatile organic chemicals. Lead, at .04 ppb, was the only metal detected, found in one sample.

IV. WELL WATER SAMPLING

A total of 13 private, non-potable wells were sampled to determine if a plume of groundwater contamination could be detected. With one exception, these wells were neither in the health survey nor control area (see map 1.)

9 wells had detectable levels of volatile organic chemicals. The chemicals included tetrachloroethylene, 1,1,1, trichloroethane, chloroform, benzene, and toluene. All of these chemicals are commonly found in contaminated wells in Jamaica, Queens and are widely used both domestically and commercially. 6 wells had less than 5.0 ppb, 1 well had 32 ppb and 2 wells had greater than 300 ppb total volatile organic chemicals. Tetrachloroethylene was the chemical present in the 2 highly contaminated wells, both of which were 2.4 miles from the landfill. Heavy metals were not detected in any of the samples. The location of these wells, and the distribution of contaminant concentrations gives no indication of a plume of contamination in the area of these wells.

CONCLUSION

No route of exposure to contaminants could be identified by these environmental sampling results; however, the epidemiological survey did record increased reported medical utilization. Further study will clarify whether the increased medical utilization represents actual illness and whether associated agent(s) can be identified with emissions from the landfill.

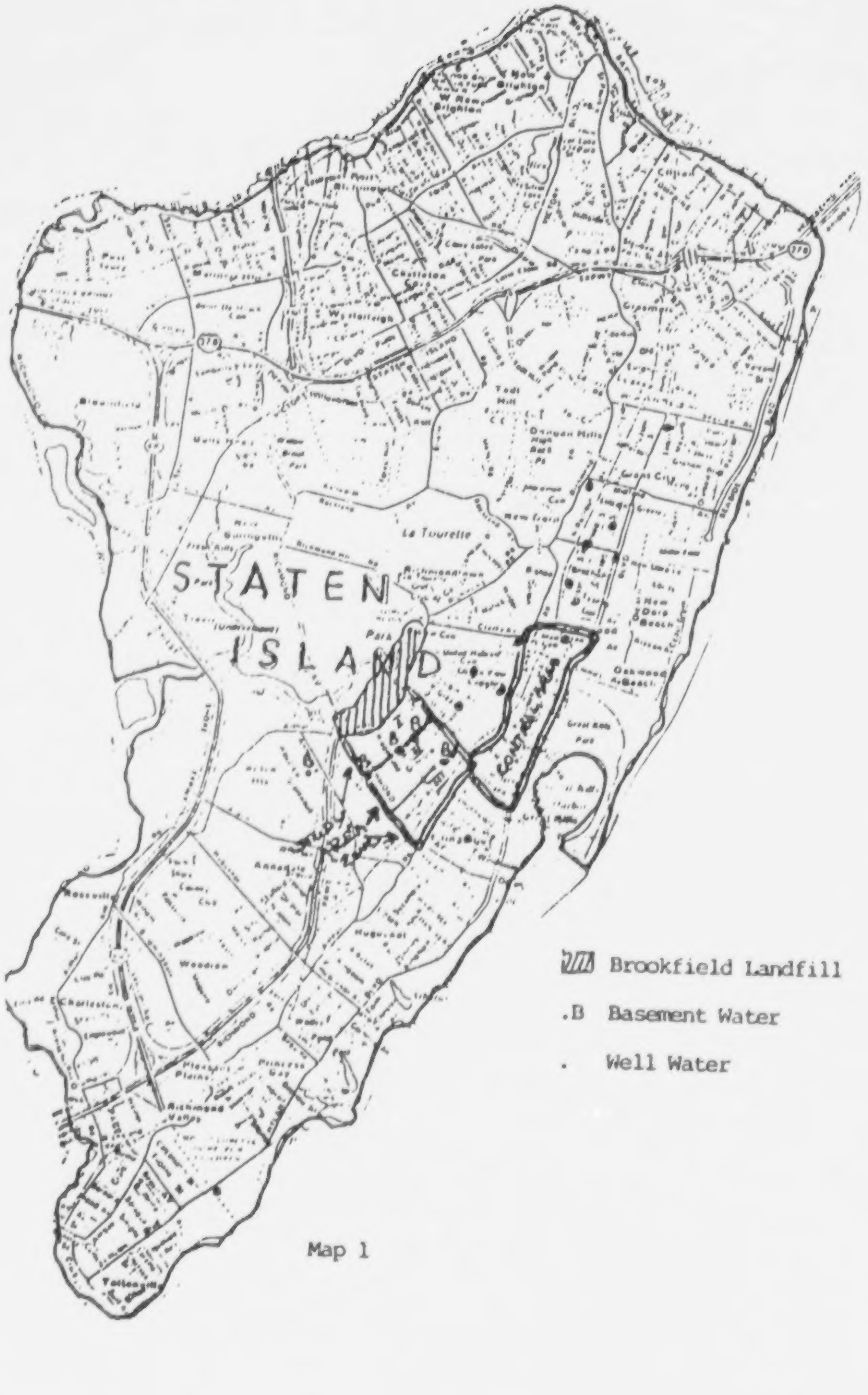
CONTINUING ACTIONS

1. Physicians will be contacted for verification and documentation of medical illness in the surveyed residents who provided releases.

2. Health survey participants who did not provide releases initially will be recontacted and requested to cooperate.

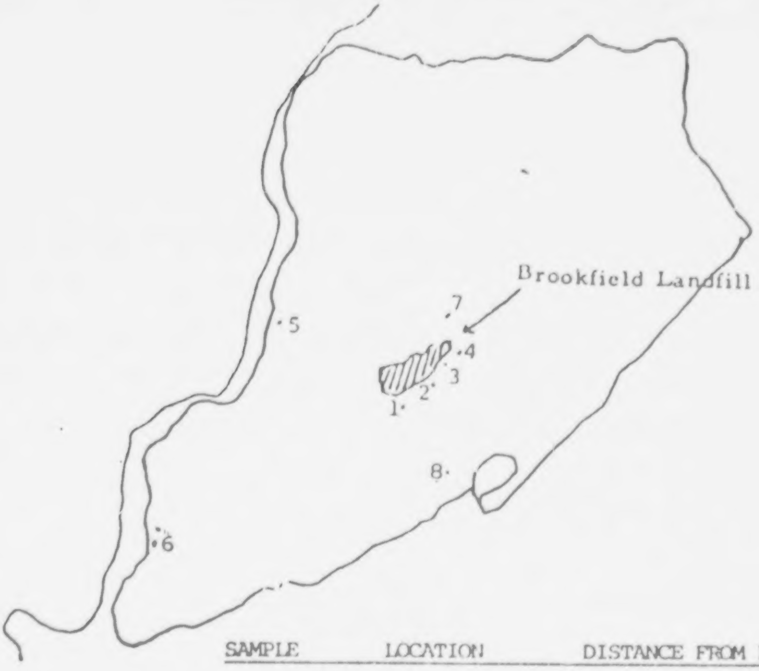
3. A sample of children within the community near the landfill will be tested for sensitivity to specific allergens and for pulmonary function.

4. A program of air sampling will be instituted in the spring which will involve a greater number of samples per location for a longer sampling time.


STATEN ISLAND
La Tourette
▨ Brookfield Landfill
.B Basement Water
. Well Water

Map 1

NEIGHBORHOOD AND BACKGROUND AIR SAMPLING



SAMPLE	LOCATION	DISTANCE FROM LANDFILL
1	Ridgewood Ave and Sweetbrook Road	1530 feet
2	Elverton Ave. off Arthur Kill Road	175 feet
3	Tanglewood Drive off Arthur Kill Road	125 feet
4	Clarke Street off Arthur Kill Road	175 feet
5	End of Victory Blvd. (New Jersey side)	2.7 miles
6	End of Androvette St. (New Jersey side)	4.8 miles
7	Richmond Hill Road and Forest Hill Road	.75 miles
8	Hylan Blvd. and Nelson Ave.	1.6 miles

Map 2

PRELIMINARY REPORT

Brookfield Health Survey Report - Phase II

The first phase of the Brookfield Health Survey showed that in the first six months of 1982, 35.9% of the study group and 19.2% of the control group reported visits to physicians with complaints referrable to the following physical systems: eye-ear-nose and throat, chest, cardiovascular, gastrointestinal, neurological and psychological. (see Table 1) These complaints had shown a statistically significant difference between the study and the control groups and were chosen for intensive follow-up. (Hospitalization rates for this selected group varied little between the study zones and control area and was approximately 7%)

The purpose of the second phase of the Brookfield Health Survey was to confirm the residents' reports of illness and to determine the medical diagnoses and the frequency of visits.

For any individual with a complaint in one or more of the above categories, his or her medical records were sought. There were 659 such persons.(see Table 1)

Over a 4 month period numerous letters, telephone calls and home visits by Health Department staff resulted in 419 persons (63%) ultimately providing releases to have their physican and/or hospital records reviewed. In zone 1, 70% of those who were sought gave releases,in zone 2, 65.7%, in zone 3, 58.4% and in the control area, 48.4% . (see map 1)

With these releases secured, the 144 doctors who cared for the 419 patients were contacted. A questionnaire relating to the patients' visits and medical diagnoses for those visits was to be filled out either by the physician, his staff or Health Department personnel. (see Appendix 1)

Overall, the questionnaires for 328 individuals (79%) were completed. The percentage of completions was approximately the same for the study zones and control area.

A visit to the named physician was documented in the correct time period for 218 persons or 2/3 of the total. (This period was between January 15, 1982 and the variable date of the Brookfield Health Survey interview, sometime in the summer of 1982.) In the study zones 1,2,3 and the control area, 66%, 69%, 68% and 56%, respectively, of the survey respondents' visits were documented in the records of the physician named. This shows that the rate of confirmation of physician visits was somewhat less in the control area than in the study area. This supports the finding in phase I that the percentage of respondents reporting visits to a physician for a condition of interest was greater in the study zones than in the control area (35.9% vs 19.2%).

It should be kept in mind that not all physician visits were reported on the survey. An indication of this under-reporting was obtained by examining records of 63 family members who did not report a visit for a condition of interest. When the physician records of these people were reviewed 2/3 did have a visit in the correct time period for a condition of interest. It should be noted that these people were not chosen by random selection and are not counted in further analysis.

Of 218 individuals who had a documented visit to a physician in the first six months of 1982, 204 were in the study zones and 14 in the control area (see Table 2). For purposes of comparison between the study area and the control area the small size of the control group presents a problem.

The difference in average age between the study group and the control group was pointed out in the preliminary report. This difference persists in this phase of the study and is even more pronounced. (see Table 2) The zone 1 individuals average 19.8 years while the control group average 34.6 years. This disparity is accounted for by the larger number of children in zone 1.

For the 218 persons with a documented visit in the time period the average number of total visits to a physician varied from 2.0 in the control area to 2.5 in zone 1. This result shows a slightly higher average for the study area.

The average number of diagnoses for all conditions varies little among the study zones and control area. (Table 2)

Table 3 presents estimated rates of illness for the selected body systems as measured by the number of diagnoses of interest per 100 residents surveyed. The rate of illness is higher for the study group as a whole when compared to the control group; and zone 1, closest to the landfill, has the highest rate of all the zones.

The results of this phase of the Brookfield Health Survey validate the earlier phase I findings that the percentage of residents that visited physicians was higher in the study zones than the control area . It may now also be said that the estimated rate of illness was higher for the study group than for the control group.

The illnesses encountered however, do not seem to have been prolonged or complicated. This can be inferred from the following:

1) Once a visit was made to a physician, the study area residents had a slightly higher number of total doctor visits on the average than the control area residents ;these visits resulted in a similar average number of total diagnoses.
2) Among the persons with selected complaints the rate of hospitalization was similar for study zones and control area.

PRELIMINARY REPORT

BROOKFIELD HEALTH SURVEY PHASE II

Table 1

Medical Record Follow - Up

Study Area	Total Surveyed	Individuals Sought		Individual's Releases Obtained		Physician Quest. Completed		Individuals with Visit in Time Period	
		Number	% of Total Surveyed	Number	% of Total Sought	Number	% of R. Obt.	Number	% of Total Compl. Quest.
Zone 1	578	243	42.0	170	70.0	136	80.0	90	66.2
Zone 2	448	169	37.7	111	65.7	84	75.7	58	69.0
Zone 3	635	185	29.1	108	58.4	83	76.9	56	67.5
Control	323	62	19.2	30	48.4	25	83.3	14	56.0
Total	1984	659	33.2	419	63.6	328	78.3	218	66.5

NEW YORK CITY DEPARTMENT OF HEALTH

6/29/83

PRELIMINARY REPORT

BROOKFIELD HEALTH SURVEY PHASE II

Table 2

Medical Activity of 218 Residents With Documented Visits

To Physicians In First Six Months of 1982

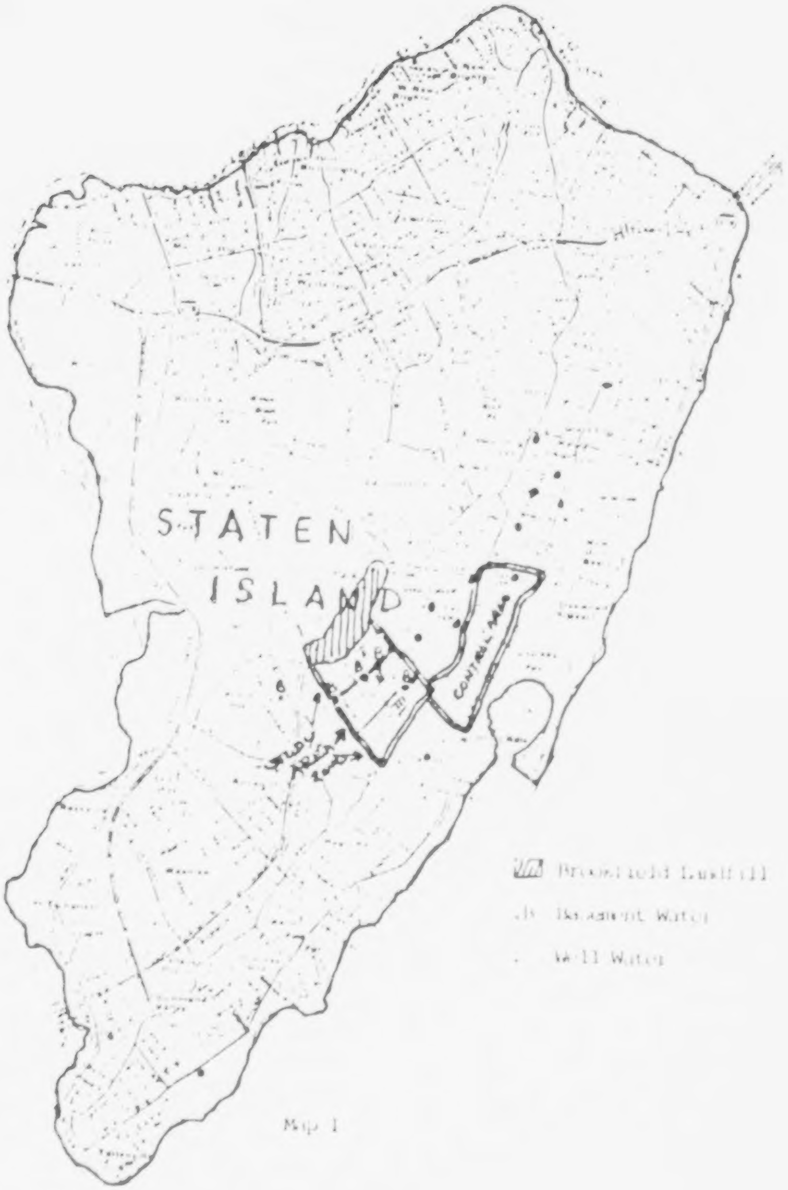
Study Area	Number of Persons with a Documented Physician Visit in Time Period	Average Age In Years	Average Total Visits	Average Number of Diagnoses
Zone 1	90	19.8	2.5	2.2
Zone 2	58	23.4	2.2	1.9
Zone 3	56	24.9	2.4	2.0
Control	14	34.6	2.0	2.0
Total	218	23.0	2.4	2.1

PRELIMINARY REPORT

BROOKFIELD HEALTH SURVEY PHASE II

Table 3

Study Area	Estimated Number of Diagnoses of Interest Per 100 persons surveyed
Zone 1	46.4
Zone 2	36.8
Zone 3	27.0
Control	13.8
Total	33.7


STATEN ISLAND
CONTROL AREA
▨ Brookfield Landfill
Basement Water
Well Water

Map I

APPENDIX 1

NEW YORK CITY DEPARTMENT OF HEALTH–BROOKFIELD LANDFILL STUDY, PHASE II

PHYSICIAN QUESTIONNAIRE

Patient ______________________ Dr ______________________ H.H. # ________

Summary of visits by complaint and diagnosis: 12/15/81 to 8/31/82

Visit date: ___/___/___
Complaints (check categories):

☐ CHEST ☐ ENT ☐ GI ☐ GU
☐ MUSC/SKEL ☐ NEUR ☐ C/V ☐ HEM
☐ GYN ☐ DERM ☐ PSY ☐ ENDOC
Other ______________________

Diagnoses	History prior to 1/15/82? yes	no		Persisted after 8/31/82? yes	no	unknown
#1 ______						
#2 ______						
#3 ______						

Visit date: ___/___/___
Complaints (check categories):

☐ CHEST ☐ ENT ☐ GI ☐ GU
☐ MUSC/SKEL ☐ NEUR ☐ C/V ☐ HEM
☐ GYN ☐ DERM ☐ PSY ☐ ENDOC
Other ______________________

Diagnoses	History prior to 1/15/82? yes	no		Persisted after 8/31/82? yes	no	unknown
#1 ______						
#2 ______						
#3 ______						

Visit date: ___/___/___
Complaints (check categories):

☐ CHEST ☐ ENT ☐ GI ☐ GU
☐ MUSC/SKEL ☐ NEUR ☐ C/V ☐ HEM
☐ GYN ☐ DERM ☐ PSY ☐ ENDOC
Other ______________________

Diagnoses	History prior to 1/15/82? yes	no		Persisted after 8/31/82? yes	no	unknown
#1 ______						
#2 ______						
#3 ______						

Visit date: ___/___/___
Complaints (check categories):

☐ CHEST ☐ ENT ☐ GI ☐ GU
☐ MUSC/SKEL ☐ NEUR ☐ C/V ☐ HEM
☐ GYN ☐ DERM ☐ PSY ☐ ENDOC
Other ______________________

Diagnoses	History prior to 1/15/82? yes	no		Persisted after 8/31/82? yes	no	unknown
#1 ______						
#2 ______						
#3 ______						

Visit date: ___/___/___
Complaints (check categories):

☐ CHEST ☐ ENT ☐ GI ☐ GU
☐ MUSC/SKEL ☐ NEUR ☐ C/V ☐ HEM
☐ GYN ☐ DERM ☐ PSY ☐ ENDOC
Other ______________________

Diagnoses	History prior to 1/15/82? yes	no		Persisted after 8/31/82? yes	no	unknown
#1 ______						
#2 ______						
#3 ______						

[illegible] Dr. [illegible] N.Y.C. Department of Health, [illegible] Street, N.Y., N.Y. [illegible]

A CRITIQUE OF THE EPIDEMIOLOGICAL SURVEY OF THE BROOKFIELD LANDFILL AND THE SURROUNDING COMMUNITY

The objective of this survey was to find out whether people living close to the landfill had a greater incidence of illness than those living further away. The study area, which is next to the landfill, was divided into three zones. Zone I is contiguous to the area, and Zones II and III further away. An area still further away was designated the control area.

In the first phase of the study, members of a sample of households were interviewed to find out which members of the household had medical complaints during the previous six months. In the second phase of the study, the personal physicians of those who had complaints were contacted to confirm the reports of illness and to obtain diagnoses.

The major findings of the survey were as follows:

- During the six month study period, 35.9 percent of the study group and 19.2 percent of the control group reported visits to physicians.
- Rates of medical complaints were highest in Zone I, the area nearest the landfill.
- The rates of diagnoses, as determined by contacts with physicians, were inversely related to distance from the landfill.
- Reported illnesses were not prolonged or complicated.
- Hospitalization rates were similar in the study and control areas.

Because of several important limitations of the study, these findings cannot be used to draw any firm conclusions regarding the effects of substances in the landfill on the health of persons who live in the surrounding areas. These limitations can be categorized as follows:

- Potential biases arising from high nonparticipation and nonresponse rates.
- Potential biases in the administration of the interview questionnaire.
- Excessive reliance on subjectively reported, rather than an objectively determined measures of incidence of illness.
- Possibly non-comparability between the study and control areas.
- Inappropriate use of tests of significance.

Each of these limitations is discussed below.

Nonparticipation and nonresponse rates

The households to be interviewed were selected by a stratified random sampling procedure, but high refusal rates were encountered in the households originally selected, ie, 42% in Zone I, 44% in Zone II, 51% in Zone III, and 61% in the control area. As a result, alternate households had to be selected until the required sample size was attained. The use of such a large proportion of alternates may not only have seriously disturbed the randomness of the sampling design, but may also have resulted in additional bias in the sample because of possible differences in the health status between those who agreed to participate and those who refused.

Another potential source of bias is the higher participation rates among persons living close to the landfill than among those living further away, as indicated above. Persons living close to the landfill may believe their symptoms and medical problems are caused by their proximity to the landfill. As a result, they would be more motivated than persons living in more distant areas to participate in the survey and report their symptoms. As a result, a person with a given symptom who lives next to the landfill would be more likely to be included in the survey than would a person with the same symptom living at a greater distance from the landfill.

Another serious potential for bias arose from the refusal of 36.4% of the 659 persons with medical complaints to give releases that would allow the investigators to review their physician and/or hospital records. In addition, of the 419 who gave releases, 328 questionnaires relating to their diagnoses were completed by their physicians. This represents a nonresponse rate among the physicians of 21.7%. Moreover, only 218 of the 328 visited a physician during the study period. Thus, of the 659 persons reporting complaints, documentation of physician visits and diagnoses were obtained for only 218, or about 33%.

The interview questionnaire

There were two potential sources of bias in the administration of the interview questionnaire. One is respondent bias and the other is interviewer bias.

Respondent bias could have arisen among persons living in the areas close to the landfill. Having been informed of possible health hazards from substances in the landfill, they would probably have a more heightened awareness of symptoms than would persons living further away. Also, they would probably attach greater significance to pain and discomfort, and thus would more likely report them to the interviewer.

Interviewer bias could also have affected responses to the questionnaire. Since the interviewers obviously knew how far each respondent lived from the landfill, they may have, consciously or unconsciously, administered the questionnaire in ways that might have tended to elicit more positive responses among persons living close to the landfill.

In properly conducted epidemiological studies, where personal interviews are used to obtain data, attempts are made, where feasible, to conduct the interviews "blind"; that is, where the interviewer does not know which respondent is exposed or not exposed to the substance or substances under investigation. Even objective measurements, such as blood pressures and electrocardiograms, taken and interpreted by physicians, are done blind in properly conducted studies.

Blind interviews were probably not feasible in this study, but this potentially important source of bias should have been mentioned in the report and qualified the inferences drawn from the data.

Reliance on subjectively reported illnesses

In the first phase of the survey, interviews were conducted to identify persons with medical complaints. In the second phase, physicians were contacted to verify the complaints and obtain diagnoses.

The use of information from physicians, however, does not remove the biases that could have arisen by identifying ill persons through subjective interpretations of symptoms by the survey respondents. For reasons discussed above, the closer a person lived to the landfill, the more likely it was that he or she would participate and report symptoms. Getting information from physicians from a biased selection of patients does not remove the bias.

In this regard, it is interesting that the complaints for which there was a higher rate in the study area than in the control area were predominantly functional disorders, such as breathing problems, stomach complaints, headaches, and emotional disorders. Since these may be psychogenic in origin, their etiology may include an hysterical component, generated, perhaps, by anxiety aroused by reports of health effects from the landfill.

On the other hand, study and control areas did not differ in indices of illness derived from objective measurements or observations. One of these was the incidence of organic diseases, such as kidney, muscle, heart, blood, and gynecological disorders. The other was the frequency of hospitalization, which was about 7% in each area.

Comparability of the study and control areas

Samples drawn from the study and control areas were compared with respect to age, sex, and race, but no comparisons were made regarding socioeconomic characteristics of the areas. It is well known that, for many diseases and risk factors for diseases, incidence rates are inversely related to socioeconomic status. For this reason, the investigators should have compared the study and control areas with respect to such socioeconomic indicators as median income and educational attainment, and to have taken these factors into account in the interpretation of the data.

Information in the report suggests that the socioeconomic status of the control area was higher than that of the study area. One indicator is the fact that the study area is near the landfill, making it more likely that it would be inhabited by lower income people. Also, it was noted that the control area residents tended to be older and have a smaller family size, two indicators of higher socioeconomic status.

Thus, if in fact the residents of the study area were in a lower socioeconomic level, a higher rate of illnesses would be expected.

Tests of significance

Tests of significance were done when certain rates were compared between the study and control areas. These tests assume random selection of subjects from the population to which inferences are to be drawn. Since randomness was lost because of large nonparticipation rates, these tests cannot properly be used to infer differences between the study and control areas.

Conclusion

This study does not establish any causal relation between substances in the landfill and medical complaints. However, the limitations of this study, as important as they are, do not exclude the possibility that substances from the landfill could have affected the health of residents who live near it.

To determine whether they have been affected would require a different way of measuring the prevalence of illness in each area. It would be preferable to assess the health of each individual in the sample by a health examination given by a physician or other health professional. The information gathered in the survey should also include smoking habits, dietary patterns, alcohol consumption, socioeconomic characteristics, and other potential confounding factors.

If such a survey does establish higher rates of illness in the study area, and if these higher rates cannot be explained by confounding or by biases arising from nonparticipation, the survey would provide suggestive evidence of adverse health effects from substances in the landfill.

Mr. **Molinari.** I would like to turn, if we could, to the problems we heard yesterday related by an attorney who was representing a victim of toxic waste exposure. The lawyer, who apparently handled a number of such matters, narrated the difficulties that are presented to an attorney who has a client who appears to have a very serious problem, occasioned by the workplace. He related to us—and I recall the term—almost insurmountable difficulties in trying to establish with scientific certainty the causal relationship: the fact that, indeed, that condition was caused on the job. And he was talking about a client who had incurred expenses—let's not talk about the personal injury value, but expenses of over $100,000.

He also mentioned that only in the case where there were very severe injuries could any lawyer consider taking on a case, because they are usually on a contingent fee basis. Getting medical testimony is difficult; there aren't that many medical experts apparently available. The way he described it to us, the cost of medical witnesses is about $350 an hour, I think was the figure he said. He found he had to pay dearly for good medical experts.

So we get now, I think, to the heart of the problem: What should a person do who has a severe medical injury, believed to be caused by exposure. Then he goes to a lawyer. I practiced law, and I can tell you that there aren't many lawyers that would be very interested in taking on a case like that because of the difficulties in proving the case.

Yet; we know that there are—in my mind, there is no question—many, many thousands and thousands of people in this country who have suffered and are suffering from this kind of exposure. It was the attorney's suggestion that we in Congress consider changing the laws so that there would be a set of criteria established where, if you met that criteria, you had reached the stage where there is presumptive evidence that the injuries were caused by exposure to that chemical contamination. And then the burden of proof would shift to the defendant to prove that, in fact, the condition was not caused by that exposure.

Having heard testimony for a couple of days—and we will be hearing more—I see that as being a very serious problem. My colleague here, Mr. Sundquist, and I had a brief conversation before where we recognized the enormity of the problem, trying to establish causation, particularly when the industry keeps talking about scientific certainty.

Isn't there a midground that could be reached so that victims don't have to prove with scientific certainty their case? Indeed, if those conditions were in place today we wouldn't be discussing the issue. There would be enough lawyers around that would be jumping at the opportunity to litigate if they had the scientific certainty. But it does not exist. And I don't know how long it will be before we will reach that point in time.

But, in the meantime, there are these people who are, in my opinion, suffering and suffering severely and are entitled to be compensated. It seems to me that there is no other way; that we have to consider changing the law. And we have to reach a reasonable set of criteria at which the plaintiff has established a reasonable case, presumptive evidence.

There are different measures or standards we could talk about as necessary to reach that plateau. But once that plateau is reached, then the burden of proof shifts. The burden shifts to the industry who has the resources, the expertise, and the benefit of all these studies, so as to give that injured party a fair opportunity to seek redress for personal injuries.

I would like to receive your comments, Dr. Karrh, as an industry representative.

Dr. KARRH. All right, sir.

Mr. MOLINARI. I would expect that you are opposed to the concept, but maybe not as opposed as I think, because an early part of your testimony indicates that you do believe that the responsible party should pay. I have heard other representatives of CMA also speak in those terms.

Dr. KARRH. That is our position; that people who are victims, if it is shown they are victims, should be compensated. I don't think we have used the term "scientific certainty." I think earlier in my testimony—I would have to look at the transcript to be sure—I stayed away from that term. I did use the term "scientific probability based upon scientific evaluation of the data that was available." If it is determined with scientific probability that the condition or conditions are related to our actions or disposal practices or operations or whatever, then we certainly should be held responsible for those.

Mr. MOLINARI. What does a lawyer have to prove in that case?

Dr. KARRH. Not being a lawyer or judge, I am sorry; I can't answer that. I can tell you from a scientific standpoint we would like to be able to look at the data available, and from a scientific standpoint determine that there is most probably a causal relationship between the exposure and the result that occurs.

If you are talking about the testimony of Mr. Drew Ranier yesterday, if I could quote a couple of sentences, he said, in general, there are adequate remedies under Louisiana law for compensation of his client.

In another sentence, he said, "I would not propose a modification of the remedies available to Mr. Hollingsworth."

What we are saying is there may be a need for a change. We may, in a year, after we have gotten studies and the ATSDR has gotten studies, we may decide that there should be a change and come back to you and suggest you make a change. We are saying, let's not jump in and put together some type of legislative remedy when we don't know what we are remedying. Let's determine if we have a problem. What is the magnitude of the problem? What is the proper solution to the problem? And then remedy it.

Mr. MOLINARI. Since you want to quote from Mr. Ranier's testimony, I think it is only fair to tell you that he did orally make the suggestion to the subcommittee that the burden of proof be shifted under circumstances such as I outlined, because of the difficulties in proving a case by today's standards. If you take somebody that was as severely injured as the client that he had with him, Mr. Hollingsworth, yes, a lawyer might be willing to shoot crap on a case like that, because if he does come in with a verdict, he is going to come in with a big one.

But what about all the rest of the people, those who didn't suffer severely but are suffering and have suffered for a period of 1, 2, or 3 years or more? They don't have enough of a case to bring in a big verdict. An attorney is not going to take on that tremendous job, almost insurmountable, in trying to prove a case with the expectation of a modest verdict which would not compensate the attorney for the time put in or the individual who has been injured.

That is really what we are here to discuss. We are here to find out whether the injured party has adequate remedies under the law, as it currently exists.

Dr. **Karrh**. I think the 301(e) study group set up by Superfund—and it was a group of lawyers—made the recommendation that there probably needed to be a change in the current system, if their original presumption was correct. The original presumption was that there was a big problem out there and a large number of victims. They made that presumption and said if, in fact, there are large numbers, then we need to change the current system.

What we are saying is we don't disagree with that. Instead of making a presumption that there are large numbers, let's take a little bit of time—and we are not talking much time at all—and do what studies we can do to determine was that presumption correct and should there be some type of legislative change for that, rather than presuming that there is a problem and saying the system will fail. Let's get some indication of the magnitude of the problem, if it exists.

Mr. **Molinari**. What are your projections for the time lapse that would be involved?

Dr. **Karrh**. We will have the UAREP study that CMA is committed to proceed with, completed by 10 months from the time it starts. We think we can start in September. UAREP thinks they can start in September. That would be July. That is a year. This would be a study done totally independently of the chemical industry. The industry will be putting up, we hope, more than half of the amount of the study, and we hope we can get others, including environmental groups and others, to participate with us in this so that the chemical industry will not control the study at all. It will be an independent scientific study.

At the end of a year, we think we will have a pretty good idea of the magnitude of the problem, if a problem exists.

Mr. **Molinari**. Who is providing funding for the study?

Dr. **Karrh**. CMA is putting in $500,000 of the $1 million study. We are in hopes we can get other industry groups, environmental groups, governmental groups to come in with us and also help fund it.

Mr. **Molinari**. Then you are not sure you are going to be able to——

Dr. **Karrh**. We are not sure we will be able to get other groups to participate, but we are sure UAREP will start the study. If we can't get others to participate, then we will have to get as much out of the study as we can.

Mr. **Molinari**. The study will be completed next July?

Dr. **Karrh**. I cannot assure that the chemical industry will be able to pay for the full study if others do not participate with us.

But I can assure you we will start it and get whatever data we can out of it for the amount of money we put into it.

Mr. **Molinari**. Thank you.

Mr. **Levitas**. Mr. Sundquist?

Mr. **Sundquist**. Thank you, Mr. Chairman.

Dr. Karrh, I started the hearings with the premise that we didn't need more legislation. In spite of your testimony, I am still hopeful we don't need more legislation.

A few minutes ago you said we didn't have a problem with chemical dumps; that there is not a danger right now, July 21, 1983; there is not a health hazard there.

Is that correct?

Dr. **Karrh**. No, sir. I did not say we did not have a problem. I said the public perceived that there was a problem; that we have not been able to determine that there is a significant health problem from waste disposal sites, but that we need to look at it more thoroughly to determine if there is.

Mr. **Sundquist**. Although the public perceives it, as both you and my colleague mentioned during his questioning, you have not determined that there is a problem in your own mind; is that correct?

Dr. **Karrh**. Based upon the data that we have reviewed, we do not believe there is a problem that needs an immediate solution. We do believe we need further study.

Mr. **Sundquist**. How do you relate that to the Toone site, the problem there?

Dr. **Karrh**. I am not familiar with the lawsuit you are talking about.

Mr. **Sundquist**. I am talking about the site itself.

Dr. **Karrh**. Yes, sir. I can only go back to the epidemiology study——

Mr. **Sundquist**. Which showed from your standpoint that there was not a problem?

Dr. **Karrh**. It showed that there were some health effects the people experienced. When the cause of the health effect was found, and corrective action taken, the health effects went away. There is a problem at that site; the acquifers are contaminated. There is a problem there. That does not necessarily mean that there are large numbers of people for which some type of legislative remedy should be put forth so that they can be compensated.

From what you have told me today, it indicates to me at least that the current existing system is capable of taking care of the victims if there are victims there.

Mr. **Sundquist**. I want to move into that in a minute. That issue involved the pros and cons of changing the system; that the judicial system can or can not handle the problem.

But, again, I find your testimony inflexible in your failure to even acknowledge that we have a problem anywhere. I point to the Hardeman site; there is, by anybody's measurement, some poison in the ground or in the water.

Dr. **Karrh**. My testimony——

Mr. **Sundquist**. Whether it is 10 people who have been injured as a result, or 100, there still is a problem with some people. It may not be a big problem.

Dr. **Karrh**. My testimony says that—and I have said it repeatedly in answer to your questions—there is a problem there. The question we have though, is there such a problem, of such a magnitude—you used the number, whatever number may be affected?

Our testimony is that we don't know if there is a need for legislation. I would hope Congress would not pass legislation for 10, 15 or 20 people. But if there are numbers of people for which—similarly situated as those in Hardeman County, then there may be a need for legislation.

Mr. **Sundquist**. I hope we don't need legislation, either. But my problem is your failure to acknowledge that there is a problem anywhere. In your testimony, you say we have not yet established a probable relationship.

Dr. **Karrh**. Mr. Sundquist, I don't think that I have ever said there is not a problem anywhere. I just finished saying there is a problem in Hardeman County, TN. My testimony says that.

In response to your questions, I have said that four or five times today. That does not mean there is a widespread problem for which some sort of legislative solution is needed.

Mr. **Sundquist**. You are saying there is not a widespread problem?

Dr. **Karrh**. For which some legislative solution is needed at this time, based on the studies we have done. There may be a problem, and we think additional studies need to be done to determine is there a problem.

Mr. **Sundquist**. A few minutes ago I thought I understood—and maybe I misinterpreted when we were talking about the dumps—that you said there is not now a problem, but there could be one in the future.

Dr. **Karrh**. What I said was, based upon the data we have reviewed, we have not seen a widespread problem that requires some type of legislative remedy now. But we are in favor of cleaning up the dumps, because we think the dumps ought to be cleaned up, because they—in addition to the public perceiving a problem, there is a possibility that as long as the dumps are there, there could be a problem in the future.

So, yes, we are in favor of cleaning up the dumps.

Mr. **Sundquist**. There is a possibility of a problem in the future. What kind of problem?

Dr. **Karrh**. I have no idea. It would depend on the dump, what is in the dump, how many people are exposed, how are they being exposed, and what is the magnitude of it.

Mr. **Sundquist**. Is it a widespread problem potentially?

Dr. **Karrh**. Based on our review of the data, there is no widespread problem now. With the number of priority sites EPA has identified, if they were all to leak, there could be a widespread problem. But we are in hopes EPA will get on with the cleanup before they leak so we won't have a widespread problem.

Mr. **Sundquist**. How many of the dumps on the Superfund priority list now are leaking?

Dr. **Karrh**. I would have to refer that to the EPA people.

Mr. **Sundquist**. If you are telling me there is not a widespread problem, I don't need to ask EPA.

Dr. KARRH. I am saying there is no widespread health problem we can determine now.

Mr. SUNDQUIST. How can you determine that if you don't know how many dumps are leaking?

Dr. KARRH. Because we have looked at what little bit of health data are available and determined that based on that, there is not a widespread problem. But more studies need to be done.

Mr. SUNDQUIST. What did you say just a minute ago?

Dr. KARRH. I said several words.

Mr. SUNDQUIST. Just before this. You want to be quoted precisely and you want to give your total answer. I want to give you that opportunity. What did you say a minute ago about a small number, or what was the term you used on which you are basing your conclusion?

Dr. KARRH. I am sorry. I don't know in response to which question you are referring.

Mr. SUNDQUIST. Could you repeat the last question I asked and his answer?

[The record was read by the reporter.]

Mr. SUNDQUIST. So, based on the little bit of health problems now, you are not aware of a widespread problem with the dumps?

Dr. KARRH. I am saying there is not a widespread problem for which some type of legislative solution needs to be put forward based on the data we have reviewed; also using the occupational exposure data and drawing some inferences from that. But we certainly think further study needs to be done to determine if there is a widespread problem. We can only use the data available now and draw whatever conclusions we can from that.

Mr. SUNDQUIST. OK. Tell me the pros and cons, if you would, of the judicial system as it is now: Is it working? Is there a widespread problem or not, which should be addressed by some legislative activities that might come out of this committee? What are the pros and cons?

Dr. KARRH. I am not an expert on the judicial system at all. I am certainly not an expert on the legislative system, either. All I am saying is that before Congress passes some type of legislation, we would hope that they would look and see if there is a problem for which the legislation is needed.

Mr. SUNDQUIST. So your testimony today is not to tell us that we should or shouldn't pass legislation based on what we know; it is to tell us that Congress ought to find out more. You are not here to tell us we shouldn't have legislation?

Dr. KARRH. I wouldn't pretend to tell Congress anything, Mr. Sundquist. What I am here to say is that we have looked at what data are available and have decided that there is not an eminent problem that needs legislation at this time. But there is a need for studies at this time, and we want—we would like to have the studies done by ourselves and by the governmental agencies, and determine, is there a need for legislation.

Mr. SUNDQUIST. Do you feel that people in Hardeman County are going to be satisfied or be taken care of through the courts?

Dr. KARRH. I don't know. They obviously have used the remedy available to them, and that is the court system. That is the currently existing solution. So far we have seen no evidence that that system has failed.

Mr. **Sundquist**. But you are here representing industry, are you not?

Dr. **Karrh**. Yes, sir.

Mr. **Sundquist**. Your industry is opposed to any new legislation. I think, with good reason, that the Federal Government doesn't always satisfy everything that needs to happen through new laws. But I find your arguments weak in that you are saying we don't have a widespread problem with hazardous chemicals in dumps that have been located, or we don't have problems in Missouri where chemicals have been mixed with soil, when most everybody in the country, if you were asking the question, and the victims who have been before this committee, are saying there is a problem.

I would say to you and your industry that your failure to acknowledge that there is a problem is weakening your arguments that we don't need new legislation, and leading me to think we do. I think you are covering your head up with the blanket, turning up the electric blanket and assuming the prenatal position, instead of acknowledging the problem, and saying let's get on with it and figure out whether the courts are handling it or not.

Will they handle it in the future? Can they handle it in the future? Should there be a better plan? What I understand you to say is you want a year to determine that.

Dr. **Karrh**. What I am saying is that I agree with you. There is a public perception of a problem. I have said that repeatedly today. We have not yet determined that there is a scientific problem. We think that before we have some legislation, that may or may not address the problem that exists, we ought to determine what is the problem and what legislation is needed, if any.

Mr. **Sundquist**. There is a scientific problem in Hardeman County; I can tell you that.

Dr. **Karrh**. I really don't know what——

Mr. **Sundquist**. Ten or twelve. You said that yourself—10 or 12 people.

Dr. **Karrh**. I am not sure what you would like me to say. I will be glad—if you tell me what you want to hear from me, I will try to say it if I can. But I don't know what you want me to say.

Mr. **Sundquist**. You have been very consistent in your answers, as my colleague said. My frustration is that you refuse to acknowledge we have a problem with hazardous chemicals in the country.

Dr. **Karrh**. And the reason I refuse to acknowledge it is we haven't found it. If we find one, I will acknowledge it. We say, let's get the scientific data, and if there is a problem take care of it. But just because——

Mr. **Sundquist**. How long have we been dumping dangerous chemicals?

Dr. **Karrh**. I don't know.

Mr. **Sundquist**. What is the earliest? You don't have any idea?

Dr. **Karrh**. No, sir.

Mr. **Sundquist**. That is all my questions, Mr. Chairman.

Mr. **Levitas**. Dr. Riordan, your testimony, which is part of the record, describes the EPA risk assessment policy. Has that policy changed—I am going to give you two timeframes—during the period that Mrs. Gorsuch was administrator from the time previous thereto, and since Mr. Ruckelshaus has become administrator?

Dr. RIORDAN. Not to my knowledge, Mr. Chairman. But Dr. Anderson can answer that, I think, exactly.

Dr. ANDERSON. The answer is certainly no; not with respect to anything that is published. We are still operating from the 1976 interim guidelines for assessing cancer risk. We proposed guidelines for assessing genetic damage and they were never made final during the last 2 years. We did not change any of the guidelines during the last 2 years; nor has there been any issuance of a new Federal cancer policy to take the place of—such as it represented that kind of thing—the IRLG document.

I think within EPA the Carcinogen Assessment Group, which is the oldest risk assessment group in the agency, has certainly continued operating as it always has. It didn't alter course one bit over the last 2 years. It regarded animal studies the way it always did. Nevertheless, risk assessment is in its infancy and several refinements are being made on a rather continuous basis as we learn a little bit more about how to better assess the risk.

But I think the short answer to your question is that we really haven't changed. We are right now under Mr. Ruckelshaus' leadership looking at the need to advance our guidelines further and incorporate some of the refinements we have been doing by practice both in the cancer area and refining some of the other guidelines that we have commenced and not completed. He has asked us to make some recommendations so that we can make a decision whether we should do something more in this area.

Mr. LEVITAS. I saw, I think it was, a two-page memorandum from Dr. Hernandez, which would have been about the second half of 1981, in which he outlined criteria for taking action based upon risk assessment under the Superfund Program. I, unfortunately, don't have a copy of that document with me. But if you remember it, fine, and you can comment on it. If not, I will have to supply it to you for a subsequent comment.

Dr. ANDERSON. Mr. Levitas, I think what you are—I don't know the memorandum, but I think what we are talking about here is a separate process; that of deciding how to manage the risk. You assess the risk scientifically and say here is the scientific situation.

Mr. LEVITAS. That is correct.

Dr. ANDERSON. Then managing the risk is a separate decision that incorporates other considerations. I think there may well have been some differences in the way the risks were managed in the last 2 years versus the preceding 4 years, because I think essentially the record has been reviewed, and there were more actions in the preceding 4 years, and so forth. I think it would take some review I can't provide now.

Mr. LEVITAS. I think you are absolutely correct that the management of the risk question that arose in the context of these circumstances exists. At what point, and based upon what information and what procedures, shall regulatory or enforcement action be taken? Now, who in EPA could address that question for us?

Dr. ANDERSON. The various program offices, I would think, in each individual case. The agency has never adopted any guidelines for risk management. So you can't look at one set of guidelines and say this is exactly what they do. I would think you would have to almost go to each program and ask what they were doing before, and if they changed it.

Mr. LEVITAS. All right. I will retrieve my copy of that memorandum, and we will make it available to the appropriate people at EPA because it was a proposal for deciding when to take action. It followed the Dallas lead situation and in effect tried to institutionalize that decisionmaking process that occurred in the Dallas situation.

I want to compare that to the preexisting policy and whether there has been any change subsequently, since Mr. Ruckelshaus has come on. I am most interested in that.

Dr. ANDERSON. I would suggest referring it to the agency's policy office. Obviously, it is easier to have one point of contact. I think the newly named director of that office——

Mr. LEVITAS. That is Mr. Cannon?

Dr. ANDERSON. Mr. Cannon. Thank you.

Mr. LEVITAS. Thank you.

Dr. Riordan, I understand you have a meeting that you have to attend.

Dr. RIORDAN. That is not Mr. Cannon but Mr. Russell.

Dr. ANDERSON. I am sorry.

Dr. RIORDAN. Director of the Policy Office.

Mr. LEVITAS. Thank you, Dr. Riordan. You are excused. We appreciate you being here today. I found your testimony most interesting and informative.

If the rest of the panel could remain, I don't think we are going to be going much longer. In fact, we can't. I have got a 2 o'clock classified briefing I have got to get to.

Dr. Karrh, although neither your oral statement nor your written statement made reference to the fact that CMA thinks we should go forward with the cleanup of the dumps, you certainly have said that on several occasions in answer to my questions.

Dr. KARRH. Yes, sir.

Mr. LEVITAS. And the only problem that I have in relating that conclusion, for which I commend you, to your differing conclusion, when it comes to providing a mechanism for victim compensation, it lies in this sort of syllogism.

Leaving aside public perception, the reason that we should do something with respect to hazardous site cleanup is because the potential of public health risk exists. And it is more, presumably, than just a speculative or hypothetical risk. If these hazardous waste sites begin to leach out their materials and people in the adjoining area are contaminated with them, there is the potential—potential—potential of health exposure and adverse health consequences. So, therefore, we ought to clean up the dumps.

Now, if we are prepared to take action based upon that potential, then why shouldn't Congress similarly take action with respect to those people who are now presently and have been exposed to those same dumps?

Dr. KARRH. For one thing, we know the dumps are there, and that they, in the future, are likely to present a problem. We are not sure of the extent of the problem as far as victims are concerned. We know there are situations where there are victims. There are situations of few clusters of victims of various things.

The real question is, should we legislate some type of legislative remedy for a very small number with the anticipation that there is going to be a large number; or should we wait a very short period of time and determine what is the extent of the problem and then put in appropriate legislation that will address the extent of the problem?

It is not a matter of our saying that no legislation is ever needed. All we are saying is, "We know the dumps are there so let's clean them up because we know that they are there and, because they are likely to present a health problem in the future. "But do we have enough scientifically proven victims at this time that we feel that we know enough to put in the proper legislation and that legislation is needed?

Mr. LEVITAS. Wouldn't it depend on what the legislation consisted of?

Dr. KARRH. I am sure it would, to a large extent. But wouldn't what the legislation consisted of depend upon the number of people you are trying to cover with it?

Mr. LEVITAS. I am not sure that logically follows. Mr. Sundquist was making the point or was at least attempting to make the point, as far as your dialog was concerned, that there are people who have been affected by chemical exposure. And, as Mr. Molinari said, "The question is how do you prove it as a matter of fact?" And there are many areas of the law where, once a certain situation is shown to exist, then the burden of proof goes to the place where that situation originated to prove that they weren't responsible for it. The old example of storing water, impounding water in land; if it gets out, the person who is hurt by it can bring a lawsuit. And it is up to the person who impounded the water to prove that some act of God, or whatever, was actually responsible.

The same is true with certain dangerous materials, or the product liability area, as well. That type of approach could be taken, couldn't it, even without knowing the numbers of people who might be adversely affected by hazardous waste sites?

Dr. KARRH. Our feeling is that it is really reversing the current system where you have the guilty until proven innocent instead of the innocent until proven guilty situation. It may well be that that type of legislation is needed in the future. but we ought to know it is needed before we put it in.

Mr. LEVITAS. Well, I think that is true. I am not sure it follows that the need for that is based upon the extent to which the population is adversely affected. It could be addressed strictly from a legal jurisprudential point of view; that in any type of situation where the evidence of proof is in the hands of the person that manufactured, or stored, or disposed of a material, that since they have got the information, they are the ones who are in the better position to answer the questions as to responsibility.

But, as you have stated, you are not an attorney, and you don't want to get into that.

Dr. KARRH. That is correct.

Mr. LEVITAS. I will say, Mr. Sundquist, that next Thursday—next Wednesday, we are having a panel to address the questions of the sort that we have just been talking about, and we extended an invitation to the Chemical Manufacturers Association to have a representative on that panel. They declined for the consistent reason that since they don't perceive the present problem, therefore, they don't think it is appropriate to suggest solutions to the problem. So, you ought to be aware of that.

Japan—are you familiar with the system in Japan?

Dr. **Karrh**. No, sir.

Mr. **Levitas**. Japan has a system very similar to workmen's compensation, where if you live within a certain defined area near a hazardous waste site and manifest certain defined symptoms, then you qualify for compensation under this plan. But, by the same token, it has a limitation of liability.

Have you heard any discussion within your industry about that type of program?

Dr. **Karrh**. No, sir.

Mr. **Levitas**. I don't have any further questions at this point. We have got 5 minutes for Mr. Molinari to ask questions.

Mr. **Molinari**. I would like to address this to Dr. Riordan's representatives from EPA.

Yesterday, we heard from Mayor Leistner, who I see is in the back of the room. She talked about the people that lived in Times Beach, MO. EPA has agreed to a buy-out, totaling some $33 million. The State is matching with 10 percent. We heard, much to my shock and surprise, that there is no record being kept of the residents and where they are moving to so that they can be monitored.

Of course, it is a very simple point that some of these problems associated with the exposure to toxic or hazardous waste may not show up for some time. Yet, you have recently opened the Office of Disease Registry——

Dr. **Jason**. We have not. That is being done through CDC. CDC should also be keeping something of a log in terms of who the individuals are that they examine, the 140 or so they got samples on. They had something of a voluntary participation, which, of course, limits, you know, the actual useability of those kind of studies—who is going to volunteer, who is going to come. Even acknowledging those, they have 140 individuals they are expected to be able to follow.

Mr. **Molinari**. I am concerned about the chemical industry's view that when dealing with small numbers, the results of those samplings may not be adequate. We had something like, was it 2,200 people?——

Dr. **Jason**. 2,200 people.

Mr. **Molinari** [continuing]. Who resided there. I would ask you to go back to EPA, maybe CDC, and inquire of them as to whether it would be feasible to monitor, to whatever extent possible, those that you are able to locate at the present time, before you lose track of where they are.

[The following was received from Dr. Jason:]

Health Study at Missouri Dioxin Sites, Including Times Beach

Question:

At the Subcommittee's July 21 hearing on Hazardous Waste Victims Compensation, Mr. Molinari requested information on the health study that the Centers for Disease Control (CDC) is conducting for residents of Times Beach, Missouri. Among other things, he questioned whether CDC is establishing a registry of former residents to continue long-term health monitoring.

Answer:

In December 1982, EPA entered into an interagency agreement with CDC to conduct a health study and other health related activities at four dioxin contaminated sites in Missouri. The four sites are Times Beach, Shenandoah Stables, Minker/Stout residences and the Syntex Facility. The total amount of tne interagency agreement is $496,000 and its completion date is December 31, 1983. The newly created Agency for Toxic Substances and Disease Registry is responsible for conducting the study.

Activities under the agreement fall into three major categories:

1) Public health protection - recommendations on levels of 2,3,7,8 TCDD in soil to support decision on habitability, and on the need for further medical evaluation of residents.

2) Medical/Epidimiologic Evaluation - medical and laboratory examinations for population groups with highest environmental exposure.

3) Central Listing and Registory of Data for Exposed Individuals - a central file of people presumed to have been exposed to dioxin at the four sites.

A copy of the interagency agreement is attached. If you would like more detailed information on the scope and methodology for these studies, contact Dr. William Foege, Administrator, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia 30333, telephone (329)236-3291.

U.S. ENVIRONMENTAL PROTECTION AGENCY INTERAGENCY AGREEMENT *(Please read instructions on page 3)*	1. EFFECTIVE DATE	2. COMPLETION DATE	3. AGREEMENT NO.
	12/9/82	12/31/83	AD75F3A448

4. NAME AND ADDRESS OF EPA ORGANIZATION

U.S. Environmental Protection Agency
Office of Solid Waste & Emergency
Response
401 M Street, SW, Wash., D.C. 20460

5. NAME AND ADDRESS OF OTHER ORGANIZATION

Department of Health and Human Services(HHS)
Hubert Horatio Humphrey Building
2nd and Independence Avenues, SW
Washington, D.C.

6. PROJECT TITLE

Agreement for reimbursement of funds obligated by HHS, Centers for Disease Control (CDC) for the investigation of complaints of illness among persons living in areas of Missouri which may be contaminated with dioxin.

7. SCOPE OF WORK

This agreement provides $496,000 to the Centers for Disease Control to cover obligations incurred for the health related activities described below. CDC will address health aspects of environmental dioxin contamination at four sites in Missouri. The sites are:
Arena 1 (Shenondoah Stables), Moscow Mills, MO; Arena 2 (Fill Areas 1 and 2); Imperial, MO; the Syntex Facility, Verona, MO; and Times Beach, MO.

This agreement covers the following site-specific activities:

1. Public Health Protection: At each site, CDC will provide recommendations or guidelines on the levels of dioxin (2,3,7,8-TCDD) in soil in order to support decisions on the habitability of affected areas, the need for relocation of residents and the need to initiate other public health activities such as medical evaluation.

Recommendations will be made in the context of cleanup strategies proposed by EPA.

CDC will develop a scientifically sound, practical working document for each site which will be reviewed by an appropriate panel, possibly including individuals from NIEHS, EPA, and outside toxicologists. Site specific recommendations may be provided (See continuation for Attachment I)

8. SPECIAL PROVISIONS

This agreement may be terminated by either Agency upon 30 days advance written notice.

1. Funding for this agreement will be made available to HHS by the Administrator for the performance of the site-specific activities described herein in accordance with section 111(c)(4) of CERCLA, as provided by Congress in P.L. 97-216, a 1982 supplemental appropriation.

2. CDC activities that pertain to ongoing Superfund responsibilities that are not site-specific in nature are funded under existing interagency agreements. (IAG# AD75F2A091) which provide funds for continuing staff support at HHS. Activities that have been proposed by CDC for investigation of potential dioxin contamination that are non-site specific, such as the development of questionnaires, providing ongoing technical support and advice to the State on dioxin in general; and
(See continuation sheet for Attachment II)

9. REPORTS

CDC will provide quarterly status reports to EPA by site for each category of activity (Public Health Protection, Medical/Epidemiologic Evaluation, Central Listing, Administration) which will contain:

1. An accounting of funds obligated to date and during the current reporting period.

2. A short narrative indicating progress made in the reporting period; and the current status of health-related activity.

(See Continuation Sheet - Attachment III)

9. EPA PROJECT OFFICER Kathy Veit	TELEPHONE FTS 382-2196	11. OTHER AGENCY PROJECT OFFICER Georgi A. Jones	TELEPHONE FTS 236-4100

ADDRESS
Environmental Protection Agency
Office of Emergency and Remedial Response
401 M Street, SW
Washington, D.C. 20460

ADDRESS
Centers for Disease Control
SIG/CEH
Atlanta, Georgia 30333

12. EQUIPMENT-PROCUREMENT OF EQUIPMENT WITH EPA FUNDS IS:

☒ NOT AUTHORIZED ☐ AUTHORIZED ☐ SUBJECT TO THE FOLLOWING LIMITATIONS:

Equipment purchased will be inventoried in accordance with the supplying agency regulations. The supplying agency will be the accountable agency for all items of equipment costing $25,000 or less. EPA will be the accountable agency for all items of equipment costing over $25,000 or which form part of an integrated equipment system costing over $25,000. An inventory of equipment purchased will be submitted by the supplying agency annually to EPA on the anniversary of this agreement. A final inventory of such equipment will be submitted to EPA within 30 days of completion of the work and services under this agreement. Title to equipment purchased shall be vested in EPA whose decision concerning the disposition of the equipment shall be final. The agencies shall mutually determine the procedure for inventorying, accounting, and disposing of jointly funded equipment.

13. EQUIPMENT TO BE FURNISHED OR ACQUIRED

None

14. ESTIMATED COST

a. TOTAL ESTIMATED PROJECT COST	$ 496,000	b. AMOUNT TO BE FUNDED BY THIS AGREEMENT/AMENDMENT	$ 496,000
EPA SHARE	$ 496,000	EPA SHARE	$ 496,000
OTHER AGENCY SHARE	$ —	OTHER AGENCY SHARE	$ —

15. METHOD OF PAYMENT

☐ ADVANCE* ☒ REIMBURSEMENT ☐ ALLOCATION TRANSFER Not to exceed $496,000

*Unexpended amounts remaining at completion of the work will be returned to EPA.

16. BILLING INSTRUCTIONS:

Request for payment will be made by Itemized SF-1081 submitted to:

Environmental Protection Agency
Financial Management Division
Room 214
26 W. St. Clair Street
Cincinnati, Ohio 45208

CDC will cite the following site specific account numbers and DCN No. when requesting reimbursement.

	Account No.	DCN No.
Arena 1:	3TFA727F40	E2B034
Arena 2:	3TFA727F45	E2B035
Syntex:	3TFA727F51	E2B036
Times Beach:	3TFA727F61	E2B038

and will cite the following accounting information:

APPROPRIATION	ACCOUNT NO.	DCN NO.	OBJECT CLASS	IAG NO.
68/20X8145	3TFA72EF00	E2B037	25.70	AD75F3A448

REQUEST FOR REIMBURSEMENT OF ACTUAL COSTS WILL BE ITEMIZED AND SUBMITTED:

☐ MONTHLY ☒ QUARTERLY ☐ UPON COMPLETION OF WORK

17. AUTHORITY

CERCLA and Executive Order 12316

18. APPROVALS G. M Kingham 1/3/83

a. NAME AND TITLE OF AUTHORIZING OFFICIAL FOR EPA Rita M. Lavelle, Assistant Administrator Office of Solid Waste and Emergency Response	SIGNATURE Rita M Lavelle	DATE 1/7/82
NAME AND TITLE OF AUTHORIZING OFFICIAL FOR OTHER AGENCY William H. Foege, MD, Director Centers for Disease Control	SIGNATURE William Foege	DATE [illegible]

EPA Form 1610-1 (6-79)

Thomas O. Hammel FmD 12/30/82

Continuation Sheet - Attachment I

7. Scope of Work

in phases as data becomes available from EPA, and as such data are reviewed in the context of what is known about the route of exposure and the characteristics of the persons exposed.

Cost: Not to exceed $13,333

2. Medical/Epidemiologic Evaluation: As appropriate to each site, CDC will provide for medical and laboratory examinations for the group of people with the highest potential environmental exposure. These people (particularly if considered for relocation or when exposed chronically to high levels) should have a detailed medical and laboratory examination looking for the known or presumed health effects of dioxins as determined from findings in industrial settings or animal experimental studies. This preliminary assessment should be considered as a pilot evaluation to see if the most exposed people demonstrate health effects which, based on the best available clinical judgment, are consistent with dioxin exposure. Further evaluation of these or other groups can then be considered in the light of these findings.

CDC may enter into a cooperative agreement with the Missouri Division of Health to develop and implement a testing protocol. It is recognized that medical and laboratory tests cannot be definitive about the presence or absence of dioxin-related disease, but the best clinical judgment on an individual basis would be helpful in the determining the likelihood of possible effects in the persons tested.

For each site, CDC may also arrange to collect information from individuals relating to alleged dioxin-related health problems.

Cost: Not to exceed $200,000

3. Central Listing and Repository of Data for Exposed Individuals: Establish and maintain a central file of people presumed to have been exposed to dioxin at the sites listed in this agreement. As appropriate, CDC may enter into a cooperative agreement with the State of Missouri for establishing and maintaining this file. CDC will provide technical assistance to the State, as required.

Cost: Not to exceed $200,000

4. Administrative Costs: Department of Health and Human Services overhead factor = 20 percent

Cost: Not to exceed $82,667

Continuation Sheet Attachment II

8. Special Provisions

the development of a protocol to uniformally evaluate skin rashes which are alleged to be chloracne, will be funded from EPA/HHS existing interagency agreements.

3. While this agreement provides funding for the investigation of health effects at four sites, EPA may request CDC to perform similar activities at other sites with potential dioxin contamination where EPA may take removal or remedial actions. As appropriate, EPA will amend this agreement and provide additional funding to HHS, if required.

4. The Centers for Disease Control will in a timely manner share and consult with EPA concerning all data obtained and all proposed recommendations/ conclusions which are based on health activities taken under terms of this agreement. Data will be shared with Missouri health officials who are working with CDC under a cooperative agreement. Medical information about individuals obtained during their health evaluation will be provided to those individuals and/or their physicians at the request of the individuals. Interim and final reports, as well as recommended actions regarding health matters, will be issued only after review and consultation with EPA.

5. Reimbursements specified herein are contingent upon receipt, acceptance and approval by EPA of the quarterly status reports as required by this IAG.

6. CDC may obligate up to $124,000 for each site. On request of CDC, the EPA project officer can approve by memo shifts of funds among categories of activities and shifts of funds among amounts allocated for obligation at each site.

Continuation Sheet - Attachment III

These reports will be submitted to EPA on the 15th day of April, July, October and on project completion.

In addition to the quarterly status reports, CDC will provide to EPA documents/recommendations described in the Scope of Work.

All reports shall be submitted to:

William N. Hedeman, Jr., Director
Office of Emergency and Remedial Response
Environmental Protection Agency
Washington, D.C. 20460

Morgan Kinghorn, Comptroller
Office of the Comptroller
Emergency Protection Agency
Washington, D.C. 20460

Futhermore, CDC will retain detailed and accurate records by sites for all costs for which reimbursement is requested under this agreement. Such documentation may be required by EPA from time to time to support cost recovery actions. Additionally, documentation must be available for audit or verification on request by the Inspector General.

Dr. **Jason**. One other thing, Mr. Molinari. I have taken something of an interest in the bills, the concerns that you have. One of the things that I would wonder about is, when we start thinking of Love Canal, when we start thinking of Woburn, MA, it was really the citizens that organized and made a claim that there was something going on. We really depend upon an enlightened public to be able to realize a problem is actually present, that they think an exposure resulted in a medical health problem.

I think the idea of saying one way or the other that there is or is not a risk puts a great deal of responsibility on the people to recognize it. In the case of Woburn, we had a minister that recognized the problem. In the case of Love Canal, we had a small number of people who pushed the issue.

I can't speak for EPA's policy as one individual. But one of the problems we have is the one of who is going to be able to look at areas and say, does this represent a significant increase?—given the fact we are not always going to have enlightened people of the particular training involved in, let's say, Woburn, for example.

Mr. **Molinari**. I don't have very much time, but it seems to me that we certainly have at this point in time—or should have—people within EPA, at some level, who could follow these people and collect health data from them, assess it, compare it and then determine whether there is a problem. We shouldn't lose sight of these people whom we suspect may eventually have additional health problems.

I have one last question. We have a problem in my own area, where it is acknowledged that there were some 3 to 10 million gallons of a variety of chemicals dumped in a municipal landfill by organized crime and others. And the people in the nearby area are all complaining about problems, the same kinds of physical problems Dr. Carnow described and I described before.

I got about 50 or 100 people to go out into the field with questionnaires developed by a professional pollster. We had them spend 20 or 25 minutes asking all the questions to try to determine whether there was a health problem. We compared the answers against several other control groups living in the community further from the dumpsite.

Concurrent with that, the New York City Health Department did the same thing, only by telephone. We thought the results would be better if we went into the field with a professional questionnaire. What we did find was that there were well over twice the number of incidences of illness in that area as in any other area in the community.

Would that be considered scientific evidence by your definition?

Dr. **Karrh**. I would have to see the data myself, Mr. Molinari. I don't know exactly what you are referring to. Incidences of what? I would be more than glad to look at the data and give you an opinion based upon that.

I would like to point out one thing in response to your question regarding EPA and the registries. Had the Environmental Defense Fund, joined by the CMA, not sued EPA and HHS, registries would not have been set up. So we feel very proud of our role in it.

Mr. **Molinari**. You should be.

Dr. **Karrh**. I would be pleased to review that questionnaire, if you want me to.

Mr. **Molinari**. Assume we didn't make any mistakes in the testing techniques and that the survey shows the incidence of these problems is twice that of another area of Staten Island, we are talking about less than a half-mile square area here as opposed to the rest of the 57 square miles—and that the incidence of problems reported to the City Health Department and ourselves, two independent groups, reveals relatively similar results.

Would that be considered scientific data?

Dr. **Karrh**. It would be considered scientific data probably, but I think you would have to look at the data themselves and determine what you were finding and determine what the possible or probable errors are and see if they could enter into the final conclusion. I know Dr. Senser of the city of New York, who would be——

Mr. **Molinari**. Fine. I know the chairman has a very important meeting. I am grateful to all of you for coming here to help us with this very complex problem.

Mr. **Levitas**. Before I conclude, Dr. Carnow, let me furnish you a copy of a letter which a professional staff member on the Surface Transportation Subcommittee of this committee received from a law firm, Coburn, Croft, & Putzell, in St. Louis--at least, that is where their main office is—on behalf of the Monsanto Corp., asking for certain information from the committee under the Freedom of Information Act concerning you and your firm. And since I am advised by our counsel that the Freedom of Information Act in this regard does not apply to this committee, you ought to be aware of this in case you desire to provide that information.

Dr. **Carnow**. I am aware of it. I am aware of the fact that virtually everything I say is either recorded or monitored by this organization with which I am involved, in an adversarial situation.

Mr. **Levitas**. I would like to express my appreciation to everyone participating in today's hearings. I certainly learned a lot, I think, and I am not sure that I agree with all of the conclusions that the witnesses drew. And there are clearly some very strong disagreements between various points of view.

I am persuaded that Congress and the regulatory agencies, when we are dealing with public health, would—if we err, should err on the side of protecting the public health, even if we later find out that that was premature, rather than facing the possibility of seeing people exposed and victimized, and find out that they should not have been, and should have been compensated for it. And, in that regard, assuming the Congress were to follow your counsel, Dr. Karrh, and defer setting in place a new mechanism for victims compensation, based upon the outcome of that study or some other study, I would assume that if Congress were to do that, that the statute of limitations in that legislation would be adequate to cover those people who were victimized prior thereto, and could gain their relief, if they are still living, in the future.

So it may be that that approach would be a more reasonable one. That is something we are going to be kicking around for a long time. But it would also seem to me appropriate that, if that were done, to make a statute of limitations sufficient to make certain that no one has been jeopardized simply because we delayed, which is one of the possibilities that I know is being considered.

But I, on behalf of the subcommittee, want to express my appreciation to all of you for extremely informative and thorough testimony. It will aid us immeasurably as we proceed further.

Next Tuesday, we will resume these hearings with a legal panel consisting of Lyman Lundquist, an attorney with the firm of Heller, Irman, White, & McAuliffe; Mr. George C. Freeman, Jr., with the firm of Hunton & Williams; and Frederick Baron & Associates, all of whom will address the question of whether or not there need to be changes in the toxic tort area.

I think that that will follow very well from the testimony we have heard today from the medical and scientific experts who would address this subject.

Again, I thank all of you very much.

The subcommittee stands in recess until Tuesday at 10 o'clock.

[Whereupon, at 2:05 p.m., the subcommittee recessed, to reconvene at 10 a.m., Tuesday, July 26, 1983.]

HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

TUESDAY, JULY 26, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 10:10 a.m., in room 2253, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Mr. Levitas. The subcommittee will please come to order.

This morning the Subcommittee on Investigations and Oversight of the Committee on Public Works and Transportation continues with its series of hearings on the matter of health and economic damages experienced by people exposed to toxic chemical wastes and the problems associated with their efforts to obtain full compensation for those damages, the so-called victim compensation issue.

Last week we heard testimony from some of those who have been affected by the presence of toxic wastes in areas where they live and work. We heard about the problems that their children and their neighbors have experienced from what they believe is the presence of a toxic chemical waste. We also heard testimony from a panel of medical experts, testimony that was most interesting and which indicated that there were obviously different points of view among those in the medical profession about the extent to which the presence of hazardous wastes in our water and soil and in the air may actually cause or pose human health problems.

I found that testimony to be most enlightening and while I have not formed any general conclusion based on the scientific portion of the testimony, it was clear to me that the difference in point of view that was expressed by Dr. Carnow and Dr. Karrh illustrates the difficulties in reaching judgments on this matter.

But with respect to the scientific portion of that testimony we will let the facts speak for themselves. I will simply observe that Dr. Karrh at least demonstrated admirably the quality of consistency even to the point of seriously damaging his credibility before the subcommittee.

In part, this difference in views appears to reflect the scientific uncertainties or limitations of traditional medical analytical techniques and the fact that frequently there may simply not be enough data or test results to answer questions about health impacts one way or the other. This, in turn, brings us to the questions we look to explore today.

Does this inability to prove or disprove with a high degree of certainty that hazardous wastes can seriously and adversely affect human health pose an insurmountable barrier to those seeking compensation for damages or harm they have experienced? Are the present remedies in the law adequate? Are the evidentiary and other requirements such that the legal system provides a reasonable vehicle of redress for those who seek compensation?

That last question, of course, is one which was explored in some detail by members of this panel and in particular by Congressman Molinari.

Just one last comment about Dr. Karrh's testimony. What I was referring to was his willingness to move himself and family into Times Beach and I was advised afterward that there is some property available for sale at good rates there and would urge anybody who shares that view to go in and make a killing right now.

With those introductory remarks, I would now recognize the gentleman from New York, Mr. Molinari.

Mr. **Molinari**. Thank you, Mr. Chairman.

I don't have any prepared text, but I would like to make an observation for the record.

Picking up where you left off, Mr. Chairman, last week we heard somewhat incredible testimony from Dr. Karrh, specifically about his willingness to live in Times Beach, MO, without the least bit of concern that his family might be exposed to dioxin. He predicated that feeling on the fact that there was no scientific evidence to indicate that exposure to dioxin on the part of human beings would pose any health problem whatsoever.

It was interesting to note at the conclusion of the week, we read a newspaper story of the NIOSH report, which I have not seen yet, but which is reported to indicate that for the first time there is some body of scientific evidence indicating that, in the case of human beings, there would be some harmful public health effects from exposure to dioxin.

I plan to get a copy of that as soon as possible; certainly that is something that we should be very aware of. Dr. Karrh spoke for the chemical industry. In particular, his testimony drew a comparison between workers in the chemical industry whose experiences, if in fact they don't have any harmful effects from working with chemicals, would demonstrate that residents of an area near a toxic waste dump similarly would not suffer health effects.

But now we are being told that, perhaps, dioxin has created some health problems for those in the industry. So that I would suspect that when we get the report, Mr. Chairman, if it does bear out what the newspaper accounts indicate, maybe Dr. Karrh would think twice about moving his family into Times Beach.

Thank you. I look forward to an interesting day of testimony.

Mr. **Levitas**. Dr. Rowland.

Mr. **Rowland**. I have no statement, Mr. Chairman.

Mr. **Levitas**. Before I call witnesses today, let me make the usual announcement. All testimony is taken under oath before this subcommittee. We request that the witnesses confine their opening statements to approximately 10 minutes, and then we will get directly into the questions after we have heard from the panel.

With that, the first witness this morning is Anthony Roisman, Trial Lawyers for Public Justice, Washington, DC.

Mr. Roisman, if you would please come to the witness table and raise your right hand.

[Witness sworn.]

Mr. LEVITAS. If you would take a seat. Identify yourself, your background, your interest and we will be pleased to hear from you.

I understand that the text of your testimony of June 28, 1983, before the subcommittee of the House Committee on Energy and Commerce has been submitted. Without objection, it will be made a part of the record. You will be speaking in part from that testimony.

TESTIMONY OF ANTHONY Z. ROISMAN, ESQ., TRIAL LAWYERS FOR PUBLIC JUSTICE, WASHINGTON, DC

Mr. ROISMAN. Yes; Thank you very much.

I am Anthony Z. Roisman, currently executive director of Trial Lawyers for Public Justice. I appear today as a private citizen and not in that capacity. Previous to this position I was the Chief of the Hazardous Waste Section in the Lands and Natural Resources Division at the Department of Justice. It was my responsibility to carry out the enforcement obligations of the Government with respect to the cleanup of hazardous waste sites.

In that context, and also in my current private practice in which I represent, among others, victims of toxic torts, I believe that I have something to contribute to this discussion which the committee has instigated on the issue of whether there should be victims compensation legislation in this area, and if so, what it should look like.

At the outset the debate seems to be framed on the question, is there really a problem?

I think that is a false issue and feel that it tends to deflect the debate. But because it seems to be the focus of so much hearing and testimony, I think it is appropriate to at least address it. First let me explain why I think it is a false issue.

If the Congress were to set up a victim's compensation scheme and there were no victims, the total transaction costs would be relatively slight. We would rather quickly learn that there was nobody who could qualify, and the Congress would not have expended a great deal of the Government's money. If, on the other hand, there is a problem, the scheme would be available to address that problem.

I feel that, at this point in time, at a minimum there is sufficient evidence available to suggest that there is a high probability that the problem not only exists but that it is quite widespread, and that if the Congress were to wait until the problem were even more apparent, the consequences in human suffering could be quite severe.

We know already as a result of congressional legislation, congressional investigations and Environmental Protection Agency investigations, that we have allowed the haphazard disposal of billions of tons of hazardous waste. We know that a substantial amount was placed in landfills and underground injection wells and other ways in which it is not totally under control. We know a lot of those dumps have leaked. In fact, an Environmental Protection Agency estimate was that as many as 2,000 of the dumps in the country may be serious enough to be classified as imminent and substantial endangerments to the public health.

Four hundred of those are listed in the Superfund list of sites which have high priority and deserve immediate cleanup.

Many of the dumps in the country have already contaminated ground water supplies, and ground water provides the drinking water for substantial parts of the population of this country.

Many people are already drinking that water or have drunk that water in the past, and a substantial number of them are sick.

The question that rises is, is that enough of a problem? Does that rise to the level at which Congress should act?

I submit that it does. In my own experience, I find that several times a month I receive telephone calls from individuals who say, we live near a dump. The dump has the following chemicals in it. The chemicals escaped from the dump; some have been found in my water. My child has this illness, my husband has that illness, my grandmother died of this other illness. I don't know but I think the dump had something to do with it.

Those calls that come to my office are but a small part of the total number of calls that trial lawyers across the country get from people who say, "Something is wrong, we are not sure what, but we want to get some kind of remedy."

The Association of Trial Laywers of America, which has 55,000 members in it, keeps an exchange called the ATLA Exchange. That exchange is available to the committee. The committee can ask the ATLA for the list of cases already filed in the toxic tort area where people are claiming they have been exposed to hazardous substances and those substances have caused them illnesses, property damage, or both.

The list is lengthy. The number of clients represented by it is quite substantial. The lawyers bringing the cases can only be compensated if they prevail, so there is a high probability that most of the cases have substantial evidence to support them. In fact, if they are filed in Federal Court, the lawyer who signs the pleading must attest to the fact that he has a good faith basis to believe that there is a substantive concern expressed which warrants recovery for his client.

All of this suggests to me as a lawyer that there really is a problem and a substantial problem. On the other hand you certainly have heard from some scientists, although not uniformly, that there really isn't a problem and that we won't know whether there is a problem until we have had many more years of study and analysis and many more autopsies in order to establish it.

I think it is best to appreciate this difference between the lawyer and the scientist by an anecdote.

If, at this moment, into the committee room were to walk a small two-legged bird covered with white feathers, waddling, making quacking noises, and you were to ask a lawyer in the room what do we have here, the lawyer would respond, looks like a duck, walks like a duck, quacks like a duck, it is a duck.

If you ask the scientists who want us to have more evidence, they would take out a knife, slice off the head of the duck, perform an autopsy and announce to all of us, it is a duck.

Now, the question really for the congressional people who are investigating this problem is, do we want to wait until all the ducks have had the autopsies done on them and find out that they were entitled to victims compensation or do we want to address the problem when all reasonable people would say it looks like a duck, walks like a duck, quacks like a duck, it is a duck.

I think, in terms of this issue, we know that we are not dealing with ducks. There are real victims out there.

Traditional barriers now exist to the recovery on behalf of those victims. But there are already moves afoot to deal with those problems. First, on the legal side, when you have a hazardous waste dump you end up with a substantial amount of waste in it from a variety of different sources. Traditionally, one might have difficulty trying to figure out precisely whose chemical got into the body of the victim and caused the damage.

Theories of joint and several liability, which provide that where the damage done to a person is indivisible from a number of different sources, the plaintiff is entitled to recover from one, the full amount of the damage and then allow that one to recover from all the other contributors, help to address the problem.

Strict liability deals with the problem that maybe the company at the time that the damage occurred tried to do the best it could and that may be a satisfactory answer as between that company and its insurance company, that company and some other company, but as between the company that puts the ultrahazardous substance into commerce and the victim who suffers from it, the law has said the liability should be strict so that the cause of action can prevail even if the plaintiff is not able to trace back and prove negligence.

Then there is the problem of causation. Once we know a particular chemical has gotten out of the chemical waste dump and has reached the plaintiff, does the plaintiff prevail if it cannot prove conclusively to a medical certainty that the cause of the illness was the particular exposure. There are some cases beginning to evolve that recognize that the concept of risk may be taken into account and that a plaintiff can recover if there is strong evidence that they have been exposed to a significant risk of an illness even if it cannot be proven conclusively that they have gotten the illness as a result of the exposure or that they will get the illness as a result of the exposure.

All of these legal developments move slowly. There are different courts in the country which are more likely to recognize them than others. But they are there. There is some development in that direction.

One of the issues that has to be faced in an investigation such as this one, is the issue of whether it makes sense for the Congress of the United States to recognize the justice of that trend and to ride that crest by writing legislation which adopts the end conclusion of where that litigation is going.

I submit that it is an appropriate step for the Congress to take when you are dealing with the appropriate level of damages.

The kinds of damages that people suffer as a result of exposure to toxic chemicals include property damages, direct human health effects which manifest themselves in large medical bills or loss of earnings or both. What we would call objective damages. They also suffer real pain and suffering, loss of consortium for wife or husband who loses their spouse, loss of companionship for a family that loses a child, and in appropriate cases the conduct of the company who has disposed of the waste is so egregious that punitive damages are there.

But all those damages, punitive damages, pain and suffering damages, loss of the intangibles, are really not objective damages. You cannot set a dollar amount and be confident that you had made it appropriate for every case for either pain or suffering, or punitive damages, or any of the intangible losses.

That suggests to me that there is a reason to have a system which has two separate tiers to it. One in which compensation is essentially guaranteed without fault on the basis of a fund of money created by the companies whose conduct has caused the problem in which individuals can come forward and subject to much modified rules of proof and much modified standards of law are entitled to recover for their objective direct undoubted losses, medical expenses, property damages, costs that are of that nature.

Then separate from that allow the private tort system which is evolving to recognize this problem, to continue that evolution and, where appropriate, to proceed to provide relief for the intangible damages, pain and suffering, loss of consortium, other intangible losses and, in appropriate cases, punitive damages.

The two-tier approach, as it is known and as you will hear from the 301(e) study panel that follows my testimony this morning, allows for both systems of justice to operate, both systems to take acknowledgement of the differences between the types of recovery available and the needs.

We are also seeing some modifications in the thinking about medicine as well as about the law. You have already heard from two of the proponents of different points of view, Dr. Karrh who represents the more traditional, let's do the autopsy on the ducks approach.

Dr. Carnow, who is a very famous and well-regarded medical professional who is looking at new ways of finding the evidence that there are toxic chemical damages in humans that can be traced directly to particular exposures and which themselves can be shown to be the precursors of serious illnesses in the human body, including cancer.

Those medical experts will become important parts both of the victims compensation scheme, and of the subsquent tort litigation which is followed in those cases where appropriate in the private bar.

Finally, and most importantly, I want to stress that this task from the victims' perspective is not an easy one. What we talk about easily here in terms of tracing the chemical from the dump site to the water supply and from the water supply to the victim and find within the victim the evidence of that chemical and the damage done to them, is an extraordinarily expensive matter. It is not done even with thousands of dollars but measured in tens or even hundreds of thousands of dollars of costs.

Any system that the Congress wants to set up which is designed to provide relief for the victims has to recognize that if the door to victims' compensation is open only to those with substantial wealth, it is essentially closed to most of those who are suffering.

What I have proposed and discussed in my prepared testimony which I submitted a couple weeks ago, is that the system should have a plateau of evidence, at a relatively low level, which entitles the victim who is seeking compensation to the resources necessary to develop the remainder of the evidentiary side of their case, that it means nothing to the victim to be told "if you can prove with a Dr. Carnow, and if you can prove with the most sophisticated toxicological evidence, and if you can prove with a groundwater monitoring survey that you were exposed to this particular substance as a result of leakage from this dump, we will give you recovery," but does not give you a way to have resources to do that, then there will be no recovery and no victims compensated as a result of it.

If, however, the plateau provides that the first compensation to the victim, which may be compensation not to one but perhaps to a 100 or 200 potential victims living in the vicinity of a leaking dump, if the first plateau provides the resources for the studies to be done to determine "Is this dump contaminating the drinking water supply or the air or the soil" and then second, through epidemiological work and the work Dr. Carnow does, whether or not certain individuals can be shown to have gotten their illnesses as a result of those exposures; if that work can be done first from the victims' compensation scheme, then the opportunity to make certain that true victims are compensated and that all victims are given the opportunity for compensation, will be guaranteed. Without it, the victims' compensation scheme will be nothing more than one more victimization of the victims.

Thank you.

[The statement of Mr. Roisman follows:]

TESTIMONY OF ANTHONY Z. ROISMAN, BEFORE THE SUBCOMMITTEE ON COMMERCE, TRANSPORTATION, AND TOURISM OF THE HOUSE COMMITTEE ON ENERGY AND COMMERCE

Mr. Chairman and members of the Subcommittee, my name is Anthony Z. Roisman. I am presently an attorney in private practice and among my clients are many families who are the victims of exposure to toxic substances that originated from hazardous waste disposed of in a manner which at the time of the disposal was characterized as "current industry practice" or "state of the art technology." I was previously Chief of the Hazardous Waste Section of the Land and Natural Resources Division of the Department of Justice. I appear here today to support this Subcommittee's longstanding effort to provide some form of compensation for the victims of state of the art hazardous disposal technology.

In 1976, when Congress enacted the Resource Conservation and Recovery Act (RCRA), it found that "disposal of solid waste and hazardous waste in or on the land without careful planning and management can present a danger to human health and the environment," that "inadequate and environmentally unsound practices for the disposal or use of solid waste have created a greater amount of air and water pollution and other problems for the environment and for health" and that "hazardous waste presents . . . special dangers to health." 42 U.S.C. 6901(b)(2)(3) & (5) (§ 1002(b)(3) & (5) of RCRA). Among the evidence Congress gathered prior to enactment of RCRA was the shocking revelation that persons living near the Love Canal hazardous waste dump in Niagara Falls, New York, had an abnormally high percentage of a variety of serious health effects and had been and were continuing to be exposed to significant levels of contamination from Love Canal.

By 1980 when Congress enacted the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund) there was broad acceptance of the fact that hazardous waste not only can but already had caused serious and widespread health problems. By that time Love Canal had become the focus of public awareness of the dangers of hazardous waste and Congress referenced the studies showing increased levels of miscarriage and birth defects among the Love Canal families.

Today, seven years after the Love Canal disaster became a matter of knowledge, after enactment by Congress of two major pieces of legislation specifically addressing the hazardous waste problem and after it became obvious that Love Canal was not only a tragedy but the first signal of a nationwide health crisis, not a single victim of that tragedy has received any compensation for the health damages they suffered and continue to suffer. Nor have any other victims of hazardous waste been compensated for the damages they suffered—not in Times Beach, Missouri; not in Dallas, Texas; not in Midland, Michigan; not in Jackson Township, New Jersey; not in Hardeman County, Tennessee. To those who may consider being uprooted from your home, placed in motels and trailer camps, fed at diners and fast-food restaurants and paid a sum of money for your family home which is inadequate to enable you to purchase a comparable home as compensation, let them offer to trade places with the victims who have been so "compensated."

It is a cruel irony that the victims whose suffering produced the groundswell of support for legislative programs have yet to benefit from this legislation. Enormous pressures from the chemical, hazardous waste and insurance industries persuaded Congress in 1980 to duck the victims compensation issue in order to enact at least the clean-up provisions of Superfund. The Section 301(e) study mandated by that law has been completed and its diverse membership endorses the need for victims compensation legislation. This is legislation which cannot be denied and which has already been too long in coming.

As this subcommittee continues its deliberations on this legislation, several key concepts will emerge as pivotal. Today I will address some of these concepts.

First, where should the legal presumptions lie under the compensation legislation? In this world of imperfect knowledge we will inveitably have to face points of decision where we cannot be certain we will be right—but nonetheless a decision must be made. In no area is that concept more relevant than compensation for victims of exposure to toxic substances. Many distinguished scientists correctly note that our knowledge in this area is still limited and that it is often difficult if not impossible to prove to a scientific certainty that a particular illness was caused by a particular toxic substance which had its origin from a particular source. As I will discuss later, there are some exciting breakthroughs in this area which greatly expand our available knowledge, but for the moment let us accept the point put forward by these distinguished scientists. We still must answer the question what should society do in the face of this inadequate knowledge particularly when we do know as a matter of scientific certainty that some toxic substances do in fact cause some illnesses and that there are identifiable sources of such substances?

It is at this point, when society attempts to decide what action to take, that the controversy arises. Those who in some way have a connection with the production, use, disposal or insurance related to the toxic substances quite understandably argue that compensation to victims should not be allowed (at least not if they have to pay for it) until we know as a matter of scientific certainty that the substance caused the particular illness and had its origin from a particular source. These same interests make a similar argument against setting standards for pollution control and favor instead the continued exposure of persons to the polluting substances until the sheer weight of the body count establishes the scientific certainty which they demand. Congress has properly rejected this approach for establishing pollution control. It has realized that we cannot tolerate the use of the public as a guinea pig and thus hazardous waste and other environmental and health laws establish standards based upon avoidance of risk. The fundamental principle at work in these statutes is that while adherence to risk avoidance may unnecessarily control a pollutant or exclude a product the harm caused by occasional "over-regulation" is substantially less than the harm which would be caused if a highly dangerous pollutant or product were allowed to be widely distributed and the feared risk were realized. In short these laws place the presumptions in favor of human health over corporate profits.

Victims compensation laws should do the same. Once it is established that a person has a genuine illness which has been shown to be of the type caused by toxic substances the presumption should be that the person is a victim who is entitled to compensation. Once a particular source is identified at which the toxic substance was present, from which it escaped and that the victim was in an area which could be reasonably expected to be affected by the substance then the presumption should be that the source caused the victim's illness. If the source can establish that the illness was not caused by the substance or that it was not the source of the victim's exposure, then the source would not be liable. In short the burden of scientific certainty should be placed on the person best equipped—financially and technologically—to carry that burden. To place on the victim a burden of proof that the victim is financially incapable of meeting is to make a mockery out of the concepts of due process of law and equal justice under law.

This brings me to my second point. Obviously a potential victim must be able to prove something to be compensated by the system. How does the victim obtain that information? A properly taken and thoroughly analyzed well water sample will cost $1,000 to $3,000. Medical tests and diagnoses can add thousands more. A groundwater study to connect the local dump to the polluted well will run $25,000 and up, depending upon the complexity of the problem. Epidemiological studies to demonstrate that among those exposed to the toxic substances there is a statistically relevant increased level of the illness suffered by the victim will add $50,000 or more. Lawyers taking toxic tort cases are anticipating a minimum of $100,000 to $200,000 in costs just for gathering the scientific and medical information required to prove a claim.[1] Unless a victims compensation law addresses this problem, the law will be nothing more than a cruel joke—offering compensation to the handful of very rich people who happen to be living near a hazardous waste dump. Even presumptions cannot solve this problem but there are at least two approaches which I believe warrant consideration:

1. Establish a very low threshold standard for a victim to meet—i.e. illness of the type caused by a particular toxic substance and a possible source of the toxic substance in the area—and once the standard is met the victim will be entitled to fund compensated scientific and medical investigations. In any recovery by the victim the amount of these costs would be returned to the fund. In this example the compensation fund becomes a revolving source of financial aid.
2. When the same threshold is met either require the suspected source to pay the cost of investigation or to conduct, under independent supervision, the necessary investigations. In this example we extend to victims compensation the same standards used by the federal government in enforcing § 7003 of RCRA (the imminent and substantial endangerment provision) or in exercising its authority under § 3013 of RCRA (ordering data gathering by the source based upn a possible substantial hazard).

These are not exhaustive, but illustrative. The principal point I want to make is that the victims must have access to the financial wherewithal to prove their point or they will be certainly shut out of the system.

My third point relates to the role of the state common law system in providing redress to victims. I am a great fan of the common law because I believe it embodies the fundamental principle upon which this democratic society is based—justice. Unconstrained by statutory language and legislative history a court and/or jury deciding a case under the common law is basically asked to listen to the facts, decide where justice lies and render a judgment accordingly. Large corporations are not surprisingly frightened by this system because once the individual has the resources to prove his facts the corporate leverage of money, political influence and sheer power is gone and the two litigants are essentially treated as equals by the judge and jury. While I am not surprised that industry seeks to avoid this system of basic justice (such as urging Congress to adopt product liability or victims compensation legislation that will essentially eliminate the common law in that area), I feel it is important to realize what motivates that position.

At a conference on toxic torts two years ago, representatives of the chemical and hazardous waste industry urged adoption of a form of victims compensaton legislation that would essentially replace common law with a statutory scheme and their principle emphasis, echoed by the insurance industry, was unless such a scheme existed there would be no economic certainty. In short, the industry wanted to know up front what its liability would be so that insurance premiums and product prices could be established accordingly. Not only does this approach turn human suffering into nothing more than a budget item and encourage cynical judgments to let a certain number of people die because the corporation "can afford it" but it unfairly shifts all the risk of uncertainty to the public which does not know who will be injured and how seriously and who will die. Surely before we start giving economic certainty to those who cause these problems we should provide certainty to the public about the nature and extent of the harm that will be caused.

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[1] These costs would not have to be incurred for each individual, but rather for each affected area. Of course if 1,000 people over a large area were affected the costs would be higher but for several dozen families in a single neighborhood with a nearby dump site these costs should cover the factual development of their case.

Thus a victims' compensation scheme should preserve state and federal common law to allow for recovery, where appropriate, beyond that provided by the compensation fund. The formula recommended by the § 301(e) Study Group is one which I feel would accomplish this result.

Should some of the liberated standards and presumptions available under a victims' compensation be included as new standards under common law? I am inclined to think not *if* the system will provide victims with the resources necessary to gather the relevant facts. Emerging doctrines of common law are, in some jurisdictions, beginning to recognize that scientific certainty is not a pre-requisite to recovery. New scientific techniques such as the work of research immunologists like Dr. Alan Levin of San Francisco conclusively establishing the presence of chemically induced immune system damage in blood samples of victims are making it possible for victims to meet their evidentiary burdens if they have the resources to make their case. However, if the resources to prove the case are not available, then the common law principles must be modified to shift the burden in order to preserve the opportunity for justice.

Finally, I do favor the principle that the federal law should clearly provide the opportunity to bring a toxic tort in federal court as a federal common law right of action. The potential for uniformity of development of common law principles and the stature of the federal judicial involvement in these issues if vital. It would be anamolous were Congress to properly recognize hazardous waste victims as a class deserving federal attention but deny them access to federal courts to redress wrongs not fully compensated by the fund.

Thank you for the opportunity to appear once again before the subcommittee. I will be pleased to answer any questions you may have.

Mr. LEVITAS. Thank you very much, Mr. Roisman.

Let me get to the question of scientific certainty on the one hand, and what you said, a lawyer's identification of a duck on the other hand.

I think you are—you do well to make such distinctions because there are two different disciplines involved and what scientists are seeking for their purpose may be somewhat different from what attorneys or courts or legislators are seeking on the other hand.

But let me get to the business end of it. Based on your experience in dealing with manufacturers of pesticides or other chemicals and marketing of them, is it your experience that these manufacturers demand scientific certainty as to all aspects of the efficacy or health effects of a product before it is actually marketed? Or are they willing to act upon information that they feel is of a high degree of probability as to either efficacy or health effects?

Mr. ROISMAN. I think it is clearly the latter, Mr. Chairman. In some way the whole dispute, if you will, if I could frame it in a legal perspective, between whether the victim is truly a victim or not, comes down to who has the burden of proof. If the presumption is that the person is a victim until they are proven not to be, then every victim will recover.

If the presumption is that the victim is not a victim until they can prove as a matter of scientific certainty that they are then essentially no victim will recover; or very, very few.

In the context of your question, pesticides, the premise of the industry applicant when they come forward with a drug or with a chemical for approval by the various Federal agencies is this is

OK unless you can prove that it is not. All the uncertainty is resolved in favor of saying it is all right.

Later down the line, if it turns out that we are wrong, you can always recall it. You can take it back. You can remove your approval.

That approach is not the approach taken down the line when someone says "I got it; I got the damage that you said would not occur." Then all of a sudden, the standard changes. Now it is required that you must come forward and prove that you are the one who is right.

So I think there is a double standard, if you will, that is applicable here.

Mr. LEVITAS. You mentioned about the 2,000 sites which EPA has already classified as imminent danger to public health. When you were at the Justice Department, did you have occasion to evaluate or take action or elect to take action with respect to any hazardous waste sites, or did that come in after you left office?

Mr. ROISMAN. No, my job was to do just that. At the time that my section was set up in the Justice Department, which was in October of 1979, the Environmental Protection Agency had already established a hazardous waste task force whose job it was to galvanize the regional offices of EPA to identify those hazardous waste sites in their jurisdiction, which presented imminent and substantial endangerments, to work those cases up, refer them to the Justice Department; and our job then was to take those that warranted legal action and take them to court.

I don't want to mislead you at all; we did not begin to make a serious dent in the 2,000 in the first year in which our section existed; we filed against 51 in that time.

Our first year ended just before the elections of 1980 and from that period, for the next 18 months, there was a total of, I think, 10 sites filed against, and, as of June 1981, until the end of the summer of 1982, there were no sites filed against by the EPA. There were no referrals to the department. I left the department in January 1982, primarily because there was no more work for me to do.

Mr. LEVITAS. Mr. Roisman, as an attorney, would you describe the cases that you dealt with as cases which were overall—not just from the legal point of view, but from the evidentiary point of view—rare, complex and difficult cases to bring? What I am getting at is, Mr. Roisman, based upon your position in the Justice Department, you had at your disposal a large amount of assistance—legal talent, research, experts, scientists, toxicologists, investigators—at your disposal.

Now, is it feasible or reasonable to accept that a private attorney handling such litigation would have the same resources at his disposal?

Mr. ROISMAN. No; not at all. If you will bear with me, let me just take you through the steps that the EPA and the Justice Department would go through to bring one of these suits, because I think it is illustrative of the problem.

To begin with, the Department and EPA then had—and still have—no authority to seek any compensation on behalf of victims. So, the whole side of the issue that goes to, did a particular person's illness get caused by a particular exposure, was not even part of our scientific investigation.

We did deal with the issue of risks because, depending on „the level of risk, you can determine what relief was appropriate.

But we never went that additional step and tried to link up an illness with a particular exposure.

Usually, the EPA would start with some kind of a paper notification; it might come from the State agency or a private citizen that said there is a dump somewhere out here, and the place would be identified.

EPA would send a team, usually made up of a couple individuals with scientific training and at least one person who had some special skills in doing analytical work or in gathering of samples. They would go out and take a look at the site.

If there was anything apparent offsite on nonprivate property, they would take a sample and take that back to a laboratory and run a scan on it, an MS/GC scan.

Those scans are costing the Government about $1,000 apiece in their own laboratories.

From that scan they would get information that would tell them whether, in fact, it appeared there was leakage coming off-site. If there were, then the next step would be either to seek permission or a court order to go on the site to take samples of what was in the dump, itself, and then to begin to drill monitoring wells around the site to try to trace where the contamination may have spread to, to figure out how much was coming offsite and what pathways it was on.

Each of the wells would cost several thousand dollars to drill; they had to be done in a particularly sound way, because the evidence gathered from the wells would be subject to challenge in court. The information that was gathered from the wells would be more samples, and each of those would go back through the same kind of a scan, although at this point you might be able to reduce the $1,000 cost because, instead of looking for 122 priority pollutants, you may only be looking for 10 or 15 that the first sample told you, or that your examination of the site told you.

After those investigations were completed, EPA technical people would evaluate the magnitude of the hazard. If they felt the hazard was serious enough that it warranted cleanup, they would bring in the Justice Department lawyers, and the EPA lawyers, and we would begin to apply the legal standards to the evidence available, and with that amount of information we would bring suit in Federal court.

The suit, itself, would then involve further investigations, and it would depend on the magnitude of the site and attitude of the defendant. A defendant who chose to fight quite hard, we would have to incur substantial additional expenses. Sometimes we would bring in the National Investigation Enforcement Center, which is located in Denver, and is EPA's creme de la creme of the technical resources, to do the ground water analysis.

Sometimes a full ground water study would have to be done because you have complicated aquifers.

We estimated that a minimum of $10,000 to $15,000 would have to be spent on any site to get enough information to even know whether we should bring a lawsuit or not, and the costs beyond that were really difficult to estimate.

But they could be quite high. All of those costs would be incurred by the Federal Government, and, I might add, that to the extent the Government brings enforcement actions with respect to particular sites, it provides a substantial amount of the information that any private citizen might need.

To the extent the Government does not exercise its enforcement authority, it not only fails to do its job, it makes it markedly more difficult for private citizens to get their recoveries as well.

Mr. LEVITAS. I have a number of other questions, but in the interest of having the other members participate, at this time I will recognize the gentleman from New York, Mr. Molinari.

Mr. MOLINARI. Thank you, Mr. Chairman.

Mr. Roisman, who are the Trial Lawyers for Public Justice?

Mr. ROISMAN. It is a public interest law firm that was established by trial lawyers to engage in litigation that would set important precedents in all areas of plaintiffs' trial law, and particularly cases that would not normally be handled by the private bar, either, because of the difficulty of the issues presented, the costs involved, or both.

There are about 350 trial lawyers who have contributed to the organization. We also receive some support from foundations.

The Trial Lawyers for Public Justice is a taxable professional corporation in the District of Columbia, and it is a project of the Citizens Legal Clinic, which is a tax-exempt organization founded in Tennessee, and has its principal offices in the District of Columbia.

Mr. MOLINARI. I take it that there are incidents of toxic poisoning, where plaintiffs may have a difficult time hiring a lawyer, which this group would look at as well?

Mr. ROISMAN. We have one case in which the trial lawyer who was retained called us and asked us if we would participate in the case with him because of the difficulty of the case, and so among our cases there is one that is of the type that we are discussing here today.

Mr. MOLINARI. If you would turn to page 6 of the testimony that you provided us with, at the top of the page you have a sentence that has a great deal of significance, and it is: "A properly taken and thoroughly analyzed well water sample will cost $1,000 to $3,000."

Would you define for us what you mean by "properly taken and thoroughly analyzed"?

Mr. ROISMAN. Yes; first of all, at the time the sample is taken, it is not as easy as simply going in, turning on the spigot in someone's home and taking a cup and filling it with water and taking it over to someone who has a lab.

If you are looking for volatile organics, you have to make sure, when you have taken the sample, that you seal the lid, or you lose the volatiles.

Second, the container must be clean, and frequently if you just go to a regular laboratory, what they clean their containers with may, in fact, be contaminants in terms of what we are concerned with, like benzene.

So, you want to be sure it is cleaned in a way that it is not self-contaminated.

On top of that, as a legal matter, you have to be sure the method by which the sample is taken does not subject it to damage in cross-examination at some subsequent time down the way.

So, you must make sure that you have run the faucet long enough that there is no danger that you are inadvertently trapping something in the water that should not be there; that perhaps someone has checked to be sure that the faucet is the right place to do the test. Maybe it should be done at the intake to the house so you don't inadvertently contaminate the sample with something in the pipes of the house, but having nothing to do with anything external to the house.

You have to do a chain of custody. You must be sure that the sample has been taken and under the control of an official who was able to testify that no one tampered with it, no one added anything to it from the moment it was taken until the moment it has been sampled and analyzed in the laboratory. So, that is the start-up side of it.

In the laboratory, itself, depending on the sample you have—it could be a water sample as we are discussing—you have to take a piece of that sample; it has to be subjected to the MS GC scans, which, as I mentioned before, the government was paying about $1,000 a scan. Private citizens are being quoted prices of $1,500 to $2,000, depending on where they are going.

A complete scan, thorough analysis, would mean that not knowing anything about the water except you want to know if there is anything in it, you would at least scan for the 122 priority pollutants that EPA has listed.

That is an expensive process, because the scanning mechanism basically produces a graph, and when you look at the graph, you are looking for certain peaks which correspond to what that machine has predetermined represents particular chemicals.

So, for 122, you are looking for different peaks. There is a fair amount of time spent in the lab checking the peaks found from the sample against the peaks established for that particular machine for the chemicals. I might add the lab must exercise a certain amount of control over the samples so that if someone wants to resample the same sample to the extent that it can be saved—volatiles are much more difficult than others—it is available to be resampled by someone.

Mr. MOLINARI. So if a citizen or group of citizens that live near a dump, suspected that there was a problem with water contamination, a sample could be taken with due care that all the custody and control techniques were proper. What would it cost to determine what is in that water?

Mr. ROISMAN. What I have suggested here is $1,000 to $3,000. It depends. Sometimes you get a lab, if it is in a university that may do it for less, or a person taking the sample may have access to an equipment lab where they don't have to pay as much. What I advise people to do is to try to find a less thorough scan that will just give them a quick look at whether or not there are organics, for instance, or whether or not there is evidence of some kind of contamination in the water.

Those scans can run anywhere from $60 to $250. They don't tell you what is in there; they tell you whether there is a likelihood that there is something in there, and then you can make the larger investment, or maybe if you are fortunate the existence of that will be enough to persuade either a State or Federal agency to run the remaining test for you.

They would use that—I must add, a fair amount of press coverage—to awaken the bureaucrats to the need to do the sample. Often, when you send the sample from your home to the State lab, they will come back and tell you your water is clean, which means they did not find any fecal coliform in it. It is potable. It does not mean that it does not have toxic chemicals in it.

Mr. MOLINARI. This is a very serious point that we don't seem to see emphasized very much throughout the country. That is, the threat to the individual who uses well water, even in New York City, where we don't rely on ground water for drinking water supplies except during drought periods when thousands of wells may be put into use. In many, many areas we have toxic contamination, and I have been very concerned about the fact that people will take a sampling to a laboratory which finds it negative for bacteria and viruses, but no one tests for toxics. Obviously, people think it is safe. I have serious concerns as to the potential for an outbreak of illness.

We have, in my district, a 6-year-old girl dying from a condition we suspect might have been caused by irrigation of a golf course across the street from her home with seriously contaminated water.

When you served with Justice, would you have characterized the emphasis of the Department during that period of time as one that would prefer to litigate or to settle?

We have heard that bandied back and forth during the current administration. What was your experience during your tenure?

Mr. ROISMAN. I was there for 1 year under the current administration and for about 1½ years under the previous administration. Under the previous administration the position which I frequently stated was that the Justice Department and the Environmental Protection Agency were anxious to reach settlements in every possible case we could reach them in, and the only way we could be assured that we could reach them—and reach them on terms acceptable to our goals of getting proper cleanup of the sites—was to litigate.

We made crystal clear to the prospective defendants that we would not tolerate the failure to negotiate quickly and completely. When we did not get settlements, we sued. When we gave drop-dead dates, as that term was used—not in our administration, but in the current one—we did not fail to meet those dates, and we did not give them lightly. When we took a case to a defendant before filing suit, which we did frequently, we already had a complaint; we had it approved at the Assistant Attorney General level in the Lands Division, which meant that it was approved for filing. We usually had it already at the U.S. attorney's office waiting to be filed; we had its approval from the EPA, from their Enforcement Director; and then we would write a letter to the defendant, and we would say within 10 days we intend to file suit against you, but if you wish to talk about resolving this matter, we will be happy to do it.

Within those 10 days, it would be obvious whether they were prepared to reach reasonable terms with us. If so, we would extend it. Once, we extended it as long as 30 days and reached a settlement with the Syntex Agribusiness Co. in Missouri. I mention that both because I think that it was an example of how the two parties came together, and also because it is the Syntex Agribusiness site from which the dioxin that contaminated the rest of Missouri originated. The settlement we dealt with was on one place where the dioxin was disposed of.

Mr. **Molinari**. Let me interrupt, if I may, because we are limited in time on the first go-around.

We have heard conflicting reports during the subcommittee's investigation of the EPA and the Superfund. Rita Lavelle was characterized as one who favored settlements. Yet when she testified before us, she was highly critical of Mr. Perry.

Mr. **Levitas**. Who she claimed, and others claimed, had a total emphasis on sue, sue, sue, and she felt that oftentimes that would interfere with the ability of the Government to settle cases where they were less than perfect.

Indeed, we had a memorandum that I had obtained, which was signed by—prepared by—Mr. Yamada. Are you familiar with him?

Mr. **Roisman**. I know Mr. Yamada.

Mr. **Molinari**. And Mr. Yamada, who is still in EPA's employ, sent a letter to all 10 regions of EPA, holding a gun to their head, demanding that they refer cases to headquarters for litigation and, in fact, went on to suggest by a graph that based on the referrals to headquarters, the allocation of full-time employees to each of the regions would be determined. I come from region 2, and they had, I think, 27 full-time employees; based on the slow referral rate they were going to get 12.

So I am having difficulty in my mind trying to equate what you are saying, what Rita Lavelle was saying, and what Mr. Yamada was saying, and, in fact, the memorandum.

Mr. **Roisman**. Perhaps the best I can do is sum up this way: Starting in January 1981, with the end of the Carter administration at EPA, we basically entered a period of the Amateur Hour. The people who were brought to the EPA to replace the people who had been there before knew nothing about what they were doing.

There is one consistent explanation that I think explains the Agency's actions. It is that they were totally incompetent and did not know what they were doing. Ms. Lavelle thought she wanted to do settlements.

Mr. Perry wanted to do litigation. You can't do one or the other. One is a carrot; the other is the stick. You have to put them together if you want the donkey to move ahead. You have to do both.

I think the EPA, when I helped in the hazardous waste section, was the kind of EPA capable of understanding the subtleties that you had to reach settlements in the atmosphere in which litigation was a known reality and not as it was in this administration, a total nonreality for over a year after it took office. There was no way to negotiate a good settlement.

So, while Mrs. Lavelle wanted to have a good settlement, perhaps, she had no chance of getting it in a conference room when the people on the other side knew that they could get a meeting with the Deputy Administrator, with the Administrator—maybe at the White House, for all I know—and get new conditions.

So, you never had an effective program going. That, I think, is the source of the problem. It is difficult to try to explain away all of the inconsistences in that administration, because I think that they are not explainable except if you start with the premise that most of those people did not know what they were doing.

Mr. MOLINARI. Would you advocate a system where, as soon as Justice had adequate facts, it would institute a suit and at the same time pursue negotiated settlement?

Mr. ROISMAN. The Justice Department position—the first major case that I had, that issue arose, and we had a defendant that said to us, please negotiate with us for 6 months; don't sue us; and we went to the then Deputy Attorney General of the United States, and we asked him his advice. He said the Justice Department does not condone the practice of forestalling litigation for an indefinite period of time that is ready to be filed in anticipation of settlement discussions.

We file the suit, and if there is a settlement discussion which will go on, it will go on after the suit is filed.

Mr. MOLINARI. Isn't there a very distinct possibility that, in the case of some large corporations who value their reputations, they would have more reason to want to settle prior to suit; that once the suit is started, and their name has been exposed, they might as well, if they feel they have a decent case, fight you all the way, and you have lost your opportunity to negotiate a settlement?

Mr. ROISMAN. That is why we normally send a letter and offer a 10-day period of time during which time we could evaluate——

Mr. MOLINARI. Is 6 months an unreasonable period of time?

Mr. ROISMAN. It was in this case. By the way, the case did settle.

Mr. MOLINARI. In dealing with a major corporation who you suspect does not want to have its dirty linen aired, and it says it would like to have 6 months to try to effectuate a settlement, as a general rule——

Mr. ROISMAN. First of all, you must understand that the government's case has probably been 6 months in the development by that time, from the time that that first investigator went out there and began to take samples.

Usually, a company who genuinely wants to settle will have made telephone calls already. We don't have to initiate the settlement discussions. If we have gone through that process and it is already 6 months down the road, we have not heard from them, we, as a courtesy, send them a letter and say, look, if you want to talk, we will give you 10 days. If in that 10 days we felt they genuinely wanted to settle, and there was good reason why we could not get the settlement completed in the 10 days, as there was in the Syntex case I mentioned to you, we would postpone. We did that. We waited 30 days. The night the settlement was reached, the team of negotiators stayed up all night, because the next day we had said we were going to file this suit.

Mr. MOLINARI. Interesting.

Mr. ROISMAN. I think it is important to remember that the two parties to this don't have exactly the same goal. If you don't keep their feet to the fire, you are not as likely to get a successful settlement at the level that you need in order to get adequate cleanup.

Mr. MOLINARI. I yield to the gentleman from Florida.

Mr. SHAW. What you are saying is that during the investigation period, a certain amount of negotiations have been ongoing?

Mr. ROISMAN. Sometimes. In the investigation period, when I was there, the Government did not make any overtures. We did not go to the company and say, "Hey, we are investigating your site; would you like to talk about settlement?"

Frankly, we did not know whether—we did not know whether we had a case and whether there was anything to settle. But the company usually knows a lot more than the Government does about what the situation is.

Sometimes the company will come to us in that time and say, let's start talking, and we would talk and investigate.

Mr. SHAW. I am a little confused, then. Are you saying that an adversarial relationship is established immediately upon the start of the investigation rather than the parties working toward an end result to get the problem cleaned up?

Mr. ROISMAN. No; adversarial relationship is not my term. What I would say happens is that the Government starts the process of investigating the site. At that time, the company can come in and begin to cooperate with the Government and say, "Look, we want to get the same answers you do, and we want to do the right thing based upon those answers," and we say, "Fine, let's cooperate." We might take split samples; we might go to certain places, open up the records and see what wastes have been dumped in the site, so we don't have to spend time sampling the site.

Mr. SHAW. Was that the usual situation, or was that the exceptional situation?

Mr. ROISMAN. That was—certainly in my tenure that would have been considered the exceptional situation.

What we found was that the company's interest in settlement usually began to rise first after we had enough information to believe that a case should be brought, and, second, after we had been in existence for a number of months and they realized we really were going to bring the cases.

Mr. SHAW. Don't you think that a little evolution may have occurred over the last few years as the toxic wastes problem has become readily recognized as being as severe as it is, that maybe the atmosphere may have changed to where companies, responsible companies, are much more willing to expedite a solution to the situation rather than look down a gun barrel?

Mr. ROISMAN. I hope so. But our premise was, and if the authority were mine to exercise again, my premise would still be that you must pursue settlement and litigation on parallel tracks, not on the same track.

One does not wait for the other. You must continue to prepare for litigation on the premise that if the settlement falls down, you are ready to go.

We were dealing with sites we had already determined were imminent and substantial endangerments. We did not feel it would be responsible to delay.

Let me just explain in my last months in the hazardous waste section, I proposed, but had no success in getting approved, an increase in the size of my section to set up within it a group of lawyers who would do nothing but negotiate. I believe it is difficult for the same lawyer to litigate and negotiate both. I think it takes a different kind of lawyer to do it, and the way you walk into the door of the prospective defendant alters, and that is, in the most classic sense of what we see on television; you know, a white hat and black hat. You need the nice guy that says I don't want to turn this other guy loose on you; he is trying to get into court; he likes to litigate. I want to negotiate and settle.

I think that that would have been an effective way to address your concern, which is that you do not want to litigate unnecessarily, and you don't want to negotiate at a level where you do not get what you need.

So that you can accommodate both in that way. For us, I think it was working.

Let me premise all that; that whole situation only existed for about a year, from October 1979 to the end of 1980. After that, it was entirely different. My section was abolished in June 1981, and the flow of cases and cooperation between the department and the EPA essentially ended. So, we don't have a substantial amount of experience with it, but it was working, I think.

Mr. SHAW. I did not mean to take up that much time.

Mr. MOLINARI. That is OK.

What I would like to do, if I could, is ask you to put your present hat on now and take us through a scenario as to what happens when you, as an attorney, have clients who come to you and say they have been suffering, they and their family, from a variety of symptoms which are readily recognizable as being the result of exposure to a toxic dump site?

How do you evaluate whether you are going to take the case? What factors go into it? Let me ask you this as a preliminary question: You have handled a number of those, I assume?

Mr. ROISMAN. We have only one in the office now. We have one other which is a substantial one under investigation, and we have consulted on some.

Mr. MOLINARI. All right.

How would you evaluate whether you are going to take a case?

Mr. ROISMAN. The first thing we do is see to what extent the Government has already done some investigation. If somebody walks in and says, I live near a certain site; the Government brought a suit against it, and we would get, either by cooperation with the Government or under the Freedom of Information Act, or from the court files, whatever evidence had been gathered to try to show what the hazard was at the dump, where it went, and all the rest.

Second, we would attempt to determine how the person was exposed. Did they have a pathway of exposure? If the dump caused only underground leakage and this couple lived upgradient from the site, it would have been difficult to see how they could have been exposed that way.

Then we would attempt to get the help of technical experts who could evaluate with us whatever information was readily available. If it appears that they were downgradient from the site, but we could not establish whether their own drinking water was contaminated, we may have to go out and get a sample, if the resources were available to do that, and find out if there was contamination of the water supply, and if it was of the same kind of chemicals in the dump.

You could have the contamination, and it could have no connection to the dump site.

We would have to check with the patient's doctor to see whether or not the doctor had found any illnesses that seemed to have a linkage to toxic chemicals. The family doctor usually does not think in terms of linkages to toxic chemicals, but he can at least identify the illnesses. Some illnesses are related to certain chemicals.

Some are so closely related that the very presence of the illness is almost a guarantee that the exposure must have been there. But in a more common case it is a class of illnesses such as kidney failure.

Kidney illnesses are related because that is where the body does a lot of its washing and cleaning, and thus if you have taken in a toxic chemical, you are likely to find something in the kidney or in the liver.

So we would do that.

Then we would see if we could find in that State law that was appropriate for recovery. First of all, there is a statute of limitations problem; second, in some States the toxic torts have a much less chance of success than in other States.

So, we would have to make a legal evaluation. The cases are driven by the facts, though, and we operate on the premise—true for good trial lawyers—good facts make good law. If you can't make the case, the plaintiff has no case, you can't win on the law if you are the plaintiff. You can only win on the law if you are the defendant.

So, you have to get the facts.

Mr. MOLINARI. I would like to ask one last question and defer to my colleagues.

From the plaintiff's lawyer's position, isn't it more than good facts make good law? Doesn't it also take good injury, good in the sense that it takes a very substantial injury in order for you to consider handling a case, because these cases, as I understand, are handled on a contingent fee basis?

Mr. ROISMAN. That is correct.

Mr. MOLINARI. Unless the injuries are severe, the investment of time on your part, or your firm's part, is so substantial and the amount of expenses in the investigative phase, laboratory work, and all the rest, is so large that the case may not be taken on. We have been told in this committee that there are few qualified medical experts you can use in this country to put on your case.

What I am driving at is that I don't think the vast majority of those suffering in this country fall into that—hopefully don't fall into that—category where there are severe injuries, severe from the standpoint where we are talking about judgments or settlements that might be in the million-dollar range.

But they have suffered; they have suffered substantially. They have had many medical problems, a great deal of doctor and hospital bills, and yet they don't fit into that category where an individual case would merit a lawyer taking the matter on.

Mr. ROISMAN. I think that is a fair statement.

Mr. MOLINARI. I see this as one of the biggest problems we have to face. By and large, the masses out there who are being injured do not have adequate remedies even under the best of circumstances regardless of what State you are talking about, and whatever body of statutes are in that State. Very simply you just have the difficulty of proving a case which is so complex that you cannot afford to take those cases. That is what we have to address, I think. The masses out there don't have a legal remedy, as I see it.

I just wanted to make that observation. I would yield.

Mr. LEVITAS. Dr. Rowland?

Mr. ROWLAND. Thank you, Mr. Chairman.

Mr. Roisman, you used an anecdote to illustrate the difference between the way a lawyer looked at this problem and the way the scientist looked at the problem.

Mr. ROISMAN. Some scientists, yes.

Mr. ROWLAND. Yes. You said that if a duck walked in and the lawyer would say it walks like a duck and acts like a duck, and quacks like a duck, so it is a duck. The scientist would look at it and before he made the decision would dissect it and look at the inside of it.

Say that a group of people in this room decided that that duck was not a duck. Do you suppose they would have any difficulty in getting an attorney to represent them in proving that that duck was not a duck?

Mr. ROISMAN. Well, I guess, to put it in crassest terms, it would depend on whether they could pay the attorney.

Mr. ROWLAND. That is the point for me there. I appreciate that. But they would if they could afford the attorney, be able to get one that would prove that that duck was not a duck if he could?

Mr. ROISMAN. I think it is a fair statement, and, as an attorney, I don't mind fessing up to it; that lawyers, regardless of how they do it, are hired guns. Some of us draw our guns for more selective clientele than others, but that is what we are. We are paid to provide services, and usually—I have been asked the question; I can't tell you how many times—can I sue so-and-so, and then I hear some story or something, and the short answer is sure, you can sue; you can't win.

I guess there are lawyers—I can't think of any plaintiff's lawyers that would take your hypothetical case because most plaintiff's lawyers are only paid if they are successful.

So, they are compelled to evaluate the merits of the case and whether it could succeed. But defendant's lawyers are different. I bring suits all the time that appear to be defended by lawyers proving that ducks are not ducks.

Mr. ROWLAND. Your anecdote established—your point was that lawyers are sort of together and scientists are really not. That was what I got from your story.

Mr. ROISMAN. I am sorry. What I really meant to suggest to you, and I apologize if I misspoke, is that the legal system as it evaluates the question, evaluates it differently than the scientific system does. Each has a good reason for doing what they do. But we are looking at a legal system approach to a problem, and I think it is important to put the scientific solution into context.

I am not an opponent of the scientific solutions. I don't think they should dominate the question as between A and B, who should recover, because if they do, the one who is entitled to recover may not learn they can recover until it is too late to do them any good.

Mr. ROWLAND. Do you feel there is enough evidence now, based on your present knowledge, to say that toxic chemicals are posing definitely a significant hazard to many people in this country?

Mr. ROISMAN. To me, yes. But I am speaking not as a scientist, but as a lawyer. I have talked with scientists; they tell me that. I believe them; I consider them credible, and I believe that.

Mr. ROWLAND. We heard some conflicting testimony here from two different people during our last meeting about whether or not there is enough proof at this time. Would you think that there is considerable confusion now among the scientific community about whether or not it is a serious problem at this time?

Mr. ROISMAN. I think there are scientists who say that it is not, and there are those who say it is. I don't know whether it is confusion. We are the ones who might be confused by it. I think each body of scientists feels confident they are right.

Mr. ROWLAND. Do you know now if there have been many cases that have been settled by people who are seeking compensation for injuries from toxic chemicals in the country?

Mr. ROISMAN. I am not aware of very many, but I don't have a good grasp on the total of all those in the country.

Mr. ROWLAND. Thank you very much.

Mr. LEVITAS. Thank you, Dr. Rowland.

Mr. Shaw?

Mr. SHAW. Thank you, Mr. Chairman.

I got my main questioning done on Mr. Molinari's time, but I have one more.

You used the term in your direct examination, "scientific certainty." Would you define that term for us, which I assume is the same as medical certainty?

Distinguish it, if it is distinguishable, from the preponderance of evidence.

Mr. ROISMAN. It is easier for me to define it in terms of an example. If you start with a toxic chemical, and you run a laboratory experiment with a set of genetically identical mice, half get it, half don't; you then look at the mice a year down the way, and you see what has happened to those who got the toxic chemical, and you find a statistically relevant number of the exposed mice got some particular illness, and you are then able to say that this chemical appears to cause this illness in these mice. I guess if the tests are run properly, you probably—and if you had a statistically large enough sample to work with—you can say as a matter of scientific certainty that that level of that chemical causes that illness in mice.

Now, when you start to ask the question, well, does it cause that illness in humans at that level or lower level, or higher level, you have gone outside of what scientific certainty demands and a scientist, as I understand, would say in that case, until we have had a comparable human exposure to the chemical at the levels in question and had a statistically relevant sample of exposed and unexposed, we cannot say, as a matter of scientific certainty, that it is true.

That is how I understand that.

Preponderance of the evidence is a legal concept that says there must be at least 51 percent of the evidence which is reliable and credible, which the decider believes favors the particular proposition.

Was the light red or green? You have a witness that stands up and says, I saw it; I was looking right at it, and it is red. Another witness gets up and said I saw it; I was looking right at it, and it was green.

We have no more evidence and both witnesses are equally credible; then we don't have a preponderance of the evidence, because we have half the evidence saying it is red, half saying it is green.

Mr. **Shaw**. Can the trier of fact in a court of law make his determination in favor of the plaintiff based upon the fact that the preponderance of evidence indicates that the rats did die or did contract an illness because of exposure to the toxic waste?

Mr. **Roisman**. You mean, is the jury entitled to make the inference?

Mr. **Shaw**. Yes.

Mr. **Roisman**. In the court system the court might, in the first instance, have to pass on the issue of whether or not that question could go to the jury. Is there enough evidence? The defendant, for instance, might move in our hypothetical to say that it is not reliable and credible evidence; that animals exposed to the chemical in question got sick, to prove whether humans exposed to it had their sickness caused by it.

So you would have a court passing first on the legal question. Some courts, I think, would say, yes, we will let that issue go to the jury. Let them decide, after hearing the other experts testify regarding the reliability of animal studies for predicting what happens to humans.

In other courts they would say, no, we will not let that go to the jury. It is not reliable enough to take it to the jury.

So, in the case where the court allows it to go to the jury, then, yes, a jury, if it decided to also believe the testimony of other experts who would make the linkages between the exposures to the animals, the evidence from that and the exposures to the humans, and what relevance that may have with relevance to the exposures to humans, you would have a jury that says, yes, that is enough evidence for recovery.

In some cases the same court, with a different jury, may get a different result. It will depend on the quality of evidence on both sides. The defendant has experts testifying on the unreliabilities.

Mr. **Shaw**. Do you see an evolution toward allowing this to go to the trier of fact rather than directing the—

Mr. **Roisman**. I would say the trend is more toward allowing it to go to the trier of fact. That is, in part, I think, because so much of what the Federal Government and State governments do in setting regulatory standards, uses the animal study data, the Delaney amendment, is based on the premise that what happens in animals has a reasonable bearing on what happens in humans. I think you have a good chance of getting to the jury.

My own sense is that you have a far better chance in front of a jury if the question you are asking is, did the plaintiff's exposure to the chemical that we know causes this problem in rats subject them to a significant risk of increased illness which entitles them to recover?

There is a better chance that that question will be answered affirmatively than did the particular illness that they now have get caused by the exposure to that chemical which has been shown to cause that kind of illness in rats.

Mr. **Shaw**. Do you see a statutory need in that area?

Mr. **Roisman**. I think in terms of providing for the recoveries that we have been talking about at the objective level, medical expenses, lost earnings, lost property values, yes, I believe that a statute is valuable and essential to make sure that that level of recovery on which we can be certain of the damages is recovered by those people who were in that category.

But I feel that at the second level, when you talk about pain and suffering, and when you talk about punitive damages, that it is more appropriate because we are dealing with a more intangible damage, that we should allow the common law to evolve on its own in the State court system.

Mr. **Shaw**. Thank you, Mr. Chairman.

Mr. **Rowland** [presiding]. Mr. Albosta.

Mr. **Albosta**. Couple of questions.

Have you represented any clients that were successful in their claims against companies for toxic contamination?

Mr. **Roisman**. No.

Mr. **Albosta**. In your opinion then do companies tend to use confusion as part of each one of their cases?

Mr. **Roisman**. Confusion?

Mr. **Albosta**. Confusion.

Mr. **Roisman**. They present the opposite point of view in their cases. None of my cases have gone to conclusion. The corollary of your first question was not that I have had any clients that have lost either. So far in the one case I am pursuing the defendant's position in the case was to deny that they were responsible even for the contamination of water and to try to compel us to prove that that had happened before discovery had been taken in the case.

So what I am experiencing is really I guess for lack of a better term, stonewalling.

Mr. **Albosta**. Let me clear my question up a little bit. Two scientists sitting side by side both with about the same experience could come up with different conclusions to a case.

Mr. **Roisman**. Right.

Mr. **Albosta**. In many cases those—the company's point of view from the scientist is that it is a more neutral position, if you will, toward any toxic substance having anything to do with health effects. Whereas the other scientist may take the view that of caution and says well, it is possible that this could do this, this or this even though the manuals and journals don't contain that information that says that.

Doesn't that create confusion? I suppose from a legal point of view a company would want to do that. How do you win then? How do you—how can a person contaminated ever get a good case other than having an arm fall off, a sore somewhere on his body sticking out or a tumor or something that is identifiable. How can they ever get a case without that?

Mr. **Roisman**. To begin with, it is quite common that plaintiffs recover in cases where defendants put on witnesses that contradict the plaintiff's witnesses. It is a matter for the jury or if you don't have a jury, for the judge to decide which witnesses' testimony is most credible, most believable, which states the facts.

On top of that, it is more difficult to recover where the nature of the injury that the plaintiff has suffered is less visible than what you have just described. But it is also possible to take the jury inside the human body and you can show the damage to a human cell as dramatically as you can show damage to the arm.

If the witness—if the evidence is there for the witness to be able to so testify I think that the plaintiff has a significant possibility of recovering in that case. If it is not there, as I said before, a plaintiff cannot win without the facts. If no scientist of any credibility is prepared to get up and testify, yes, I have seen the damage from this toxic chemical in this human body and it does this and this and this, then the plaintiff doesn't have a case. They cannot prevail. That is an important difference between plaintiffs and defendants.

Defendants can prevail on legal issues or on the failure of the plaintiff to be able to present their case. But the plaintiff can't prevail if they can't present their case.

Mr. **Albosta**. A defendant would have to show a liver that was damaged then, you say to go inside the human body, the scientist would then show the jury or the court a liver?

Mr. **Roisman**. Not necessarily.

Mr. **Albosta**. A kidney?

Mr. **Roisman**. He might have a blood sample, tissue sample. I believe Dr. Carnow—I don't know whether he testified about this but he has done work in the immune system where you are able to take blood samples from humans and examine the immune system and see whether we have a damaged immune system, one that does not respond to the variety of attacks that come from outside the body as well as it should.

And make conclusions regarding what caused that system to be damaged and what are the consequences of that damage, what might happen to the patient even if nothing very serious has yet happened.

Mr. **Albosta**. From your point of view, legally, isn't it very, very difficult to take a case of toxic poisoning that one person may have and another one which would simply not show any poisoning from the same exposure and take that to court and win? The question better put would be, isn't there a difference in people where one will withstand the exposure to toxics better and another will not?

Mr. **Roisman**. True.

Mr. **Albosta**. Isn't that very difficult in court to show that there is a cause from that exposure when you have a person that is perfectly healthy and another one that says my cancer was caused by the exposure, same exposure, yet nothing happens to the other guy?

Mr. **Roisman**. We have medical people who say that that is not difficult. If you start with the premise that each human is different then it follows one will be affected differently. One basic principle of the law is you take the plaintiff as you find him. If the person has as a result of a prior accident some propensity to damage and then they get damaged by something that would not cause any of the rest of us to be damaged, the person who damages them is responsible for that even though that person happened to have this much higher propensity for being damaged. That would be equally true here.

Now, obviously the person who has not been harmed at all will not recover. He shouldn't. But the one plaintiff is not substantially damaged by the presence of some one who has not been damaged if there is medical testimony to link the plaintiff to the toxic exposure.

Mr. **Albosta**. What is your opinion about what Congress can do to improve on the laws? I don't think I should say to make it easier. What I would like to say is to make it so that people that are damaged by some toxic exposures can recover somehow. What exactly in your opinion should be done, seeing you worked in the Justice Department, and in the environmental area.

Mr. **Roisman**. There are several things. In terms of existing legislation I think the Congress should continue to put pressure on the Government to use its existing authorities to investigate the hazardous waste problems of the country, to settle or litigate as to those, to complete the work that the Superfund law imposed upon the Government which includes setting up a health registry for the Nation that would give us a lot of the answers.

The Government had to be sued about that. They eventually settled that suit but are still not committing the resources to it that they should be committing and it is all Superfund resources, so it is off-budget money, it is not budget money so to speak.

The Government should be doing the onsite investigations that would give us a better idea as to where the hazardous waste dangers are and where the victims are; if there are any, they are most likely to be physically located there.

I think victim compensation legislation should also exist which would provide a recovery for the objective kinds of damages that we have talked about here today, the medical, the out-of-pocket medical costs, the lost wages, property damages.

And that that system should include in it a mechanism by which a party who comes forward and establishes a minimum level of evidence such as I consumed the toxic substance, there is a site near my home that has that toxic substance in it, it is in the ground water, it is in my well, at least that much, ought to then entitle that person to some financial help—if the Government hasn't done its own work on the site to complete the evidentiary basis to be able to then establish are they really a victim and entitled to recover.

Then the Congress should allow the private tort system to continue with respect to other kinds of intangible damages and allow the, if you will, the marketplace to sort out which of those plaintiffs are entitled to go on and pursue punitive damages, pain and suffering, losses and other things that are in the intangible area and not either preempt that law or attempt to superimpose upon it a new set of standards.

If the Congress could compel the executive to carry out the authority that it imposed on the executive when it passed the Superfund law, much of my concern about the costs for victims' compensation legislation in terms of being able to give the victim the resources to prove their case, would be unnecessary. I think you have put into motion what could have been a mechanism to give a lot of the factual answers to these questions and that would then have enabled people to come forward without these enormous financial burdens imposed on them personally to prove their case.

Mr. ALBOSTA. Do we relieve the producer of the toxic substance of responsibility in your opinion when we reach a settlement out of court and then sign papers to alleviate them of any further damages?

Mr. ROISMAN. Yes.

Mr. ALBOSTA. Does that happen?

Mr. ROISMAN. What happens is that there will be a Government suit in which the Government absolves the defendant of any further liability to the Government. Now obviously they cannot absolve them of liability to private parties unless the private parties were party to the suit. When the Government does it what it does is say we the Government will accept the risk of the unknown. In settlements we have reached when I was at Justice, we would not sign such a settlement. We would require the settlement terms impose the risk of the unknown on the defendant.

Mr. ALBOSTA. Are they signing those settlements now?

Mr. ROISMAN. Not to my knowledge. I have not looked at any in the last couple months.

Mr. ALBOSTA. Thank you, Mr. Chairman.

Mr. LEVITAS [presiding]. Thank you, Mr. Albosta.

I have just one last question and I will recognize the rest of the members.

Does it make any difference, if you know, if one develops a health problem from exposure to a specific hazardous chemical as opposed to exposure to a hazardous waste site in which that chemical is contained. Specifically, what calls this to mind, I heard last week that I think it was EPA announced that they now have conclusive evidence or proof, whatever, that dioxin is a very poisonous carcinogen, and whatever. I thought it—I was laboring under the assumption it was for a long time but anyhow they came out with that report.

It is also my understanding that very small quantities of dioxin can create health problems in an individual. Does it make any difference whether that exposure is a result for example of spraying of dioxin-containing materials on asphalt or incinerating PCB's at appropriate temperatures in apartment houses or hospitals or direct exposure or in proximity to a hazardous waste site that contains the dioxin?

Mr. ROISMAN. I am not confident that I can answer the question. I think I understand where the answer lies. One of it has to do with the form in which you were exposed to the dioxin can affect you differently, whether it is on your skin or whatever. I don't believe you get it in through the skin although chloracne is a phenomenon with it. I know if you inhale it can affect you differently.

Also in any of those different media it may be mixed with something else which could cause a synergistic effect which might mean that combinations of dioxin and one other chemical is worse than each of those chemicals separately.

So that there are factors but I don't know the answer specifically to the question of which one of those places makes it worse or which one makes it better. I don't know whether the scientific community has an opinion on that either.

Mr. LEVITAS. Thank you.

Mr. Molinari.

Mr. MOLINARI. On that question, I think I recall information to the contrary. I think you can absorb dioxin through the skin. That is one of the points of entry. I think that is what makes it particularly dangerous.

I would like to get an answer on victims' compensation. You do outline in your testimony a suggestion. I would like to address that briefly.

Your suggestion is that you establish a low threshold standard for the victim to meet. Once that standard is met then the victim is entitled to a fund-compensated scientific and medical investigation.

Mr. ROISMAN. Yes.

Mr. MOLINARI. Earlier in your testimony you indicate that for a good and proper medical and scientific workup lawyers taking toxic tort cases are anticipating a minimum of $100,000 to $200,000 in costs just in gathering the scientific and medical information required to prove a claim.

When I applied that cost to my own State, because of the familiarity I have with the figures in my State—I think New York has 22 Superfund sites, we have over 1,000 what they call "mini-Superfund sites" being investigated at the present time, and we are obviously talking about very large numbers of individuals.

Perhaps thousands, that have some health problems attributable to this exposure. So when I start to multiply the figures out I come up with a phenomenal amount of money nationwide in order just to get past point one.

Am I correct in that assumption or am I missing something?

Mr. ROISMAN. No, no, only in this regard, that if you have—assume that at each of the 22 sites you have 100 potential victims, 2,200 people. The investigation that is good for one victim will be good for all at the site. So that it is easier probably to see those numbers in terms of per site rather than per individual necessarily.

Second, the revolving fund nature of this proposal means that over time the fund gets its money back as the victim is able to recover, they of course would recover their costs and they would be expected to pay those back into the fund so that the fund—I am talking now about those victims who go—using the section 301(e) study language—from the first tier to the second tier. In the second tier the person who recovers would have to pay back to the compensation fund their pro rata share of these costs.

So there is the opportunity for some revolving fund. But your premise that it is an expensive effort to make the evidence available and draw the distinction between the true victim and the non-true victim is correct, it is an expensive business.

Mr. MOLINARI. There is no question about that.

Mr. ROISMAN. I don't think it is out of proportion to the need but it is there.

Mr. MOLINARI. There is no question about the fact that it is expensive. I think most of us are prepared to move towards providing some type of relief. But there is one thing that I think either you are missing or I am missing. When you talk about the revolving fund, my experience with most sites—I don't know what the percentage would be—is that most of them are sites that may have been operated by shell corporations, people who don't have any money. They hit and run, and the chances of recovery would be rather slim if you are going after the responsible parties.

So I am puzzled as to how that revolving fund will replenish itself.

Mr. ROISMAN. This same issue arises under Superfund itself.

Mr. MOLINARI. Yes indeed.

Mr. ROISMAN. It is my sense that at the time the fund was passed that that law was designed to use the enforcement mechanism to deal with the site where the responsible party was located, either in litigation or settlement, and that the bulk of the funds in the fund were to be used for the sites where there was no responsible party available for the true orphan site as opposed to an abandoned site situation.

I don't know—I am not as confident on this—that of the total number of sites there are more orphans than there are abandoned or active ones. But it is possible that the fund cannot, certainly it will not revolve to 100 percent, there will be diminution over time. We have not had enough experience with the Superfund to get a sense of that because the Government has not pursued much, if any, recovery suits yet.

But let me just go back to the other point and be clear on this. I mentioned this to Mr. Albosta, also, and that is that the current law, the current Superfund law has in it in section 104(i), that the public health service is to establish an agency for toxic substances and disease registry.

Then in sections 104(e), 104(b), and 104(a), the EPA through the President is given the responsibility to investigate these sites. We have already made the commitment as a country through the Superfund to gather much of this raw data. My feeling that we need to make sure that the victims' compensation scheme provides for the resources to do the medical and scientific investigations is based in part upon my cynicism about the present administration's willingness to use Superfund for what it was intended to be used for, and also that there will be some instances in which a site is causing a problem but for one reason or another has not gotten priority enough to get the Superfund money for it.

But it may be that a lot of the money we are talking about has already been anticipated—maybe the fund itself, the $1.6 billion Superfund—isn't nearly large enough to accommodate that because there wasn't enough momentum at the time that the bill got passed to put in the larger sums of money.

But those costs are going to have to be incurred and many of them, not all of them but many will have to be incurred by the Government if it is going to move against the site to be sure it is cleaned up so the number of victims doesn't continue to increase.

There is no benefit here to anyone to be able to say, let's keep it open and operating so that in 20 years we will have another group of victims who can bring another multiple dollar lawsuit. The Government should be shutting down the source as quickly as possible.

Mr. **Molinari**. That leads me to one important question and I think based on your own background you can answer it. My feeling is that regardless of all we have done here at the congressional level, and the inordinate sums of money we are talking about, the one thing I do not see is enough discussion on prevention. We hear of examples like Times Beach, where for less than $100,000 we could have legally disposed of the dioxin. Yet for only one buyout, and it is only one portion of the area contaminated in Missouri, the Federal cost is $33 million.

The emphasis seems to be on going after a condition after it is discovered and then trying to cope with the problem under very difficult circumstances. It seems to me that we should have much greater emphasis on detection, prevention and enforcement so that we don't wind up with these tremendously costly and difficult problems. I don't see it happening. I just wonder what your observation is along those lines?

Mr. **Roisman**. I think you are absolutely right. I don't think the will to approach the problem in the way you just described it exists today in the executive.

Mr. **Levitas**. I just want to make this observation in light of your comment, Mr. Roisman, before the last one.

In fairness to Dr. Karrh, even though it was not in his prepared testimony, Dr. Karrh did say he felt and the chemical manufacturers felt that it was necessary and proper and appropriate for EPA to go forward with Superfund to clean up the hazardous waste sites because of the potential for health effects down the line.

So just for the record, I want that to be clear in fairness to Dr. Karrh.

Dr. Rowland.

Mr. **Rowland**. Thank you, Mr. Chairman.

In response to Mr. Albosta's question about recommendations you would make to Congress about what could be done, you said that more study needs to be done by the Government and I certainly agree with that, much more study needs to be done by the Government and also by private companies, I think.

I think it ought to be a joint effort on the part of private companies and Government.

The point just made by Mr. Molinari about prevention is important, I think there has not been enough done in that area either.

Then you also said that victims' compensation laws needed to be enacted and you said that when there is objective damages, I believe, that that would be compensated.

My question is is there enough evidence now of objective damage so that compensation would be justified in some chemicals, and if so what are those chemicals?

Mr. ROISMAN. Your question and my statement to Mr. Albosta are slightly—I think we are talking about two slightly different things.

I want to clarify that before I answer the question.

My point about objective damage is those kinds of damages that people suffer that are directly quantifiable like medical bills and the like. I think your question is to causation, in other words, is the causation connection objective.

I don't feel qualified to give you a list of what those chemicals are and I think to some extent it may be that there is no list of the chemicals. When I talked to the medical doctors about toxic substances they have a general sense of what is a toxic substance but they tend to work from the patient back rather than from the substance forward.

If you have a patient, the patient has something that other people don't have, there is reliable evidence to show that the patient's damages could only have been caused by the exposure to the substance, then the substance is believed by the medical doctor to be the cause of the problem.

You have ruled out the other possibilities.

The doctor can then treat the patient by removing that source and comparable sources and actually seeing if the patient got better.

For some people it might be trichloroethylene, for others dioxin, for others it might be a chemical we have never thought could do that before.

I think it is very difficult. It is probably the case that for purposes of legislation you are going to have to put some parameters on. My guess is that you start with something like the list of hazardous substances now in the Superfund law. Put that kind of a parameter on it for lack of anything more concrete than that, I believe.

We don't have all the facts necessary to give the definitive list of health damaging chemicals in the country and then write a law to fit that.

My view is we shouldn't wait until we know them before we go ahead and provide a mechanism, a process by which if a particular person can prove that they have been damaged as a result of the exposure that they should be entitled to recovery.

Mr. ROWLAND. Reasonable doubt still exists then insofar as causation?

Mr. ROISMAN. Reasonable doubt exists in terms of the abstract question, which particular chemicals cause which illnesses? Among the scientific community of which Dr. Carnow is a member, I think individual analyses of individuals does not have that reasonable doubt in it.

Mr. **Rowland**. We talked about some variation in individuals that may be manifest in one whereas it is not in another. So there may be reasonable doubt.

Mr. **Roisman**. Well, it may be that as Mr. Albosta said one individual is affected differently. It doesn't mean as to the one affected that there are not competent medical experts who can—who are unable to say with confidence yes, this caused that.

So as to that individual I think again your question is can we indict a particular chemical. Can we say this chemical across-the-board will always hurt everybody if they are exposed to it at this level? I don't think we know that. We pass regulations based upon risk avoidance. But now we are dealing with a different question. It is not risk avoidance, it is causation and it is appropriate that it should be a different question.

Now we have a particular person, they come up and say, I have been exposed to this chemical and this is the level to which I have been exposed and look what happened to me. The doctor gets up and says I have examined you and it is the chemical that did it.

But should everyone be held to that level as a standard? The answer is no to the question of whether everybody should be subject to that level in order to recover.

Mr. **Albosta**. If I may, what happens if the defendent gets before the jury and the doctor says, "Yes, I think that the cancer or the pneumonia that this person has was caused by the toxic substance?"

But, on the other hand, the other person, the plaintiff says through their legal advice that, yes, but that is happening all the time, in fact, in the 1800's, before we had toxic substances, people were getting pneumonia.

How can you possibly boil this thing down to where you can say definitely that there is anything there in the exposure to the substance? Already we have two people, both exposed, one with the problem and one without a problem.

From your point of view, as a legal expert, how would you defend your person under those circumstances?

Mr. **Roisman**. Well, obviously, I would have my doctor testify much more than just what you have summarized and explain the basis for the doctor's conclusion that the exposure caused the particular illness.

I would cross-examine the defendent's expert to see whether the defendant's expert could really rebut those assumptions. I would not frame it as Dr. Rowland and I were discussing, in terms of whether everybody would have this effect if they got exposed.

I would focus it on that particular victim, did this victim get this from this particular exposure? If you are asking me, do I know that that always wins, no, no, it won't always win.

The legal system allows the person to lose when they should have won and to win when they should have lost, and we sort of have that built into the legal system, the process can still be "due" and therefore constitutionally permissible without always giving us truth.

The legal system gives us validity. I think the system is the best mechanism we have got for resolving the dispute. The other options, trial by combat, torture and so forth, I am not a proponent of.

Mr. **Albosta**. I yield to you.

Mr. **Rowland**. Would you say it would depend on how sharp one attorney is as opposed to another?

Mr. **Roisman**. I think it probably depends more on how good the expert is than the attorney.

Mr. **Rowland**. Or how well the attorney can bring out how good the expert is?

Mr. **Roisman**. You mean could the expert do it without the attorney? As a lawyer, I will say no, I think the attorney is at least a midwife to the case.

Mr. **Rowland**. I never looked at it that way, but thank you.

Mr. **Albosta**. Thank you.

Mr. **Levitas**. That last series of questions between Dr. Rowland and Mr. Roisman goes to the reaches of jurisprudence more so than this particular matter, although there could be relevance to it.

As a former teacher in law school, I remember starting off my classes by saying to some of my students, "Don't use the word justice in this class. You can talk about the law, but we will leave justice for the philosophers and theologians. We have to deal with something else here, and the cumulative effect of the system results in justice if it actually occurs."

That is the problem.

As Mr. Roisman said, there are people who have won cases who shouldn't have, and there are people who lost cases that shouldn't have. All we can do is fashion a system with its rules and procedures and rules of evidence that overall achieves justice in a broader sense.

Thank you very much, Mr. Roisman, for your extremely helpful testimony. We appreciate your contribution and particularly coming from your viewpoint as a former member of the Justice Department dealing with these matters.

You have made a significant contribution to our proceedings. Thank you.

Mr. **Roisman**. Thank you.

Mr. **Levitas**. The next panel of witnesses is a group I have been looking forward to hearing from for quite some time now, because we move closer to the nub of the problem on the subject of toxic torts or the tort system and compensation which is a part of this phase of our inquiry.

So I would ask the panel of Mr. Lundquist, Mr. Light, and Mr. Baron, if they would come forward and raise your right hands.

[Witnesses sworn.]

Mr. **Levitas**. In passing the Superfund law in 1980, section 301(e) provided in subsection 1 the following, "in order to determine the adequacy of existing common law and statutory remedies in providing legal redress for harm to man and the environment caused by release of hazardous substances into the environment, there shall be submitted to the Congress a study within 12 months of enactment of this Act"—and it goes on to describe the study and what it is attempting to achieve.

That study has been prepared and there is still remaining controversy about it. It is for that reason, as we approach the whole question of compensation through the legal system and the toxic tort area, the victims compensation, that the testimony of these three witnesses is extremely pertinent.

Mr. Albosta, I know for a fact some of the questions you were just raising, and Dr. Rowland was just raising, will be addressed by these witnesses in the course of their testimony.

So, we welcome you gentlemen. We appreciate your being here and we look forward to what you have to say.

Mr. Lundquist, why don't we begin with you?

TESTIMONY OF WEYMAN I. LUNDQUIST, ESQ., HELLER, EHRMAN, WHITE & McAULIFFE, SAN FRANCISCO, CA; ALFRED R. LIGHT, ESQ., HUNTON & WILLIAMS, RICHMOND, VA; FREDERICK M. BARON, ESQ., FREDERICK M. BARON & ASSOCIATES, DALLAS, TX

Mr. **Lundquist.** I am from San Francisco, I am Weyman Lundquist, I am a trial lawyer by profession.

I say trial lawyer in that I mean I spend a good bit of my time in the court. I represent individuals in contingent fee cases, I represent corporations in the defense of their interests, I have had some experience many years ago in representing the Government as an Assistant U.S. Attorney.

I think it may be helpful to this group to give you a little background both as to how I came to be on the Superfund Committee, and the Superfund Committee makeup itself.

I came on as a member of the American Bar Association. There were four groups to be involved, American Trial Lawyers, American Bar Association, the American Law Institute, and the Association of Attorney Generals.

The reasoning underlying the makeup of that committee was to give both balance to the views and also a diversity of experience.

Within the ABA, I believe my reason for being here is that I have had a long interest in environmental matters, I have been involved with them, I have had some direct involvement in the courtroom, but much of it in study and in related activities of a public nature.

I also have, over the years, worked a good bit with study of the tort system. I serve on the ABA committee with Griffin Bell, which is studying the tort system. I have had much to do with procedural matters within the courts of the United States, particularly Federal, in terms of civil procedure revisions that have been undertaken there.

Now, more directly to the Superfund and what we have come up with, to all person's views is derived from their own experience. I think one of the reasons for the need for Superfund Inquiry Fund Recommendations and why this group came up with them is that we are basically facing a new type of problem with toxic injuries. Let me explain.

We know today more and more why people die, as people continue to live longer and longer. We get over a point at World War II and where most people were dying for biological or natural causes, where the human longevity was in the area of 50 to 60, years Following World War II with increased health care, sanitation and otherwise, people lived longer and longer.

Generally, as you become injured at an older age or sick at an older age, there is less compensation available and, indeed, lawyers are less interested in representing you simply because your case is not worth as much money.

A further paradox to this is that the older people very often are older because of the very things that will bring about their demise. Specifically, my father is 78 years old. Since he emigrated from Sweden, he has spent his employment life in a factory in the United States making grinding wheels.

Now, my father's longevity has already far exceeded what he could have expected at the time of his birth. I would say that it is very probable if an autopsy is performed at his death that you will find that the cause of his death will probably have something to do with his working in an abrasive factory. At the same time, that very work has enabled him to live a long and full life.

That is a paradox.

The other aspect of this is that there is no question that some people are severely affected by toxics and die early of diseases such as cancer. Indeed I can remember in that same factory where my dad worked, some people only had one lung because that was happening.

You can't ignore these people. So we are faced with a need for balance. Our system is in essence faced with trying to balance these two problems.

We have had further problems, some on the medical side already alluded to. Some on the business side which is, of course, interested in profitability, with generally short-term motivation.

There are some on the legal side. Lawyers have vested interests which they seek to defend, both defense lawyers and plaintiff's lawyers. So all of these things were to some extent tensions and yet, accommodations which have to be worked out in the Superfund, personal injury recommendations.

As I now view it, I think it is a relatively satisfactory proposal.

What the recommendation does in essence is provide for people who are generally not now, in my judgment, well provided for. These are the cases that would come into it. These are the cases which lawyers advise be brought into it—who don't have distinct, obvious, easy-to-prove injuries. Nonetheless, they have medical bills, they have concerns, they have some disability.

In my judgment, most of them, probably will be older because when you get to mass groups, these are latent problems and age enters into it. If you get the younger person already alluded to, generally that person doesn't have difficulty finding a lawyer who will undertake to present that case on a contingent-fee basis and if it is at all good probably will prevail with it.

It is worth the effort.

But for the larger, older group, it is not. So, our Superfund group proposed that in more limited damages and with easier proof, you could come into the Superfund compensation system.

One of the things——

Mr. LEVITAS. Let me interrupt you there, when you say Superfund, are you talking about the Superfund Committee?

Mr. Lundquist. I am talking about our recommended proposal, yes. I should have been more precise.

Mr. Levitas. I just didn't want the record to be confused when it is read subsequently. The present Superfund law does not provide for what you are talking about, but the proposals of the 301(e) committee do.

Mr. Lundquist. Yes, I appreciate that. The context of my remarks is all in that direction. I am not speaking about Superfund other than in the context of the recommendations of our committee.

Our proposal, Tier 1, as we came to call it, was basically an administrative system administered by the States which might give people not the full expanse of compensation they might seek in some courts—certainly in the courts of California—but more limited, specific, and one would hope expeditious receipt of benefits.

It also, I think, did something else which was important, it created presumptions in connection, that is it made it easier for the person who could show that they were engaged in an occupation or suffered exposure from a hazardous substance, to establish they had an injury or disease which was known to derive from that exposure which have created a presumption of injury entitling them to compensation.

The process was further eased by providing that you could develop toxic substance documents. These would be scientifically established, governmental based documents, which would make the nature of the proof easier. One wouldn't have to go out and show that an individual suffered causation in this way. It was a way of minimizing scientific problems and giving a more generalized overview approach.

The interesting thing about the presumptions is that on the defense side, they are essentially not liked in some respects, because it would make it easier to prove a case. To my surprise, they were also resisted by people at the plaintiff's bar of the regular tort system because they said their view is that these make it a minimum standard, and a court would say: If you don't have that presumptive case, you won't have a case in our court.

I, myself, when I am a plaintiff, would rather have the benefit of presumptions, but the plaintiff's bar took the view that that was not the case.

This shows that presumption can be helpful in the level and type of case we are talking about with Tier 1.

Tier 2 doesn't use presumptions. It uses the traditional court system. I think it should be said that our court system has generally functioned well. People don't always like it, but it has been useful in causing evolving law, indeed, the law that we speak of today is entirely different from the law we knew a decade and certainly two or three decades ago.

We need to keep that evolving part of it, too. I am a firm believer in a system which utilizes juries, because I think juries make social judgments. They must understand and apply the law. In a democracy, laws are an essential part of it.

So, I think it is necessary that that part of the system remain there.

Finally, two points, because I don't want to run on unduly.

Mr. **Levitas**. Let me say this, your entire testimony, Mr. Lundquist, and that of your colleagues, will be made a part of the record, without objection. But you may proceed.

Mr. **Lundquist**. I understand that. I didn't know it was going to be part of the record until Friday morning, so I apologize for its brevity because I think there is more to be said.

That was one of my two final points.

The last point that I want to make, and it is the one to me that is the most important, I think victims should be compensated, I think it has to be done, and it has to be practical and effective.

One of the problems about the toxic type of claim is that it is clogging the system. It is taking far too long to get through the system. This claim is aberrational because you get the people who are responsible fighting among themselves, be they insurance companies saying: "We didn't have the coverage," or "It isn't there," or companies saying, "It is not our problem." This makes it aberrational in the sense that most claims that we would face in life are settled not in court, not even at the lawsuit level of court, 98 percent of claims are settled at the claims level when somebody goes to a company or an insurance company and it stays out of the judicial system.

We get, once into the judicial system, 98 percent of the cases settled after the 1 to 2 percent which enter the system. We have aberrational problems here where the nature of these claims, involving latent defects, means that you can't find responsible people. These cause delay and expense and slow the process.

For that reason, I think we do need a different sort of solution. I have more information, as we see Minnesota has just passed a law recognizing this. Before long we will have better information as to how it works.

But the other thing we must consider in all of this is that compensation essentially reflects a short-term approach. Each and every one of us is a steward of our children's futures and as I noted in my conclusion to the study group report in a few paragraphs which I will now read, which express my own views:

"The significant lessons learned from our work is that further emphasis, economic, legislative, legal, should focus on avoiding toxic injury problems. Today and tomorrow's efforts and legislation should concentrate on having generators of hazardous waste develop ways to make them benign. Emphasis should be on the treatment of waste materials to make them safe.

While I favor compensation, I urge that the legislative efforts be pursued and incentives be given to those who will solve the problems which we might otherwise perceive 20 years down the road.

Thank you.

[The statement of Mr. Lundquist follows:]

TESTIMONY OF WEYMAN I. LUNDQUIST BEFORE THE SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT OF THE COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION

JULY 26, 1983

I am pleased to be here. My name is Weyman Lundquist. I live in San Francisco, California. I want to emphasize at the outset that my remarks express personal views. I do not speak for any client or organization with which I am associated.

Mine is a longstanding interest in environmental concerns. I have handled environmental litigation and have been directly involved in environmental activities in Massachussets, Alaska, and California. More significantly, I have been associated with a number of organizations generally recognized as having environmental concerns, including California Tomorrow, The Natural Resources Defense Council, The League To Save Lake Tahoe, and the Sierra Club. Again I speak for none of these organizations today.

With those general remarks let me turn to the matters that I have been asked to address here. I have been asked to comment on the following subjects: the reason for the establishment of the 301(e) Study Group under the Comprehensive Environmental Response, Compensation and Liability Act of 1980; the nature of the Study Group's membership; the Study Group's recommendations; and the compromises that were necessary to achieve these recommendations. In addition, I will present my own views about the need for a special legislative emphasis on the prevention of ongoing toxic problems.

REASONS FOR ESTABLISHING THE 301(e) STUDY GROUP

The Comprehensive Environmental Response, Compensation and Liability Act Of 1980 (CERCLA)[1] created what is now known as Superfund. Superfund is a trust fund that is designed to finance the cleanup of hazardous waste sites and toxic spills. CERCLA, however, does not provide for the compensation of personal injury or property damages that result from the exposure to hazardous wastes. Instead, in Section 301(e) of CERCLA, Congress authorized the creation of a Study Group to evaluate the problem of compensating personal injury and property losses associated with hazardous wastes.

The Study Group's statutory mandate literally authorizes it "to determine the adequacy of existing common law and statutory remedies in providing legal redress for harm to man and environment caused by the release of hazardous substances into the environment. . . ."[2] The Study Group was authorized to examine the nature, adequacy, and availability of existing remedies to compensate for these harms and the barriers to recovery for such harms within the current legal system. The Study Group was also asked to submit appropriate recommendations to Congress regarding the need for revisions in statutory and common law so that such personal injury and property damage would be adequately compensated for.

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[1] Comprehensive Environmental Response, Compensation And Liability Act of 1980, Pub. L. 96-510, Section 301(e), 94 Stat. 2767 (1980).

[2] *Id*. § 301(e), 94 Stat. 2807.

I should point out that the Study Group's explicit statutory authorization asks it to study injury and damage resulting from "the release of hazardous substances into the environment."[3] As noted in Frederick R. Anderson's statement following the Study Group's Report, "our mandate directed us to examine recovery for injury from the broad category of hazardous substances, not merely from hazardous wastes."[4] In fact, the first compromise the Study Group made was to narrow the focus of the study by concentrating only on the problem of hazardous wastes. As Mr. Anderson continues in his statement, however, "the proposals discussed herein, and our conclusions about the inadequacy of the common law, both can be generalized to apply to the entire spectrum of common law recoveries for injury from hazardous substances. . . . [T]he framework for analysis and the basic criticisms remain the same."[5] I concur.

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[3] Id.

[4] Special Report to Congress, Injuries and Damages From Hazardous Wastes -- Analysis and Improvement of Legal Remedies, In Compliance with Section 301(e) of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 by the "Superfund Section 301(e) Study Group" 289, 293 (reprinted as Comm. Print for the Senate Comm. on Envtl. & Pub. Works, Serial No. 97-12, 97th Cong., 2d Sess., 1982) (Statement of Frederick R. Anderson) [hereinafter cited as Report].

[5] Id.

THE DIVERSITY OF THE 301(e) STUDY GROUP MEMBERSHIP

For any group to speak with authority concerning legislation, it should be a group representing a spectrum of views and having no particular bias. Ideally, this constituency should include the views of academicians, the government, lawyers who represent individuals, lawyers who represent commercial interests, and appropriate associations. The Study Group was made up of such a membership. Study Group members were appointed by four separate organizations. They were the American Law Institute, the American Bar Association, the American Trial Lawyers Association, and the National Association Of Attorneys General.

It is clear from the composition of the Study Group that Congress did not intend and did not create a group that would reach an easy consensus. The variety of interests that were reflected in the Study Group, however, did not lead to an inconclusive report. The final report reflects substantial agreement by the Study Group members. Although some of the Study Group members submitted separate comments to the report, these comments generally did not dissent from the basic two-tiered remedial proposal. Even the dissenting comments, those of Judge Breitel and those of George Freeman, supported the establishment of an administrative compensation remedy while allowing claimants an opportunity to pursue common law remedies.

It should be noted that some of the committee members - both plaintiffs' lawyers and defense lawyers -- have a vested self-interest in their espoused views. As the discussions of the Group progressed, it became evident that certain other Group members, particularly academicians, sometimes also had a vested interest in views they had expressed over a number of years. Not surprisingly, each constituent group of this committee was conservative in favoring what it knew best. This was as true of the plaintiffs' lawyers as it was of the defense oriented counsel.

THE GROUP'S DELIBERATIONS

The Study Group had no doubt that toxics are a problem. They are a problem because there is some individual harm. They are a problem because of public concern about potential harm. They are a problem because of the latent harm that they may yet create. As they continue to be developed, toxics present an increasing problem in American society. Among the more difficult dilemmas the Group faced, however, was evaluating how immediate the problems are and what solutions might be most expeditious and economic. The Group was also confronted with questions involving bars to recovery and the transactional costs of resolving these problems.

A major conundrum underlay the Group's deliberations: what is the size of the problem? We know without question that large numbers of people have been exposed to toxic risks. The extent of physical detriment suffered by these people is less well known and less well established. Developing medical science increasingly enables us to learn why people become diseased and die. A few short decades ago such determinations would not have been possible. Some who develop diseases may be significantly disabled by them, and in the main more people continue to live longer. Thus, the actual harms caused by toxics are difficult to measure.

Because of the multifactorial etiology of most of the injuries that are related to exposure to toxics, most of the scientific literature is very inconclusive as to injury causation. Disease incidence in populations that have been exposed to a given toxic substance is usually reported either simply as incidence, without any conclusion as to causation, or at best as a correlation. Some of the paucity of the causation conclusions is related to the high burden of proving statistical significance in scientific stuidies. Some is related to the dose-response curve; researchers who are willing to make causal conclusions regarding high exposure levels may be unwilling to make the same conclusions regarding lower exposure levels -- exposure levels that may still be toxic, but for which the incidence of injury is so low that the data necessary to prove causation are not available. Furthermore, for a great number of scientific conclusions regarding toxic substances, whether the conclusions have been in the form of correlations or causal connections, further studies have either failed to replicate the original results or have reached contradictory results. In addition, the synergistic effect of toxics with other harmful substances -- particularly smoking -- makes it difficult to give definitive answers.

Even for those substances where a causal relationship between the toxic substance and an injury is accepted, there is almost a complete absence of data that indicates the age groups affected, which in turn would indicate how many lives are actually being shortened or severely impaired by the injuries that result from exposure to these toxic substances. Both the Love Canal data and the asbestos data suggest that most injuries manifest themselves in older populations. However, substances such as DES and DBCP have been proven conclusively to have severe effects early enough in the lives of the victims so as to seriously disrupt their lives, lifestyles and life expectancies.

Since the Supreme Court's Benzene[6] decision, administrative agencies have not been able to regulate exposure to toxic substances unless there is sufficient data to prove that there is a "siginificant" risk of harm from these substances. Thus various substances that may be toxic cannot be regulated at the present time because of insufficient scientific evidence of harm, or because of insufficient scientific data regarding the toxicity of these substances at low exposure levels. Because of the long latency period between exposure to toxics and the resultant injuries, it is thus possible, and in fact likely, that mass toxic tort injuries caused by these substances will manifest themselves in the future. As Judge Breitel noted in his separate comment to the Study Group report, "[t]here are toxic substances and wastes which were, on any test, deemed not harmful and even beneficial at one time. And there are substances today, without any determinable responsibility, which appear to be utterly harmless or even beneficial or essential, which later, or tomorrow, will have been discovered to be toxic singly or in synergistic relation with other substances."[7] Indeed, the lack of scientific conclusions as to causation does not indicate that various substances are not toxic or will not cause toxic harms. Rather, the absence of data merely indicates that researchers have not yet been able to prove that particular substances are not safe. The Study Group was thus confronted with the situation where the extent of these harms has not yet been quantified.

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[6] Industrial Union Dep't, AFL-CIO v. American Petroleum Inst., 448 U.S. 607, 653 (1980) (plurality opinion).

Furthermore, a few short decades ago, people's life spans were substantially shorter. Indeed, one of the ironies of what we face is that the lives of many people will ultimately be adversely affected by toxics and yet their life expectancies have been increased by the very societal developments that were made possible by these substances. Asbestos and pesticides are good examples of this. Thus, we are seeing people who ultimately die or are disabled by exposure to the very substances that have permitted the lifestyle that has extended their lives.

With this background of factual uncertainty, the Study Group focused on some of the problems that confront an injured plaintiff who attempts to seek compensation for a toxic injury. The most significant of these problems are the statute of limitations, joinder of parties, and proof of causation. Because of the long latency period before the manifestation of injury that results from exposure to toxic substances the statute of limitations presents almost a complete bar to recovery in those states that have not accepted a discovery rule. Plaintiffs may lose their right to bring a lawsuit before they have learned of their injuries. Ownership of a hazardous waste site, or of a corporation that produces a toxic substance may have changed during the long latancy period. The questions of liability and apportionment of liability thus become complex. Because of the complexities inherent in toxic tort suits, the costs of pursuing an action become a substantial barrier to a plaintiff's recovery unless joinder of plaintiffs or class action is available. Because of the multifactorial etiology of the injuries most commonly associated with toxic substances -- cancer and lung disease -- proof of causation is extremely difficult. Medical and scientific testimony, used to establish causation, is prohibitively expensive, even in cases in which the etiology of a disease may be rather clear. In the face of such problems, many of the committee members were of the view that the costs of disposing of toxic harm claims were excessive and, indeed, that the victims of toxic harms are frequently compensated less than those who act on their behalf -- their lawyers.

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[7] 1 Report, supra note 4, at 297 (Comments by Charles D. Breitel).

Further, there is currently no federal statute that expressly provides a remedy for personal injury due to hazardous waste exposure in non-occupational settings and state statutes creating such remedies are rare. Plaintiffs in most situations must rely on common law tort causes of action.

THE COMMITTEE'S RECOMMENDATIONS

Because of the substantial barriers a private plaintiff faces in an action for damages caused by hazardous waste exposure, particularly where the individual claim is small, the Study Group recommended a two-tiered administrative-tort proposal. Zazzali & Grad's review of the Study Group's Report well summarizes the committee's recommendations. I will quote directly from their summary.

> The first step of the proposal, Tier One provides no-fault compensation for personal injury resulting from hazardous waste, to be managed by the several states under a federal program. This compensation plan, operating in a similar manner to workers' compensation, would provide full recovery for a person's medical expenses and, with certain limits, recovery for two-thirds of a person's loss of earnings. Death benefits for close surviving dependents would also be provided. In addition, injured persons would have the choice of proceeding with a personal injury claim under existing tort law, but the amount of any compensation award recovered by the claimant in Tier One must be deducted from, and be paid back out of, any judgment or settlement in a Tier Two plenary action. A person electing to seek compensation under both tiers must repay the Tier One compensation award out of any payment obtained in the personal injury action. A person who recovers under both tiers will also be required to pay certain costs if the Court found that the settlement or award in Tier Two did not substantially exceed the

> compensation award at Tier One. This provision is intended to avoid frivolous claims.8

The administrative compensation plan designed in Tier One would be established by federal legislation, would be operated by the states, and would provide compensation without a showing of fault. It would cover injuries resulting from exposure to hazardous wastes, but excluding occupational exposure covered under the workers compensation system; it would thus be co-extensive with the coverage of CERCLA. To ease the problems with proof of causation, a rebuttable presumption was designed, such that if a plaintiff was exposed to a hazardous waste and suffered an injury that is known to result from exposure to this hazardous waste, the exposure would be presumed to have caused the injury. This presumption would be effective only in the Tier One setting. The compensation awards would be financed by the creation of a fund from industry contributions, analagous to CERCLA's Superfund. This fund would be subsidized by federal contributions or taxes and to some extent would be replenished by the collection of subrogation claims.

The second tier of the two-tier remedial proposal suggests alterations to common law and statutory remedies for hazardous waste exposure. The Study Group recommended the unified adoption by all states of a discovery rule, such that a plaintiff's cause of action would not accrue until the plaintiff discovered or reasonably should have discovered the injury and its cause. The

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8 Zazzali & Grad, Hazardous Wastes: New Rights and Remedies?, 13 Seton Hall L. Rev. 448, 464 (1983) (footnotes omitted).

Study Group further recommended the adoption of liberal joinder rules, a shifting of the burden of damage apportionment to defendants who contributed to the injury, and the application by the states of a theory of strict liability in toxic exposure cases.

An "exit toll" was designed to minimize frivolous suits and the potentially increased costs that could result from a scheme that allows for dual recovery. Tier One compensation would be deducted from, or paid back out of, any settlement in a Tier Two action. If a Tier Two settlement does not yield compensation that exceeds the Tier One settlement by more than 25%, the judge would have the discretion to assess costs against the plaintiff.

COMPROMISES INCORPORATED IN THE RECOMMENDATIONS

The diversity of the 301(e) Study Group membership necessitated that various interests would have to be reconciled to achieve a compromise recommendation. The Plaintiff's Bar, with its contingent fee interests and its concern for preserving the right to jury trials, was reluctant to endorse any recommendation that would have included a binding election between administrative and tort remedies. The academicians in the Group were more inclined to take a systemic view of the problem, and to be concerned about the forum-clogging, burden on judicial resources, and excessive disposition costs that extended judicial procedures might create. They were thus concerned about the duplication allowed by a scheme that does not require an election of remedies. The Attorneys

General, having a more practical orientation, were more willing to compromise so as to reach a consensus recommendation. In my view, because in current dollar terms the average damage claim for hazardous waste injury is likely to be in the $10,000 to $15,000 range, I am inclined to believe that our Tier One-Tier Two Recommendation is practical; most claims will be handled by the administrative remedy and will not necessitate a tort action in court.

As noted in their separate statements that follow the Study Group Report, a number of the study group members felt that an alternative to the recommended relationship between Tier One and Tier Two remedies would be more advantageous. Under this scheme, an election to obtain the administrative award would be binding, and a plaintiff would need discretionary judicial approval to bring a Tier Two (tort) action. A plaintiff would only be allowed to pursue a tort remedy in the event of an unanticipated cause of action for damages available under tort law beyond that recoverable at the administrative level. Those Study Group members who prefer this binding election between remedies, rather than the exit toll created by the Study Group's recommendations, do so because they find the availability of combined Tier One and Tier Two remedies to be unnecessarily duplicative, inefficacious, and inequitable. Judge Breitel in particular has expressed the view that "[t]he 'exit toll' is not severe enough to avoid 'gambling', that is, obtaining easy recoveries at the administrative level, and then pursuing the plenary action."[9] In my view, if the majority of claims are not large, then they are not likely to be pursued in court -- "the game is not worth the candle." It may be that some restrictions will have to be placed on lawyers amassing numbers of claims to prevent undue economic motivation for lawyers, if not their clients.

Another area of compromise concerned the definitions of causation, presumptions, and discovery of injury. Mr. Freeman in particular, as I am sure he will testify to at length before this Subcommittee, was critical of the statutory presumptions proposed by the Study Group. Both Mr. Freeman and Judge Breitel objected to the use of legislative fiats that reflect policy biases as a substitute for the truth in an area in which the truth is elusive.[10]

Judge Breitel and Mr. Freeman were both particularly concerned that an insufficient cost-benefit analysis had been performed.[11] Both question whether subrogation of claims and taxing of various selected industries are the most appropriate ways to fund such an administrative compensation scheme.[12]
Mr. O'Connell was concerned both that the availability of Tier One,

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[9] 1 Report, supra note 4, at 299 (Comments by Charles D. Breitel).

[10] Id. at 299-300 (Comments by Charles D. Breitel), 314-19 (Comments of George Clemon Freeman, Jr.).

[11] Id. at 301 (Comments by Charles D. Breitel), 314 (Comments of George Clemon Freeman, Jr.).

[12] Id. at 301-02 (Comments of Charles D. Breitel), 311-14 (Comments of George Clemon Freeman, Jr.).

subrogation, and Tier Two proceedings would place an undue burden on the judicial system,[13] and that the availability of compensation from a Superfund-type trust fund, in addition to payments available under private health insurance, sick leave and disability insurance, would result in a windfall to claimants.[14] A number of the Study Group members were also disconcerted that the Study Group's recommendations did not include awards for psychological, social, and related injuries in the Tier One administrative remedy.

As I stated in my final comments to the Study Group Report,[15] I endorse the Report. I am also comfortable with the Anderson, Breitel, Freeman, O'Connell position (binding election between Tier One and Tier Two with judicial discretion to allow tort actions after an administrative remedy has been elected),[16] and could join this position should Congress deem it advisable.

EFFICIENT USE OF LEGISLATIVE RESOURCES

Having giving the preceeding descriptive, I would like to make some personal observations. As I noted above, we lack good basic information as to the nature and extent of the toxic injury problem. There is no doubt that those who are disabled should be compensated in the most expeditious, efficient manner possible.

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[13] Id. at 354 (Comments by Jeffrey O'Connell).

[14] Id. at 353 (Comments by Jeffrey O'Connell).

[15] Id. at 349 (Final Comments by Weyman I. Lundquist).

[16] Id. at 286 (Joint Statement of Messrs. Anderson, Breitel, Freeman, & O'Connell).

Every effort should be made, however, to ensure that our concerns in this area do not allow us to overwhelm ourselves with bureaucratic institutions. Indeed, a sunset provision should be included in any compensation scheme that is established; the need for such a program should be reexamined at least every decade.

I am particularly concerned that our current focus on compensating individuals for toxic injuries will blur our ordering of priorities. Our emphasis should be on avoiding toxic injury problems, rather than creating compensation mechanisms for them. As I noted in my individual comments to the Study Group Report:

> The significant lessons learned from this [the Group's work] is that further emphasis -- economic, legislative, legal -- should focus on avoiding toxic injury problems. . . . Today and tomorrow's efforts and legislation should concentrate on having generators of hazardous waste develop ways to make them benign. Emphasis should be on the treatment of waste materials to make them safe. . . (51)
>
> It is not farsighted to justify current industrial activity on the grounds that economic pressures at home or from abroad mandate that we permit today what we can anticipate will be a greater problem tomorrow. . . . It would be nice if one could anticipate that free-market economic forces, left unfettered, would lead to the goals we seek. That has not happened. It is unlikely to happen when short-term gain conflicts with long-term problems and costs. It is difficult to ask that we act now to protect future generations. It is however both wiser and kinder to do this than for us to ask those yet unborn to pay for what we enjoy. . . .
>
> . . . The very need for this committee's study arises from the fact that past unconsidered conduct has given rise to substantial present harm which we must now seek to redress with dollars as the best available substitute for health or damage to property.
>
> Unless we live within the capabilities of our resources and knowledge, we are putting at risk the futures of others. One of the sound tenets of our economic system has been that one saves now to invest for the future, for oneself and others. Unless we adopt this

> prudence in our approach to toxic injury and damage, we run the risk of bankrupting the lives and futures of others.17

We must adopt a more prudent orientation toward toxic substances. I endorse the Study Group's compensation recommendations, however, I would not like to see Congress focus on such a compensation scheme to the exclusion of a focus on preventitive measures.

Finally, we mus be certain that we do not attempt to circumvent the hazardous waste problem by exporting our hazardous wastes. Such an attempt would be both inefficacious and unjust. We should under no circumstances permit the export of products -- and of the problems and injuries concommitant with these products -- which we would not be willing to accept within our own domestic boundaries.

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17 Id. at 350-52 (Final Comments by Weyman I. Lundquist).

Mr. Levitas. Thank you very much, Mr. Lundquist, for a very pertinent and succinct testimony.

Dr. Light, perhaps we can hear from you at this time. By way of introduction, would you give us a little of your background and so forth?

Mr. Light. I didn't realize they were going to put Dr. Light on the witness list. I am not a medical doctor like Dr. Lawland, I am a Ph.D. in political science and, as an aside, I am a relatively new lawyer. I have been with Hunton & Williams for about 1½ years, and one of the first projects that I was assigned was to be a staff member to George Freeman, Jr., who was a member of the Superfund study group.

Mr. Freeman wishes to apologize to the committee for his not being able to be here personally. A dear friend of his for many years unexpectedly died this past weekend, Angus Macaulay, and Mr. Freeman was responsible for funeral arrangements.

I am here in his stead.

I ought to start off by saying I am not a trial lawyer, and not a hired gun, as my colleagues here. I am really probably as much an academic as I am a hired gun. My own experience in this area has been on a scholarly level as a teacher and political scientist.

I have prepared a summary of my own views on this matter for the National Science Foundation, but the document distributed here is the testimony which Mr. Freeman would have presented had he been here, and I am here basically to summarize that testimony briefly, and answer questions to clarify any aspects of that testimony, which is quite lengthy.

However, to the extent that we go into matters which are not covered in that testimony, Mr. Freeman has volunteered to provide any responses to the questions you might prepare for him, and I will be happy to convey them to him.

I have an assistant here, Mr. Paul Mirengoff, who will be taking notes and trying to keep me honest on that.

Mr. Levitas. We will accept Mr. Freeman's testimony and make it a part of the record, and we regret that circumstances are such that it prevented his participation today.

We welcome the opportunity to present other questions by way of followup to him, and appreciate your participation.

I must say that Mr. Freeman's name rings a bell in my head somewhere, and I am trying to figure out where I know him from.

Mr. Light. Mr. Freeman is a representative of the utility industry on a number of matters and has appeared before various congressional committees.

He is active as Mr. Lundquist is, in the American Bar Association, and I suspect on the basis of your background, that you have seen him in connection with the regulatory reform legislation.

Mr. Levitas. I think so.

Mr. Light. S. 1080, H.R. 2327, and so forth.

Mr. Levitas. OK.

Mr. Light. Our general perspective within Mr. Freeman's office at Hunton & Williams has been from the point of view of administrative lawyers and to a lesser extent corporate lawyers, as opposed to trial lawyers. His own practice has been primarily before administrative bodies such as EPA, NRC, and so forth.

So, he and I are administrative rather than trial lawyers. On some trial issues, you may well find the advice of my colleagues more pertinent than mine.

In general, Mr. Freeman supported the 301(e) approach. There were several matters on which he had some serious disagreements with the majority and felt compelled to outline in a separate statement in the report.

On the whole, this rather weighty document which I brought a copy of, the Superfund 301(e) report, does in my view adequately summarize the present state of the law in this area, and in some related areas.

There are some gaps which in some cases may have been deliberate and in other cases just oversight. But, if you read the report itself, and the separate statements, I think you gentlemen will get a general feel for most of the issues, some of which you have already discussed in previous hearings and so on. The report is a good rehearsal of the congressional debate over this victim compensation legislation.

There were five areas where Mr. Freeman had such serious enough disagreements that he felt he should outline them in his remarks. They were as follows: First, I think it turned out that Fred Baron and Mr. Freeman, at least at the conclusion, tended to be in the same camp on this matter of statutory presumptions.

Mr. Freeman, however, went along with the toxic substance rulemaking idea which Mr. Lundquist outlined. However, he felt that their preparation was an appropriate function for only some Federal agencies. He would not put it in EPA because of its regulatory responsibilities, but might put it in one of the National Institutes of Health or in some other scientific agency divorced of regulatory responsibility.

Nevertheless, he felt that writing statutary presumption into the statute without an adequate factfinding record on a particular substance might lead to a situation that would just be more confusing than the present situation.

That, coupled with what he heard from the plaintiff's bar in connection with the two-edged nature of the thresholds or criteria which might be written into the statutory presumptions, compelled him to dissent on that point.

On another issue, the funding of the fund, analogous to Superfund, which would pay for injuries to individuals who decided to apply for the administrative relief, he dissented from the study group's recommendation that this be an add-on to the Superfund tax or a similar tax. This recommendation is incorporated as a recommendation into the present Markey-Florio bill, H.R. 2582.

And his reason for that goes to the scope and dimension of the problem question, which you all had discussed today and discussed in some of your previous sessions with CMA and others. Who should be the taxpayers is an issue which he felt that the Commission didn't have enough evidence to give Congress any advice on, so he felt as a matter of principle, that he had to dissent on that.

Third, there is a significant issue outlined in Mr. Lundquist's testimony and referred to in Mr. Freeman's statement, the issue of the relationship between the new administrative remedy, the tier 1 remedy, and the current ongoing litigation of tort claims, the tier 2 remedy, in what are principally State courts at this point.

Mr. Freeman felt, after deliberating over this for the year or so that the Superfund Study Commission held its meetings that there had to be some barrier put up controlling access to tier 2 once a claimant had chosen to pursue his administrative remedy in tier 1. The fear is that a person would seek his administrative remedy and have a recovery there and then use that money to finance his access to tier 2. Or at least the system would be subject to more abuse. His preference was for a binding election in which one chose his easier, quicker recovery in the administrative system, or one chose to pursue a remedy in State courts with its higher uncertainty but with potentially greater rewards.

Another issue that the group did not confront in Mr. Freeman's opinion because the Commission was probably divided on it based on what its minutes revealed—is the issue of punitive damages. In an area of the law where there is a comprehensive scheme of civil and criminal penalties enforceable perhaps through citizen suits as under the Water Act, or at least where the claimant has the ability to go to a law enforcement official at the State and/or Federal level to secure the punishment of the person violating the regulations or standards, he felt that on top of that to allow the plaintiff to go into court and to seek punitive damages was tantamount to allowing a kind of vigilantism, and in his own separate statement, the remark was "vigilantes have no place when the sheriff takes over."

The final area where Mr. Freeman made some comments in his separate statement which were at some variance with the majority was his view of strict liability, both with respect to the view of strict liability under the present law, at least one theory which is set forth in part 3 of the report, and the view of strict liability which the Commission adopted as the one which it would apply to defendants in the so-called tier 1 subrogation action where once the fund has paid out money it goes after an allegedly responsible party. The view of strict liability which was adopted by the Commission, Freeman thought was tantamount to a kind of absolute liability, and was not appropriate.

So those are the basic five areas where he felt compelled to dissent. On many other areas such as in the toxic rulemaking area, he certainly has taken a well advanced position compared with others with whom I have spoken on the defense side, and he spoke for himself. As we went into this Superfund study group, we followed the history of public service within our law firm, Hunton and Williams to leave our clients at the door in such bar activities. He spoke only for himself in his statement and did not represent any client's interest.

We do not represent any clients in this undertaking.

I guess I have just a couple other notes that I would like to make. One is that during the Superfund study group's deliberations Mr. Freeman personally did not feel that he had a lot of understanding of the insurance implications of what the study group was about, and so advising him and subsequently actually advising the study group, we drew upon some experts which we were able to identify in the insurance industry and they devoted some time to reviewing an early draft of the Superfund study group report and out of that—this is reported in the body of the report—came an idea for handling the issue of apportionment of liability, the so-called channeling concept, which Dick Schmalz with the Hartford put forward in an article I have appended to this testimony.

We think that idea certainly deserves some consideration in the context of victim compensation and perhaps also in the context of some amendments to the Superfund act itself regarding cleanup of hazardous waste sites.

The other part of the testimony which—the first part of Freeman's testimony distributed today—tries to relate some of the issues which the Superfund group dealt with in victim compensation with some of the problems we see in our practice and in our studies with respect to the Superfund liability system and we would be happy to talk about that if you would like.

Thank you.

[The statement and attachment of Mr. Light follows:]

STATEMENT OF GEORGE CLEMON FREEMAN, JR.

HUNTON & WILLIAMS
RICHMOND, VIRGINIA
WASHINGTON, D.C.

BEFORE THE

SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT
OF THE
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION
UNITED STATES HOUSE OF REPRESENTATIVES

CONCERNING
HAZARDOUS SUBSTANCE INJURY COMPENSATION

JULY 26, 1983

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I. INTRODUCTION

Mr. Chairman, Committee Members, Ladies and Gentlemen -- my name is George C. Freeman, Jr. I am a partner in the law firm of Hunton & Williams, with my offices both in Richmond, Virginia, and Washington, D.C. I am also a member of the Virginia, D.C. and Alabama bars.

It might be helpful to indicate briefly where I am coming from on the issues before you today in terms of past and present practice and bar related activities.

My active practice is primarily in administrative law in the environmental and energy areas. During the past decade, I have been senior counsel to the Utility Water Act Group (UWAG) and the Utility Air Regulatory Group (UARG) in EPA rulemakings and appeals under the Clean Water Act and the Clean Air Act, respectively. These clients are ad hoc groups of electric utility systems and their trade associations, the Edison Electric Institute, the National Rural Electric Cooperative Association and, in the case of UARG, the American Public Power Association. In the representation of these groups, my firm has on occasion also participated in rulemaking proceedings under the Comprehensive Environmental Response Compensation and Liability Act of 1980 [the "Superfund Act"]. I also represent the Edison Electric Institute (EEI), the trade association of the investor-owned segment of the electric utility industry, in connection with the current efforts to amend the Clean Air Act. In addition, I am a member of the recently organized EEI Task Force on Compensating Persons Exposed to Toxic Substances.

My firm, Hunton & Williams, represents individual utility companies, oil companies, paper producers, pharmaceutical manufacturers and chemical manufacturers that own or operate facilities that are subject to numerous federal and state environmental requirements and liabilities, including specifically the Clean Water Act, the Clean Air Act, the Toxic Substances Control Act, the Resource Conservation and Recovery Act (RCRA) and the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA or the Superfund Act). As the senior lawyer in the firm's Energy and Environmental Law Team, I have usually been in charge of these efforts.

In bar activities, I have been a member of the Council of the American Law Institute since 1979. I am also a member of the American Bar Association and active in its Section of Corporation, Banking and Business Law. I was a member of the Council of that Section from 1976 to 1979, Chairman of its Ad Hoc Committee on the Federal Criminal Code from 1979 to 1981, and I am presently Chairman of its Program Committee and a member of its Tort Law Reform Committee. I should add that my opinions on the subject matter of this hearing are derived not only from my practice and participating in bar activities but also from my recent experience as one of the twelve members of the Superfund 301(e) Study Group.

I am testifying here today, however, not as a spokesman for my firm or any of its clients or any professional association with which I am connected. The views I express are solely my own, and they should not be attributed to my firm or any of its clients, the American Law Institute, the American Bar Association or the Superfund Study Group.

The Superfund 301(e) Study Group was composed of lawyers from four diverse professional legal organizations: the American Bar Association, the American Law Institute, the Association of Trial Lawyers of America and the National Association of Attorneys General. I was one of the three members designated by the President of the American Bar Association. Congress wanted a diverse group to foster the exchange of varying views, and Congress got it. There are virtually no issues in the current debate over various Congressional "Superfund" toxic injury compensation proposals which we did not discuss in the Superfund Study Group during 1981-82. The debate within our Group on some of these issues sometimes became so lively that it appeared no Report would ever be delivered to Congress. But with a few notable exceptions, such as its view of strict liability under the common law and punitive damages,[1] the resulting Report's discussion of existing statutory and common law is adequate. But as to the Study Group's recommendations, you should be aware that they were not in all respects unanimous. The Group agreed, accordingly, to the inclusion of separate statements of the individual members as a part of the Report.[2]

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[1] Superfund § 301(e) Report, Part 1, pp. 298-299 (Judge Breitel); pp. 319, 322-323, 337 n.33 (Freeman); see also Id., 344 n.48 (Freeman).

[2] Superfund § 301(e) Report, Part 1, pp. 286-359; note also the letters and other materials which are included as "Comments

(footnote continued)

For proper perspective on policy alternatives, you need to read these individual views as well as Professor Grad's report for the majority.

Since Weyman Lundquist, another member of our Superfund Study Group, who is scheduled to precede me today, will summarize the Report's recommendations, I will not repeat what he will say. It might be more helpful if I summarized my perception of the positive contributions made by the Report to the overall policy debate and then expressed my reasons for concurring in some but not all of the Report's recommendations. In the area of positive contribution:

1. The Report recognizes that data as to the scope of the problem are not now available -- therefore the problem may be one of perception instead of actuality.[3]
2. The Report correctly interprets the City of Milwaukee and Sea Clammers cases to conclude that no federal "common law" of tort or nuisance exists in this area but that state common law is alive and well.[4]

(footnote continued)

to the Appendices" at the rear of Part 2 of the Report, but which were inadvertently omitted from the Part 2 Table of Contents.

[3] The Superfund Study Group had much the same experience as this Subcommittee originally had in not being able to obtain much information on Superfund related matters from EPA. See e.g., Superfund § 301(e) Report, Part 1, pp. 21-39, 310-311; Part 2, Letters of Dec. 16, 1981, and Mar. 2, 1981, in the Comments to the Appendices. All references to page numbers are to those at the top of pages in the Congressionally printed Report.

[4] See Superfund § 301(e) Report, Part 1, pp. 87, 323-324; Part 2, pp. 151-152, letters of Dec. 16, 1981; Apr. 29, 1982; May 17, 1982, in the Comments to the Appendices.

3. The Report unanimously recommends against creation of a Federal statutory judicial tort or nuisance (Tier Two) remedy.5/

4. The Report recognized the problems associated with the Black Lung Act and rejected it as a model for recommendations in this area.6/

5. The Report rejected endorsement of the Enterprise and Market Share Liability Theories.7/

6. The Report, in recommending creation of a Tier One (administrative compensation system), rejected the notion of retroactivity in subrogation of Tier One claims.8/

7. The Report recommends that injuries covered by Workmen's Compensation be exempt from any Tier One system.9/

8. The Report recognizes that the statutory presumptions proposed for the Tier One administrative system would be inappropriate in the Tier Two judicial system.10/

9. Finally, the addition of Section III B of Report on insurance law and thought-provoking criticism of current joint and several liability rules were major contributions to thoughtful consideration of tort law reform generally.11/ In particular the recommendation of consideration of the substitution of "channelling" for "joint and several

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5/ See Superfund § 301(e) Report, Part 1, pp. 290-291, 307.

6/ See Superfund § 301(e) Report, Part 1, pp. 195, 178, 228.

7/ See Superfund § 301(e) Report, Part 1, p. 262.

8/ See Superfund § 301(e) Report, Part 1, pp. 176, 245-246.

9/ See Superfund § 301(e) Report, Part 1, p. 207.

10/ See Superfund § 301(e) Report, Part 1, p. 60.

11/ See Superfund § 301(e) Report, Part 1, pp. 162-192.

> liability" in both Tiers One and Two merits far more attention than it has so far received.12/ The adoption of "channelling" would result in swifter, full compensation with lower transaction costs and the elimination of duplicative insurance. The net result would be better for persons injured or damaged and for the public, which bears the ultimate costs of the system through the costs of goods and services.

Turning to the Report's recommendations, I concur in the recommendation for creation of a Tier One administrative compensation system for the reasons set forth in my separate statement at pages 308 to 310 of the Printed Report.13/ But there are five areas where I have problems with the recommendations and the Report:

1. I did not think we knew enough to make a recommendation to Congress on the source of the Tier One funds. For that reason, I did not concur in the recommendation to fund it through an add-on to the existing Superfund taxes.14/

2. I strongly concurred with Judge Breitel's dissent from the recommended statutory presumptions in Tier One.15/

12/ See Superfund § 301(e) Report, Part 1, pp. 181-183, 258, 324.

13/ For the convenience of the Committee, a reprint of my separate statement in the Superfund Report is reproduced as Attachment A to this testimony.

14/ See Superfund § 301(e) Report, Part 1, p. 245; but see, Freeman's Statement. Part 1, pp. 311-314.

15/ See Superfund § 301(e) Report, Part 1, pp. 213-219; but see Breitel's and Freeman's statements, Part 1, pp. 299-300, 314-318.

3. I joined Judge Breitel and Professors Anderson and O'Connell in deploring the failure to recommend the requirement of a limited binding election between the Tier One administrative remedy and the Tier Two adjudicatory remedy.16/

4. I thought Judge Breitel's criticism of the Report's overbroad description of "Strict Liability" was right on the mark.17/

5. I deplored the failure of our Group to address the punitive damages issue in the Report, despite our consideration of that issue in our meetings.18/

There are a number of bills pending in both Houses that deal with some of the issues we addressed in the Superfund § 301(e) Report. Unfortunately, some of the more interesting of the Study Group's suggestions such as consideration of the substitution of "channeling" for "joint and several" liability as the means for apportioning liability,19/ have not yet made their way into the political debate. Some of the Congressional

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16/ See Superfund § 301(e) Report, Part 1, pp. 197-205; but see, Joint Statement of Anderson, Breitel, Freeman and O'Connell. Part 1, pp. 286-288.

17/ See Superfund § 301(e) Report, Part 1, pp. 122-123, 265-266; but see, Breitel's Statement. Part 1, pp. 298-299; Freeman's letter on this in the Comments to Appendices. Part 2, Letter of Mar. 10, 1982.

18/ See, Freeman's Statement, Superfund § 301(e) Report, Part 1, pp. 322-323.

19/ See Superfund § 301(e) Report, Part 1, pp. 177-184, 258, 262-263; Schmalz, Superfund and Tort Law Reforms: Are They Insurable? 38 Bus.Law. 175 (1982) (Attachment D to this statement).

"Superfund" proposals, even those in the Senate which supposedly are intended to "implement" the Study Group's recommendations, contain provisions we considered and rejected.

II. AN EXAMPLE NOT TO FOLLOW: THE SUPERFUND ACT

Before I discuss some of the pending bills, I would like to summarize what has happened during the two and a half years since the Superfund Act was enacted because many of the issues raised in the interpretation and application of that act in determining who should pay for cleanup of hazardous wastes are directly relevant to who should pay to compensate persons injured or damaged by these wastes.20/

In the Congressional compromise that enabled the Superfund Act to be adopted in December 1980, the federal cause of action provisions in the Senate Bill21/ were deleted and the Superfund § 301(e) Study substituted. Thus private plaintiffs who seek recovery because of hazardous substances exposure have continued over this period to rely on state law, usually the

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20/ Part of this analysis of the Superfund Act follows an analysis prepared as a public service by my associate, Alfred R. Light, for the National Science Foundation. See National Science Foundation, An Addendum to a Research Report on Compensation for Victims of Toxic Pollution - Assessing the Scientific Knowledge Base (April 1983) (NSF 83-46).

21/ S. 1480, 96th Cong., 2d Sess. (1980).

state common law of tort and nuisance. To date, there have been relatively few damage awards associated with exposure to toxic substances except for asbestos and some substances which have been the subject of product liability litigation.22/ Despite the small number of awards, a growing number of court suits are being filed which request very high damages. EPA reported in 1982:

> If any of these suits are successful the potential third party damage costs associated with operating hazardous waste facilities would become much larger than currently available data show.23/

Congressional consideration of changes in what are today essentially state statutory and common law rules for compensating those with toxic substance related diseases involves complex interrelationships. As Judge Charles D. Breitel explained in his separate comments in the Superfund 301(e) Report:

> There are inextricable relationships among the issues of responsibility and liability, statutes of limitations, causation, components of damage recovery, latency of harm caused, social cost-benefit analyses, government regulation, and national interest in encouraging some (but not all) hazardous activities.24/

22/ Superfund § 301(e) Report, Part 1, p. 32.

23/ 47 Fed. Reg. 16550 (Apr. 16, 1982).

24/ Superfund § 301(e) Report, Part 1, pp. 295-296.

The witnesses here today are lawyers, but this and other appropriate Committees need to hear about these interrelationships from a broader community of experts, including economists, businessmen and political scientists as well as medical and scientific experts. Your series of hearings, involving witnesses from the scientific, legal, environmental and insurance communities is a useful step in this process.

The tough balancing of social costs and benefits that should be made before any drastic changes are made in liability rules needs information and insights that go beyond the legal profession. Lawyers, whether they usually represent plaintiffs or defendants, stand to profit more and more as litigation escalates; but that is not true of society as a whole. As litigation costs mount, awards increase in number and amount, and insurance premiums escalate. Most of these increased costs are ultimately imposed on the public through higher costs of goods and services. Often the percentage of these costs of the whole system that actually go to compensate persons injured is quite low. We must continually ask ourselves whether those extra, "transactional," costs are justified and if there are not better ways or better systems.

If you do decide that additional legislation is needed, I urge you to take great care in drafting it. To illustrate this point, it might be useful to examine some of the problems that have resulted from the poor drafting of the present liability provisions regarding waste site cleanup in the Superfund Act.

A. Preemption and Savings Clause Provisions

The Superfund Act's preemption provision and savings clauses have created substantial uncertainty and greatly contributed to the volume of litigation.

The Superfund Act's section 114(c), 42 U.S.C. § 9614(c), states in part:

> Except as provided in this Act, no person may be required to contribute to any fund, the purpose of which is to pay compenstion for claims for any costs of response or damages or claims which may be compensated under this title.

This provision has cast a substantial shadow over state superfund-type taxes.25/ The floor debate on this provision, the only legislative history available, is confusing.26/ At

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25/ See, e.g., Superfund § 301(e) Report, Part 1, pp. 268, 271 and Part 2, letter of May 17, 1982, in "Comments to the Appendices," at the end of that volume. H.R. 6705, 97th Cong., 2d Sess. (1982) (Cong. LaFalce) would have removed these doubts by repealing this provision.

26/ Compare 126 Cong. Rec. S14,981 (daily ed. Nov. 24, 1980) (colloquy between Senators Randolph and Bradley) with 126 Cong. Rec. S15,004 (daily ed. Nov. 24, 1980) (remarks of Sen. Helms regretting the preemption), and see 126 Cong. Rec. H11,788 (daily ed. Dec. 3, 1980) (remarks of Rep. Florio); see Grad, A Legislative History of the Comprehensive Environmental Response, Compensation and Liability ("Superfund") Act of 1980, 8 Colum.J.Envir.L. 1, 31 (1982).

present, at least 35 states, including New Jersey, California, New York, Minnesota, and most recently Missouri, have enacted some type of state superfund legislation.27/ Section 104(c)(3) of the Superfund Act requires that the states provide at least ten percent (10%) of the funds for remedial actions at privately owned sites, fifty percent (50%) of such costs at sites owned by the state or its subdivision.28/ The states must also provide for future, long term maintenance. The extent of this preemption of the state's potential sources of revenues for those purposes is thus no academic matter. The Superfund Study Group recommended "that Congress clarify its intent relating to the preemptive reach of § 114."29/ Any legislative amendments to the Superfund Act should address this issue.30/

27/ United States Congress, Office of Technology Assessment, Technologies and Management Strategies for Hazardous Waste Control 369 (Mar. 1983); Minnesota's Superfund bill, perhaps the most comprehensive yet enacted, passed the Minnesota legislature on May 5, 1983 (H.F. 76).

28/ 42 U.S.C. § 9604(c)(3).

29/ Superfund § 301(e) Report, Part 1, p. 271; *see also* U.S. Congress, Office of Technology Assessment, *supra*, at 368.

30/ Not only should Congress clean up the present ambiguity, but it should also look at the consequences of other proposed changes in that Act in light of this prohibition. For example, several Senate bills would expand the "Superfund" reimbursement to cover "victim compensation" payments which may presently be covered by state victim compensation funds and state workers' compensation programs. Section 114, if not amended, could be interpreted to bar further direct assessment of "Superfund taxpayers" for those state funds.

The Superfund Act's saving clauses have generated some controversy. Section 302(d) of the Act, 42 U.S.C. § 9652(d), provides:

> Nothing in this Act shall affect or modify in any way the obligations or liabilities of any person under other Federal or State law, including common law, with respect to releases of hazardous substances or other pollutants or contaminants.

At least one Senator at one point had the rather unusual notion that the existence of this clause amounted to a Congressional authorization for federal courts to infer or create a federal common law private cause of action for damages.31/ This confusion over the meaning of the Superfund Act's as well as other environmental statutes' savings clauses has led to the unusual attempt to create new federal remedies by modifying such clauses for the stated purpose of creating or "reviving" the federal common law of tort and nuisance.32/ But congressional attempts to create private rights of action in this indirect way would,

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31/ See 126 Cong. Rec. S15,640 (daily ed. Dec. 16, 1981) (remarks of Sen. Stafford stating that the superfund law "expressly preserved both Federal and state common law," although the Supreme Court has never found a federal private cause of action to exist in this area).

32/ E.g., 126 Cong. Rec. S12,249 (daily ed. Oct. 27, 1981) ("technical amendment" to Clean Water Act in S.1716); S. 2867, 98th Cong., 1st Sess. § 11(c) (1983); see H.R. Rep. No. 98-198 (Part 1), 98th Cong., 1st Sess. 49 (1983); H.R. Rep. No.97-570, 97th Cong., 1st Sess. 21-22 (1982) (H.R. 6307, § 10).

if enacted, prove futile because they would probably be held unconstitutional.33/

The creation of federal remedies (through whatever mechanisms) can preempt state law implicitly as well as explicitly. For example, state punitive damages for nuclear occurrences may be impliedly preempted by the Atomic Energy Act.34/ These cases suggest that great care must be taken by Congress to state expressly and clearly in statutory language the relationship between existing state remedies and any new federal remedy. Both preemption and savings clauses produce confusion and unnecessary litigation when the Congress leaves its intent unclear.

B. Apportionment and the "Joint and Several" Issue

EPA received much criticism in the past for the relatively slow pace of its Superfund cleanup program. The Superfund § 301(e) Report stated, for example:

33/ *See* Superfund § 301(e) Report, Part 2, Letters of Dec. 16, 1981, and May 17, 1982, in "Comments to the Appendices;" *see* pp. 69-74 below.

34/ *See* *Silkwood* v. *Kerr-McGee Corp.*, 667 F.2d 908, 922-923 (10th Cir. 1982), *juris. postp'nd* No. 81-2159 (Jan. 10, 1983). *UNC Resources, Inc.* v. *Benally*, Civ. No. 80-750B (D.N.M. Oct. 12, 1982).

> It has come to the Study Group's attention that there have been some administrative delays in the management of claims for Superfund support in cleaning up hazardous waste problem sites, and . . . that only a small number . . . are likely to be cleaned up by Superfund contributions in the foreseeable future.35/

Other criticisms have been less circumspect.36/

Some trial judges faced with EPA attempts to sue responsible parties for Superfund "reimbursement" *prior to* spending any government funds appear to be growing impatient. For example, a federal district court recently wrote in a memorandum opinion:

> For reasons unclear, neither the United States nor New Hampshire has sought to make use of its own funds for the purpose of emergency clean-up, removal and containment action pursuant to sections 104 and 107 of CERCLA, 42 U.S.C. §§ 9604, 9607 and New Hampshire's Hazardous Waste Cleanup Fund, RSA 147-B (Supp. 1981) ("HWCF") . . .
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> . . . . .

35/ Superfund § 301(e) Report, Part 1, pp. 270-271.

36/ *E.g.*, Baise and Bourdeau, *EPA's Implementation of the Superfund Response Programs*, 3 Envir. Analyst 3 (May 1982); *Administrative Delays Plague Cleanup Under Superfund, Indiana Congressman Says*, 13 Envir.Rep. (BNA) 167 (Jun. 11, 1982); Pain, *Mega-Party Superfund Negotiations*, 12 Envir.L.Rep. 15054 (Nov. 1982); Rogers, *The Generators' Dilemma in Superfund Cases*, 12 Envir.L.Rep. 15049, 15053 (Nov. 1982) ("EPA and the Justice Department must be more responsive and more consistent, if the CERCLA cases are not to turn into enormous "reverse" class actions that consume millions of dollars in legal fees, retard cleanup efforts, and end up frustrating all involved.").

> The circumstances herein depict federal and state governments which are either unable or unwilling to take the necessary financial steps to protect the public from what are long extant but apparently recently discovered dump sites containing hazardous wastes, and the same plaintiffs are similarly unwilling to proceed against current owners who are in posession of such sites.[37]

While some of the past delays in Superfund cleanup have been blamed on EPA personnel, much of the delay is attributable to Congress' poor draftsmanship. One of the most critical areas has been the Act's failure to articulate expressly and unequivocally who can be held liable for what and when. The Act articulates no principles of joinder or apportionment of liability among liable defendants. It also is ambiguous as to whether "strict liability" was intended to involve traditional balancing notions or to be equated to "absolute liability." Finally, the act and its legislative history are ambiguous as to the extent Congress intended it to be retroactive, and by retroactive I mean to impose liability under federal law on persons for past actions which at the time they occurred would not have been illegal or actionable under federal law.

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[37] United States v. Johns-Manville Sales Corp., 13 ELR 20310, 20311-20312 (D.N.H. Nov. 15, 1982); appeal docketed No. 83-1152 (1st Cir. Mar. 3, 1983).

Because the Act may cast a broad net of liability apparently to include as jointly liable many who may have had only a tenuous connection to a waste site and whose conduct may have been legal and otherwise nonactionable under then existing law, the relevant principles of apportionment have become very important in presently pending Superfund cleanup cases. Congress, however, refused to address the issue directly. "Joint and several" liability language in early drafts was eliminated without adequate explanation. Comments of Congressmen and Senators during floor debate as to the meaning of this deletion are diametrically opposed.38/ EPA and the Justice Department have asserted a broad application of joint and several

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38/ 126 Cong. Rec. S14,964 (daily ed. Nov. 24, 1980) (Sen. Randolph); 126 Cong. Rec. H11,787 (daily ed. Dec. 3, 1982) (Cong. Florio), 126 Cong. Rec. S15,004 (daily ed. Nov. 24, 1980) (Sen. Helms). In late 1981, Senator Stafford continued the debate in post hoc legislative history. He stated:

> As disappointing as it is to me, I am realistic enough to recognize that it is the role of modern Washington lobbyists to press their clients' interest to the bitterest end. Those charged with the responsibility for deciding these issues, however, must bear in mind that such arguments are being advanced with a stated goal in mind, not for the sake of an abstract pursuit of justice or accuracy.

127 Cong. Rec. S9335 (daily ed. Sept. 9, 1981). "Joint and several liability" is being challenged in several pending Superfund cases. E.g., U.S. v. Conservation Chemical Co., No.82-0983 (W.D.Mo.); U.S. v. Chem-Dyne, No. C-1-82-840 (S.D.Ohio).

liability notwithstanding the deletion. On the other hand, one distinguished academic commentator has stated that "it is . . . clear that joint and several liability was not intended."39/ The most probable "actual" Congressional "intent" is that Congress deliberately failed to resolve the political impasse leaving to "common law principles to determine when parties should be severally liable."40/

Thus as often occurs, the political compromise to "leave it to the courts" led inevitably to delay and litigation rather than to the rapid cleanup of hazardous waste sites. The lesson to be learned is that legislative compromises that leave statutes fuzzy and unclear and thrust the task of resolving the ambiguities and conflicts upon the federal courts often postpone rather than expedite the solution to the substantive problems to which the legislation is addressed. Given the already overcrowded dockets of our federal district courts, it literally takes years in many instances to get initial decisions. Then decisions in these key test cases are appealed to the overburdened Circuit Courts of Appeals, where eventually conflicting decisions will be handed down. And then, but only

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39/ 1A Grad, Treatise on Environmental Law, § 4A.04, at 4A-114.

40/ 126 Cong. Rec. S14,964 (daily ed. Nov. 24, 1980) (Sen. Randolph).

when those decisions are numerous enough, will the overburdened Supreme Court grant certiorari to resolve those conflicts. In the intervening time -- five or six years -- everyone involved in the process is forced to litigate the issue where the financial stakes are large. The social costs of such a system -- including the "transactional costs," or to call a spade a spade, the legal fees -- are enormous. If, on the other hand, Congress had fought the matter out and written the legislation carefully and clearly, the beneficial results intended -- cleanup of hazardous waste sites -- could have been obtained much more quickly, and often through voluntary efforts and settlements rather than litigation.

California, perhaps learning the lesson of the folly of leaving such important issues to "common law" development, provided explicit apportionment rules in its 1981 state superfund act.41/ Minnesota also did the same thing in May.42/ Should Congress move forward in this session with hazardous substance injury liability legislation, it should follow the lead of these states and include an addressing of the apportionment problem.

41/ Cal.Health & Safety Code §§ 25360-25366 (1981).

42/ H.F. 76, enacted May 10, 1983.

Furthermore, "joint and several" liability, it is now clear from the Superfund experience, is no answer at all because it simply shifts the burden of seeking contribution and the apportionment of liability among multiple defendants to the "deep pocket" defendant. The Superfund § 301(e) Report recognized this by recommending that "alternative approaches to apportionment" be examined in connection with its notion that "the burden of allocating separate contributions" be placed "among a number of [liable] defendants."43/ One of our most compelling reasons for that recommendation was our concern about rising insurance costs. If everyone in every stage of the industrial process of generating, transporting, or storing hazardous wastes must insure against total liability resulting not only from his own activities but also from the activities of others subsequently in the process, it is obvious that we have duplicative -- indeed treble -- insurance costs imposed on the consuming public. Where multiple generators dispose of their wastes at a common RCRA licensed site -- and with the commonly supervised disposal at such sites encouraged by that law -- imposition of joint and several liability on all who dispose of their wastes there for all that might happen there could well impose insurance costs on U.S. manufacturers that

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43/ Superfund § 301(e) Report, Part 1, p. 258, 344n.48.

place their products at a severe competitive disadvantage with foreign competitors -- both at home and abroad. As I emphasized in my separate Superfund comments[44] -- and in my earlier writings to my fellow members of that group[45] -- we need to look carefully at where -- and with what effect -- the incidence of the costs imposed by changes in the law will fall. We must take care that in "internalizing" one set of social costs to help one segment of society -- here the costs of cleanup and compensation for the injuries or damages caused by exposures to hazardous substances -- we do not impose unintended and unfair costs on other segments -- such as unemployment or the unavailability of socially desirable goods and services. If you cast the net of joint and several liability wide enough and make the imposition of high costs capricious enough, then the risks of doing business may become so great as to deter people from engaging in necessary or socially beneficial economic activities in this country. A careful balance of competing social values must be made. As Judge Breitel said in his Comments in the Superfund Report:

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[44] Superfund § 301(e) Report, Part 1, pp. 311-314.

[45] Superfund § 301(e) Report, Part 2, letters of Mar. 2, 1982; Mar. 10, 1982; Apr. 12, 1982 in "Comments to the Appendices."

> There are many hazardous substances and activities which must be balanced against the then - or presently - available alternatives. In preventing many of humanity's ills, disasters, and malnutrition, elimination of hazardous substances without a then-known alternative or one now known, the costs in death, disease, and environmental harm may be or may have been greater than that caused by the hazardous substances or activities. The point is that the pristine jungle is not a safer place for human habitation or activity than the inevitably danger-ridden civilized society which has given us the boon (problem) of enormous growth of population with increasingly greater longevity and health.[46]

Another aspect of the "joint and several" liability debate involves the insolvent "responsible party." To date, EPA's Superfund and RCRA enforcement policy position generally has been that the remaining solvent responsible parties must pay the entire cleanup costs, including the insolvent party's "share," without any contribution from the Superfund. The Fund is used to finance a cleanup only when enough responsible parties refuse to settle for total cleanup costs. In a situation where insolvent parties contributed substantially to the problem requiring cleanup, a solvent responsible party (or parties) is likely to resist a government "offer" to settle because of his recognition that his right of contribution against other responsible parties is a hollow one. EPA officials claimed, however, that this was not a practical problem in the first round of Superfund cases because the generators they targeted generally *are* solvent.[47]

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[46] Superfund § 301(e) Report, Part 1, pp. 297-298. And *see, generally*, National Science Foundation, *Addendum to a Research Report on Compensation of Victims of Toxic Pollution -- Assessing the Scientific Knowledge Base* (April 1983); Light, *Toxic Injury Compensation: One More Overview of Legislative Proposals*, 15 Nat. Resources L. Newsletter 2, 8-11 (Spring 1983).

Some recent legislative proposals would further delay Superfund cleanup if they are enacted. These proposals would piggyback claims for compensation for damages to person or to property into cleanup proceedings through either broadened citizens suits that permit pendent jurisdiction over claims under state law or through a new federal cause of action[48] or through a private right of intervention in EPA enforcement actions.[49] In my opinion, enactment of either proposal would delay rather than expedite the attainment of the twin goals of (1) protecting of our people and the environment from unnecessary and undesirable waste generation, handling and disposal practices and (2) providing fair compensation to those injured or damaged through the functioning of our economy.

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[47] A related problem arises when *all* the responsible parties the government can identify are insolvent. The courts in this situation must confront the "clash of policies protecting the environment and policies protecting the bankrupt and its creditors." Rosenberg, *Bankruptcy and Environmental Regulation: An Emergency Conflict* 13 Envir.L.Rep. 10099 (Apr. 1983); *see* Moore, *Bankruptcy Stays Thwarting Cleanups*, Legal Times (May 9, 1983). A bankruptcy stay may frustrate government efforts to force cleanups. *See In re Kovacs*, 681 F.2d. 454 (6th Cir. 1982); *U.S.* v. *Johns-Mansville*, 13 ELR 20310 (D.N.H. Nov. 15, 1982), *appeal docketed* No. 83-1152 (1st Cir. Mar. 3, 1983); *In Quanta Resources*, Civil No. 82-3524 (D.N.J., Jan.24, 1983); *In re Crystal Chemical Co*., Bankr. No. 81-02901-HB-4 (S.D. Tex., filed Jul. 15, 1982); environmental prosecutors can be expected to argue vigorously that "enforcement actions for mandatory injunctive relief to compel environmental cleanup are not subject to the bankruptcy stay." D. Muchnicki, *The Bankruptcy Code and State Police Power Enforcement*, Envir.Prot.Rep. 1, 5 (Nat. Ass'n of Atty. Gen. Mar. 1983). In this as well as the last Congress, Congressman Florio introduced an amendment to the bankruptcy laws that would give priority to government enforcement claims over secured and unsecured creditors; H.R. 2767, 98th Cong., 1st Sess. (1983); H.R. 7172, 97th Cong., 2d Sess. (1982). It is unclear whether the secured creditor whose security is impaired by a government claim will have a right against the Superfund for reimbursement.

The process of enforcement of laws and regulations to avoid or mitigate exposures to hazardous wastes is a police function. Over the years we have learned that law enforcement is best left to the policeman and not to vigilantes. Moreover, we normally do not permit criminal prosecutions to be tried together with civil damage actions. Finally, experience shows that the process of compensation for injuries or damages, whether under an administrative (insurance type) system or a litigative system, functions best where most of those who must pay do so voluntarily. And in a litigative system, the transactional costs are magnitudes lower where actual litigation is the exception and not the rule. If we do not halt the current trend toward encouraging more and more litigation and do not find better means for resolving most of these problems, the whole process will choke on itself, leaving our environment despoiled, those injured uncompensated, our courts hopelessly overburdened and our economy gravely threatened.

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[48] See H.R. 2867, 98th Cong., 1st Sess. § 11(b) (1983) (modifying RCRA citizen suit § 7002).

[49] E.g., S. 1363, 98th Cong., 1st Sess. § 20 (1983) (giving private parties right to intervene in government RCRA § 7003 suits).

C. Generator ("Vicarious, Strict") Liability

Another issue where Congress was deliberately equivocal concerning the Superfund Act involves the standard of care to which defendants are to be held under the statute. Specific references to "strict liability" were deleted, and instead the Act defines the Act's standard of liability to be the same as that under § 311 of the Federal Water Pollution Control Act.[50] While it is clear that this standard is one form of strict liability for the owner or operator of a vessel from which there is a hazardous release, it is much less certain what the reference means in other contexts such as the liability of hazardous waste generators arising out of disposal of wastes by others at sites off of the generator's property.

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[50] 42 U.S.C. § 9601(32).

Superfund appears to impose liability for cleanup costs on generators, transporters and owners and operators of waste facilities. However, there are now some indications that courts will not impose liability on those who do not contribute substantially to (are not a legal cause of) the problem requiring cleanup.[51] That is, in the absence of negligence, courts may hold that a generator's activity did not proximately cause the damage resulting from an accident at an offsite disposal facility operated by another.[52]

The scope of generator liability within the context of federal enforcement of RCRA is also a subject of hot dispute today. A question under RCRA section 7003 has been whether a generator is always a person "who contributes to" an imminent hazard at someone else's disposal site. A post-hoc interpretation of these words appears in a Senate Committee report on the 1980 Amendments to RCRA. The report states:

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[51] *City of Philadelphia* v. *Stepan Chemical Co*., 544 F.Supp. 1135, 1143 n.10 (E.D.Pa. 1982) (dicta); *see State of New Jersey Department of Environmental Protection* v. *Ventron Corporation, et al.*, 182 N.J.Super. 210 (App.Div. 1981) (New Jersey spill compensation Act); Rodberg, *Apportionment of Damages in Hazardous Waste Litigation*, in Mott. ed., Hazardous Waste Litigation 1982 183, 213 (1982); Dore, *The Standard of Civil Liability for Hazardous Waste Disposal Activity: Some Quirks of Superfund*, 57 Notre Dame Law. 260, 276 (1981).

[52] *Cf*. *United States* v. *Wade*, 546 F.Supp. 785 (E.D.Pa. 1982), *appeal pending* No 82-1715 (3d Cir.) (RCRA § 7003 and Superfund § 106 do not apply to past non-negligent off-site generators of wastes).

> For example, a company that generated hazardous waste might be someone "contributing to" an endangerment under section 7003 even when someone else has deposited the waste in an improper disposal site . . . where the generator had knowledge of the illicit disposal or failed to exercise due care in selecting or instructing the entity actually conducting the disposal.53/

Justice Department prosecutors, who have the clearest interest in the broadest possible theory of generator liability under federal statutes, concede that the particular factual setting of an incident is important to deciding whether there should be generator liability.54/ And the Carter Administration's Interagency Task Force on Compensation and Liability for Releases of Hazardous Substances did not consider absolute generator liability to be among its options for liability under the Superfund Act.55/

53/ S.Rep. No. 172, 96th Cong.,2d Sess. 5, reprinted in [1980] U.S. Code Cong. & Ad. News 8605, 8669; cf. U.S. v. Wade, 546 F.Supp. 785, 791 (E.D.Pa. 1982) (the full Senate Committee discarded the strict liability approach).

54/ E.g., see Briefs in pending United States v. Chem-Dyne, Civil Action No. C-1-82-840 (S.D.Ohio) (parties appear to agree that the Superfund Act does not require joint and several liability in all cases); Reply Brief of the United States on Motion for Partial Summary Judgment and Injunctive Relief, United States v. Petro Processors of Louisiana, 2 Chem. & Rad. Waste Rep. 120, 126 (M.D.La. 1981).

55/ See Department of Justice, The Superfund Concept, June 1979, at 74-77.

The generator ("vicarious strict") liability and apportionment of liability ("joint and several") problems are inextricably intertwined, as various commentators have pointed out.

> In the debate some seem to have lost sight of, or at least glossed over, the real significance beyond Congress' omission of joint and several liability. Inclusion of joint and several liability in the statutes would reflect a legislative judgment of public policy akin to what underlies master-servant or partnership vicarious liability. In other words, a generator who is a substantial factor in bringing about some of the injury might constitutionally be held liable for all of the harm caused by the owner/operator of the facility on public policy ground regardless of legal (proximate) causation in the common law sense, or the indivisibility or not of the injury. The absence of such a joint and several liability provision in Superfund means that Congress has not come to grips with any public policy issue.56/

In my view, the single most important issue in the current tide of Superfund litigation today is whether a nonnegligent generator can be held liable for a major share of cleanup costs simply because of evidence demonstrating that his wastes were present when a release occurred or was threatened at a facility handling the wastes of many generators. The potential for grossly disproportionate vicarious liability

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56/ Rodberg, Apportionment of Damages in Hazardous Waste Litigation, Hazardous Waste Lit. 1932, 183, 204-205 (1982).

explains why some companies refused to settle in the recent Chem-Dyne agreement.

> "We didn't put it there and you'll never take us alive," was the reasoning attributed to some companies by an EPA official.57/

D. Retroactivity

Intimately related to generator liability issues in Superfund litigation is an issue that, quite remarkably, has not been raised directly in the first Superfund suits originated by the United States to reach initial judicial opinions: retroactivity. A court in one Superfund action brought by a state did find that Congress intended for the statute to be applied retroactively in general.58/ Retroactive application of a more stringent liability standard can render an unobjectionable legal standard unconscionable.59/ In addition, retroactive application of a vicarious, strict liability raises

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57/ Ward, Settling at Chem-Dyne, 1 Envtl. Forum 7, 15 (Dec. 1982).

58/ Ohio v. Georgeoff, Civil No. C-81-1961 (N.D. Ohio, May 3, 1983). The question of the constitutionality of this retroactivity was reserved for future resolution by the trial judge in this case.

59/ Cheek, Risk-Spreaders or Risk Eliminators? An Insurer's Perspective on the Liability and Financial Responsibility Provisions of RCRA and CERCLA, 2 Va.J.Nat.Resources L. 149, 167 (1982).

serious constitutional due process questions analogous to the explicit prohibition of *ex* *post* *facto* laws in the criminal area.60/ Indeed, the concepts of fairness and substantial

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60/ U.S. Const., Art. I, § 9, cl. 3 (Congress forbidden to pass ex post facto laws); U.S. Const., Art. I, § 10, cl. 1 (state legislatures forbidden to pass ex post facto laws); the framers of the Constitution intended the clauses to apply to retroactive civil legislation as well as criminal legislation. *See* Crosskey, *The Ex Post Facto and Contract Clauses in the Federal Convention: A Note on the Editorial Ingenuity of James Madison*, 35 U.Chi.L.Rev. 248 (1968); Field, *Ex Post Facto in the Constitution*, 20 Mich.L.Rev. 315 (1922) but the clauses have been construed to apply *per se* only to criminal statutes. *Calder* v. *Bull*, 3 U.S. (3 Dall.) 386 (1798); the following state constitutional provisions prohibit all retroactive legislation: Colo. Const., Art. II, § 11; Ga. Const., Art. I, § 3(2); Idaho Const., Art. XI, § 12; Mo. Const., Art. I, § 13; N.H. Const., Art. I, § 23; Ohio Const., Art. II, § 28; Tenn. Const., Art. I, § 20; Tex. Const., Art. § 16; *cf*. *Murrill* v. *State Board of Accountancy*, 97 Col.App.2d 709, 218 P.2d 569 (1950) (retroactivity violated due process clause); *Gayle* v. *Edwards*, 261 Ala. 84, 72 So.2d 848 (1954) (state constitutional violation found in failure to disclose in title of legislation its retroactive effect); Sutherland, Statutory Construction, §§ 41.02 - 42.12; *Lundquist* v. *Coddington Bros., Inc.*, 202 F.Supp. 12 (W.D. Wis. 1962) (statute may not apply retroactively to revive a cause of action already barred by the statute of limitations); *Haase* v. *Sawieki*, 20 Wis.2d 308, 121 N.W.2d 876 (1963) (statute attempting to reinstate cause of action objectionable); *Massey* v. *Sullivan County*, 464 S.W.2d 548 (Tenn. 1971) (statute abolishing rule of no contribution for joint tortfeasors cannot be applied retroactively). A law will not be construed as retroactive unless the act clearly, by express language or necessary implication, indicates that the legislature intended a retroactive application. *United States* v. *Whyel*, 28 F.2d 30 (3d Cr. 1928); *Crawford* v. *Worth*, 447 F.2d 738 (5th Cir. 1971); *First Nat. Bank in Billings* v. *First Bank Stock Corp*., 197 F.Supp. 417 (D.Mont. 1961); *Elliot* v. *Volpe*, 328 F.Supp. 831 (D.Mass. 1971); *Douglas Aircraft Co*. v. *Cranston*, 58 Cal.2d 462, 24 Cal.Rptr. 851, 374 P.2d 819 (1962) ("a statute should be given the least retroactive effect that its language reasonably permits."); *Mau* v. *Superior Court*, etc., 101 Cal.App.3d 875, 881; 161 Cal.Rptr. 895, 898 (1980)

(footnote continued)

justice which underlie these constitutional concepts also serve as the philosophical underpinning for the general rule of construction that a statute is to be given only prospective application.61/ When a New Jersey appellate court was presented with the argument that the liability standard for cleanup costs under the New Jersey Spill Control and Compensation Act62/ was unconstitutional as retroactively applied, the court held that there was no problem because the statute did not actually impose a new "substantive liability." Instead, it held that this state precursor of the national Superfund law "was remedial only, providing a procedure for the imposition of costs to pay for the cleanup of prior wrongful discharges."63/ One legal commentator who has thought about these issues in the context of the federal Superfund Act has adopted a somewhat similar solution. He construes the "due care" defense to liability as

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(footnote continued)

(unfairness of retroactive application to insurers who relied on the previous law in setting premium rates).

61/ See United States v. Heth, 7 U.S. (3 Cranch) 399 (1806).

62/ N.J.S.A. 58: 10-23 et seq (1977).

63/ New Jersey v. Ventron, 182 N.J. Super. 210 (App. Div. 1981) (emphasis added); cf. State Dept. of Env'tl Prot. v. Exxon Corp., 151 N.J. Super. 464, 376 A.2d 1339 (1977) (constitutional difficulties preclude retroactive application of New Jersey Spill Compensation and Control Act).

preventing the "imposition of strict liability" on past off-site generators under the Act.64/ While this same commentator has concluded that Congress generally intended for the liability set forth in the Act to apply retroactively, he admits that the issue is not free of doubt:

> It is easy to argue, of course, that if retroactive effect were intended, Congress could have simply inserted a clause clearly stating that the liability provisions were to apply to pre-enactment waste generators. The noticeable absence of such a clause may create enough ambiguity to require an examination of the Act's legislative history to determine if Congress displayed a clear intent to permit retroactive construction.65/

The Senate Report on S.1480 explained that the justifications for the imposition of strict liability in the Superfund context were to promote fairness and equity, to encourage the exercise of greater care, and to cause "the costs of injuries [to] be borne by the activities which caused them."66/ But none of these underlying purposes is served through retroactive application of such a standard. As many commentators have pointed out, such application would "externalize, rather than

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64/ Note, Generator Liability Under Superfund for Clean-Up of Abandoned Hazardous Waste Dumpsites, 130 U.Pa.L.Rev. 1229, 1263, 1263 n.176 (1982).

65/ Id., at 1241.

66/ S.Rep.No. 96-848, 96th Cong., 2d Sess. 33-34 (1980).

internalize, costs by imposing liability for harm on innocent parties . . . [whose] conduct which, under the laws applicable at the time, was neither unlawful, nor tortious, nor a nuisance . . . ."67/ Professor Kenneth Abraham of the University of Maryland, said recently in explaining the insurance implications of the combination of strict, joint and several, retroactive liability:

> Recent developments in tort law also may hinder accurate insurance pricing. Such doctrinal innovations as shifting burdens of proof as to causation and apportionment of damages, the market share liabilty [sic] concept, and joint and several liability by independent tortfeasors are not merely alterations in the traditional burdens of proof. In practice, the doctrines enable some plaintiffs to recover damages from defendants who have not actually caused the loss (or the entire loss) in question. Minimally culpable defendants, for example, may be held liable for all damages suffered by a victim when, in fact, his injuries resulted from the combined activities of many other actors. Insurance premiums will have to take account of this liability exposure.68/

Finally, the imposition of these doctrines on a retroactive basis could push the cost externalization over that Plimsoll line of "due process."69/

67/ Cheek, supra, at 167-168. And see my letter to Frank Grad of March 10, 1982, which is included in the "Comments to the Appendices" in the back of Part 2 of the Superfund § 301(e) Report.

68/ Abraham, Cost Internalization, Insurance and Toxic Tort Compensation Funds, 2 Va.J.Nat.Resources L. 123, 129-130 (1982).

69/ Fikes v. Alabama, 352 U.S. 191, 199 (1956) (Frankfurter, J., concurring).

Notwithstanding all this, neither the generators nor the amici Chemical Manufacturers Association initially raised the retroactivity issue in the Wade case, which has become a central focus for judicial interpretation of the Superfund Act. As the United States pointed out in its reply brief:

> Nowhere do the Generators or CMA confront the anomaly created by the decision below: the very same generators who are subject to recoupment after EPA undertakes remedial action are not subject to an order under the endangerment provisions to assist in abating the hazards to which their wastes have contributed.70/

But the anomaly cuts both ways. It would also disappear if the court recognized that no new substantive liability was created under the Superfund Act with respect to past acts, i.e., if a due care standard embodying previous common law is the liability standard of section 107 as applied to past acts. And if this is not the correct interpretation, courts will ultimately have to confront the constitutional questions.

If Congress decides to enact new legislation creating a federal administrative remedy or a federal cause of action for persons who have been injured or whose property has been damaged by hazardous wastes, it should scrupulously avoid imposing liability retroactively. As I pointed out earlier, one

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70/ Reply Brief for the Appellant, U.S. v. Wade, No. 82-1715, at 4, Haz.Waste Lit.Rep. 3859 (Apr. 5, 1983).

of the most important decisions in our Superfund Study deliberations was our unanimous recommendation against any form of retroactivity in federal legislation.71/

E. Punitive Damages

Moving from pending Superfund litigation and issues that have been or should have been raised in those cases, to issues in future cases, a major issue is the availability and standard for punitive damages under the Act. This issue is quite recent because the Justice Department had not filed a case in which it felt that it was appropriate to ask for punitive damages until the Western Processing case filed on July 1.72/ An award of punitive damages must follow a defendant's failure "without sufficient cause to properly provide removal or remedial action upon order of the President," actually his designee under the Act--the EPA.73/ In the Stringfellow litigation, for example, the government decided not to pursue a punitive damages remedy because of the necessity for a time consuming issuance of administrative orders which would then be

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71/ Superfund § 301(e) Report, Part 1, pp. 245-251, 319-320.

72/ United States v. Western Processing, et al., No. C83-252M (W.D. Wa.).

73/ CERCLA § 107(c)(3), 42 U.S.C. § 9607(c)(3); see Exec. Order No. 12316, 3 C.F.R. 168 (1982).

violated. Also in that case, since California wished to seek recovery of its costs in the lawsuit, an enforcement problem could result if the government followed the administrative order rather than immediate litigation route. That is, since EPA questions its own authority to order persons to make payments to a state administratively under Superfund § 106, California might still have to sue the Stringfellow defendants independently to recover its incurred response costs even if the defendants complied with EPA's order.

It is clear, however, that punitive damages requests under the Superfund Act will be forthcoming, perhaps with more cases that could be filed early next year, according to former Assistant Attorney General Carol Dinkins. EPA and the Justice Department may seek such damages in any fact situation where the statute allows and will, of course, seek such awards in egregious cases. However, all the interpretational and constitutional problems we have discussed above with respect to the implication of rights not expressly granted in the statute, the joint and several issue, vicarious strict liability, and the retroactive application of any of these concepts are multiplied when the focus is an award of punitive damages. In addition, while EPA is apparently only now developing its criteria for when it will seek punitive damages, to the extent common law punitive damages requirement such as scienter and outrageous conduct are lacking in those criteria, there obviously will be challenges in the courts to their imposition.74/

Whether or not Congress amends Superfund or RCRA to give persons injured by wastes regulated under either statute federal remedies in addition to those now existing under state law, it needs to face up to the punitive damages issue. In this regard, I repeat what I said in my separate statement in the Superfund Report:

> I also believe that punitive or exemplary damages have no socially desirable role . . . when a comprehensive scheme of regulation such as RCRA, encompassing civil and criminal sanctions, is in place. I regret that my colleagues who were divided over the issue decided not to address it one way or the other in the Report. For my part, I believe that where Congress has exercised its commerce power to impose a comprehensive system of regulation over an industrial activity or practice and elects to leave private remedies to State law, it should nevertheless preempt any State punitive and exemplary damages since in essence they duplicate the criminal and civil sanctions provided in the Federal statute. Vigilantes have no place when the sheriff takes over. In my view, that duplication also constitutes an unreasonable burden on interstate commerce since it may furnish a major deterrent to engaging in

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74/ To illustrate, in my view if punitive damages are sought where the acts which created the situation involved were not unlawful when they occurred, then the statute must face the constitutional hurdles applicable to federal criminal statutes and would violate the Ex Post Facto clause. A good discussion of the constitutional issues raised by punitive damages is Wheeler, The Constitutional Case for Reforming Punitive Damages Procedures, 69 Va.L.Rev. 269 (1983).

> worthwhile economic activity. A third reason is that unless class actions are to be required, permitting punitive damages to be imposed on activities regulated under RCRA might result in overcompensation of a few claimants and their lawyers to the detriment of many equally worthy claimants.75/

F. Financial Responsibility Requirements

There are other important policy issues regarding the present Superfund Act which are the subject of an ongoing policy debate. For example, the Superfund Act contains mandatory financial responsibility requirements as part of its scheme of statutory liability for hazardous waste releases. These regulations are to go into effect no earlier than December 11, 1988. The Act attempts to restrict the contractual freedom of insurers providing coverage by providing for direct actions against them.76/ The Act also "nullifies policy terms and conditions essential to assuring policyholder compliance with applicable laws and regulations and contractual undertakings by providing that insurers may not invoke these terms and conditions as defenses to payment."77/ These and other requirements could make liability insurance "extremely difficult to obtain

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75/ Superfund § 301(e) Report, Part 1, pp. 322-323.

76/ 42 U.S.C. § 9608(c).

77/ Cheek, supra, at 171.

and terribly costly, particularly if the liability concepts in CERCLA are retained."78/

Insurers are not likely to submit voluntarily to a regulatory requirement that they compensate victims of their insured regardless of the intentional or grossly negligent nature of the latter's behavior.79/ By apparently trying to make an insurer's liability essentially coextensive with those of his insured, the Superfund Act approaches national insurance regulation, an area that has long been left to the states.80/ Insurers' resistance to national regulation generally, to interference with insurance contractual relationships, and to the Superfund Act's liability scheme with its potential for unlimited exposure could "have a dramatic impact on both the availability and affordability of insurance protection . . ."81/ This impact would undermine the cost spreading assumptions upon which the Superfund Act's liability/financial responsibility system is based.

78/ Id., at 174.

79/ Abraham, Cost Internalization, Insurance, and Toxic Tort Compensation Funds, 2 Va.J.Nat.Resources L. 123, 146-147 (1982).

80/ See Prybutok, Federalism Without Washington: Insurance Regulatory Environment in the United States 12 Publius J. Federalism 79 (1982).

81/ Cheek, supra, at 163.

I summarized this issue during the Superfund § 301(e) deliberations as follows:

> Most courts and commentators have not believed that the "deep pocket" was the sine qua non of social desirability and that having found the pocket ends the matter. Rather they have recognized that effects, both primary and secondary, on society, as well as ideals of fair treatment for individuals, have to be brought on to the balance. Thus, to me retroactive imposition of costs on an industry may be socially desirable if those costs can be spread to one portion of society through insurance (other insureds) and/or another through increased prices (consumers). Once a strict liability system, such as workmen's compensation, respondeat superior, etc., emerges from a successful transition phase, that is what normally occurs. But I want to be relatively certain that cost spreading is what will in fact occur, before I sign off on initiating such a process. Likewise, I want to know the probable social costs of the transition itself, its probable duration, and the likelihood of its successful termination.82/

The history of the Superfund Act's implementation since December 11, 1980, suggests that Congress did not consider the likelihood of cost spreading or the fairness of its new liability regime during the hurried lame duck session in which it hastily drafted the provisions of the Act.

82/ Letter of March 10, 1982, Superfund § 301(e) Report, Part 2, pp. 5-6 (Comments to the Appendices).

G. Tax Equity

Another important problem that has arisen in the implementation of the Superfund Act which also has obvious import for any victim compensation funding scheme is tax equity. The problem exists whether or not there is subrogation by a compensation fund against potentially responsible parties for claims paid. Fairness requires that those who provide the "up front" money for a fund should be roughly the same persons who are the potential subrogees. The issue is, if anything, more important where identifiable responsible parties are non-existent or judgment-proof or where there is no subrogation. In these cases, the ultimate incidence of a claim falls on the special taxpayers collectively.

To the extent that the special taxpayers, even collectively, pay costs largely attributable to other causes of disease (*e.g.*, a claimant's own habits, or his exposures to hazardous substances generated by non-taxpaying industries), the system distorts the marketplace for the products or services of the taxpaying industries and in effect forces their consumers to subsidize persons injured in whole or in part by other products or causes.83/

83/ *See* generally Klemme, *The Enterprise Liability Theory of Torts*, 47 U.Colo.L.Rev. 153, 156-165 (1976).

The Superfund Act's taxing scheme basically assumes that the oil and chemical industries are collectively responsible for the hazardous waste problem sites serious enough to require disbursements from the Superfund to finance remedial actions. EPA's priority lists of sites requiring cleanup, however, have included many sites which have absolutely no connection with either industry. For example, some western states have listed abandoned coal mines as sites. The chemical industry persuaded EPA to mitigate this unfair situation by considering "a Fund financed cleanup of [mining] sites only after all available enforcement efforts fail."84/ The basic problem, however, is that the "wrong" industry in these cases is being taxed.

Furthermore, there is some indication that the potential for abuse of special funds is greater than where a government program is funded by general tax revenues. Periodic scrutiny incident to the annual budget and appropriations process may be less rigorous, based on recent experience with the Federal Black Lung Benefits program and the New Jersey Spill Compensation Fund.85/ Former OMB official Robert G. Penner, now

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84/ EPA Guidance Memorandum on Establishing National Priorities List Under Superfund (Dated June 26, 1982), 13 Envir.Rep. (BNA) 339, 341 (Jul. 2, 1982).

85/ See Lopatto, Amendments Fight Excesses of Black Lung Statute, Legal Times (Mar. 22, 1982); N.J. Auditors Question

(footnote continued)

at the American Enterprise Institute, recently concluded, "the real problem is the lack of careful scrutiny of these things."86/ Funding a Federal administrative program for providing compensation to persons injured by hazardous wastes by an "add on" tax on the oil and chemical industries without knowing what impact the present tax is having on their ability to compete in international trade or what incidence the additional tax would have would be irresponsible. As I said in my Separate Statement in the Superfund Report:

> Based on the information available to the Study Group -- or, more accurately, the lack of information -- I cannot join in the Group's recommendation that the Tier One System be funded by a special tax on the chemical and oil industries, and perhaps other selected industries, rather than from general tax revenues. The Study Group knows virtually nothing about the probable magnitude of direct costs of a Tier One system. Those costs would include funds paid to claimants and administrative costs. Potential claims would include (a) those attributable to exposures to wastes disposed of prior to

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(footnote continued)

Spill Fund Management Controls, Hazardous Waste Lit.Rep. 2,444 (May, 18, 1982). Effective Date of New York Bottle Law Delayed by Measure Approved by Governor, 14 Envir.Rep. (BNA) 242 (June 6, 1983) (includes report on May 13 audit report by N.J. state legislature's Division of State Auditing on N.J. Spill Compensation Fund).

86/ Hershey, U.S. Insurance: Critics Grow, 131 N.Y.Times (Jun. 8, 1982), at 1, col. 2; 6, col. 1.

the enactment of any Tier One system and (b) those resulting from exposures to wastes disposed of after that date. The special tax the Study Group proposes would have to fund the total costs of "pre-enactment" disposal based claims.

It is reasonable to suppose that most of the pre-enactment activities involving waste transportation or disposal were not licensed under RCRA, while virtually all of the post-enactment activities will be licensed. Little is known about the actual nature, extent and location of hazardous wastes disposed of in the past across this country. Also, virtually no information is available on the nature and extent of any personal injuries or property damage such wastes may have caused. Turning to claims based on future activities, the potential costs of a Tier One system are also equally unpredictable. It is reasonable to suppose that the frequency of claims based on future disposal is more controllable because of the much greater care RCRA licensing will require. But there is always the problem that things viewed as harmless from today's knowledge may prove hazardous from tomorrow's hindsight. The only way to guard totally against the dangers of the currently unforeseeable consequences of economic activities is not to undertake them. But I do not see now anyone could seriously advocate society's prohibiting all activity that has not been proven to be "risk free." The economic and political consequences of imposing a "risk free" standard of care on all economic activity would literally wreck our society.

The Parkinson's law of bureaucratic programs insures that claims will be made and money spent under any government compensation system. If in such a system cancers which have multiple actual causes are presumed by law to have been caused solely or primarily by exposure to hazardous wastes, the potential for abuse is apparent. Thus while we could be confronting potential claims that aggregate only several millions, these claims might soar into billions. Indeed, any dollar estimates of the costs of a Tier One system made in the near future may prove as erroneous as those made for the Black Lung Fund when it was amended in 1977. The principal incidence of these unknown costs would fall, through the Tier One tax, on the chemical and oil industries. We have no idea what the economic effects would be on these industries and our economy.

In contrast, we know that our American economy is part of a world-wide economy, that many American industries are now being severely challenged by foreign producers and that our economy is in the midst of a severe recession. The day-to-day livelihood, health and security of our people depend upon the continuing ability of our domestic industries to compete successfully with foreign producers. This reality leads me to conclude that proponents of funding a "Tier One" system through increases in the current Superfund Act tax on specific American industries should have the heavy burden of proving that its incidence would not impede the ability of our domestic producers to compete with foreign producers in our own and foreign markets. If the ultimate result of this Group's proposals and other efforts to internalize the social costs of activities that produce hazardous wastes is to impair substantially that ability of our domestic industries to compete with foreign producers, then not only would these

> efforts at internalization fail, but also other direct and indirect external social costs would be visited on other citizens. Those external costs could substantially exceed the external costs now being borne by others because of hazardous wastes disposal.
>
> In summary, we must be careful in righting wrongs flowing from incomplete internalization of the social costs involved in the generation and disposal of hazardous wastes not to go overboard and impose a whole new set of non-internalized social costs -- loss of trade, unemployment the disappearance of useful products -- that are equally capriciously and unfairly imposed on other segments of our society. Two wrongs can not make a right.[87]

H. Intergovernmental Relations

An additional reason for the delays in implementing the Superfund program, which has import for any victim compensation plan, has been friction between the federal and state levels of government created by the hierarchical model of federalism upon which the intergovernmental relations in the Superfund program is based.

Interestingly, the Superfund Act's inter-governmental relations were a very controversial aspect of the debate over the Act in the House of Representatives during 1980.

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[87] Superfund § 301(e) Report, Part 1, pp. 311-314. The analysis of these effects might be an appropriate role for the Congressional Budget Office.

Congressman Stockman led the critics of this aspect of the legislation:

> We have an open-ended enabling statute that makes EPA the czar, and I say this without hyperbole -- makes EPA literally the czar over every hazardous waste site in the entire country, and that includes every landfill, every waste site, every junkyard, every leaching pit, every industrial lagoon on the back of an industrial property, every municipal dump, even every backyard rubbish pile that has been accummulated anywhere in the country . . . The EPA has plenary power, and there is absolutely no guidance as to where all these costly activities to clean up hazardous waste will be triggered.88/

The House bill upon which Congressman Stockman was commenting (H.R. 7020) is similar in most aspects of its intergovernmental operation to the Act which passed. Essentially, that Act authorizes the President to undertake any "response measure consistent with the national contingency plan [which he] deems necessary to protect the public health or welfare or the environment."89/ However, it requires that the President consult with the state and obtain numerous state commitments before taking action.90/ The state role is that of order-taker, not that of a partner.

88/ 126 Cong. Rec. H9439 (Sept. 23, 1980) (remarks of Congressman Stockman).

89/ 42 U.S.C. § 9604 (a)(1).

90/ 42 U.S.C. § 9604(c).

Congressman Stockman's proposed alternative to the broad delegation of authority to EPA evident in the Superfund proposals was a grant program for the states to provide money to clean up inactive waste sites, distributed mainly on the basis of a formula tied to population and industrial production. Congressman Florio, in defense of his H.R. 7020 and in opposition to Stockman's alternative, stated:

> EPA, under this proposal would provide formula grants to states with approved plans, after we went through all of the time necessary to get those plans formed . . . The arguments against the substitute are fairly obvious: Time and delay. Time is of the essence in many of these instances. The need is right now. I can point to some 397 sites . . . EPA is already involved and has identified some 397 specific sites that they are at work on. So we do not have the time to develop these programs . . . Many states, quite frankly, are not interested and are not capable of going forward and they do not want to. They want to defer to the Federal Government . . .[91]

When the Senate compromise bill reached the House in December, Congressman Dannemeyer briefly referred in floor debate to the defeated Stockman proposal:

> If there were no other way to attack the hazardous waste problem except through EPA, perhaps a case could still be made for this bill. After all, no one is arguing that the waste sites should not be cleaned up. But, to assume that only EPA can do the job, or do it best and quickest as this bill assumes,

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[91] 126 Cong. Rec. H9441 (daily ed. Sept. 23, 1982).

> flies in the face of historical experience. Since when did a Federal regulatory agency ever move quickly on a problem, to wit RCRA, or solve it efficaciously, to wit -- air and water pollution . . . . [I]t may be that state government would be able to handle the problem, which suggests that the block grant to states approach, as suggested by our distinguished colleague from Michigan (Mr. Stockman) would be preferable to this means.92/

Congressman Rudd also praised the Stockman alternative:

> A commendable alternative approach offered by the gentleman from Michigan (Mr. Stockman) . . . would have provided a block grant to the states. By this method, the states would be able to set up procedures to deal with the hazardous waste sites within their individual borders according to the priorities they determine . . . . Rather, this bill will perpetuate a Washington based "solution" administered through the Environmental Protection Agency . . .93/

In light of the states' experience with the practical operation (or, more accurately, general paralysis) over the past months, Congressman Stockman's block grant alternative to the present Superfund dole system merits reconsideration. Unfortunately, Congressman Florio's hope that EPA would act expeditiously on cleanup has not materialized.

92/ 126 Cong. Rec. H11,797 (daily ed. Dec. 3, 1980).

93/ 126 Cong. Rec. H11,802 (daily ed. Dec. 3, 1980).

In our Superfund Report, spurred on by the dissatisfaction of the states with the way the present Superfund had been administered as expressed eloquently by our three state attorneys general, we did not suggest that the federal administrative compensation scheme follow the pattern of that Act. We turned instead to the Clean Water Act for a pattern of federal-state cooperation that has worked well in the past and is working well at present.[94] Should Congress choose to go forward with any legislation in this area, it would do well to heed that advice.

III. PENDING LEGISLATION

There are many hazardous substance compensation proposals now pending in Congress.[95] Most of the proposals seek to expand the Superfund Act's liability regime to provide for coverage of personal injuries. Congressman John J. LaFalce's H.R. 2330 and H.R. 2482 are virtually identical to his H.R. 7300 of the last Congress, except that the federal agency designated to administer his compensation system has shifted from EPA to HHS.

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[94] Superfund § 301(e) Report, Part 1, pp. 252-254, 307, 328-329 n.6.

[95] The best comprehensive overview of legislative proposals in the trade or legal press is Ember, Legal Remedies for Toxic Victims Begin Taking Shape, 61 Chem. & Eng. News 11 (Mar. 28, 1983).

Congressman James J. Florio of New Jersey and Edward J. Markey of Massachusetts have introduced a RCRA amendment, H.R. 2582. In the Senate, the "Superfund" proposals include S.917 and S.945, Senator Robert T. Stafford's and Senator George J. Mitchell's rework of the provisions of S.1480 which were subsequently deleted from the Senate Bill in the 1980 compromise that permitted enactment of the Superfund Act. Senator Mitchell has also introduced S.946, which implements some of the Superfund § 301(e) Report's recommendations but departs from them in several important respects.

These proposals are being widely discussed. For example, the Center for Environmental Management and Inside EPA Weekly Report conducted a briefing on "victim compensation" in April. In May, the ABA's Standing Committee on Environmental Law devoted its annual conference at Airlie House to an analysis of these proposals. More important, the Reagan Administration has taken the matter seriously enough to create a major interagency task force, now at the White House Cabinet Council on Legal Policy, to examine a number of victim compensation options, involving representatives from the Departments of Commerce, Labor, Justice, EPA, HHS and OMB. The Chemical Manufacturers Association, the National Association of Manufacturers, the Business Roundtable, the American Petroleum Institute, the Edison Electric Institute, the American Paper

Institute/National Forest Products Association and various bar groups have organized studies of the issues involved in these proposals. One of the most important bar efforts now underway is by the Ad Hoc Committee on Tort Law Reform, of which, as I indicated earlier, I am a member, of the ABA's Section of Corporation, Banking and Business Law.[96]

The presently pending "Superfund" or RCRA proposals are part, and perhaps only a small part, of the overall Congressional debate concerning toxic exposure compensation. Congressman George Miller's bill in the 97th Congress, H.R. 5735, has been redrafted in the area of occupational disease compensation. The new bill, H.R. 3175, is designed in large measure to replace asbestos tort litigation with an administrative compensation plan. The Asbestos Compensation Coalition, an industry group, has developed its own occupational disease compensation plan. South Carolina's senators have introduced a brown lung bill.[97] Representative Thomas A. Daschle and many others have introduced bills to compensate those who were exposed to Agent Orange in the military.[98] Senator Robert W. Kasten, Jr.'s, industry-backed federal product liability reform legislation, S. 44, which the Commerce Committee reported in the 97th Congress, has already been the subject of hearings in the new Congress.99/ The oil spill compensation plans which have been debated over the past several Congresses have re-emerged.100/ Radiation exposure is the subject of Senator Orrin G. Hatch's and Representative Paul Simon's bills.101/ Rep. Albert Gore, Jr., has been holding subcommittee hearings on toxic exposure compensation in the House Science & Technology Committee. Rep. Don Albosta has asked the Congressional Research Service to examine toxic exposure compensation legal issues and their ramifications. Congress' Office of Technology Assessment recently completed a major study, which recommended "programs to provide assurance that in the event of damages to health or the environment, or indirect economic effects, injured parties will be able to obtain equitable compensation expeditiously."102/

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[96] See, e.g., Young, "Legal Group to Eye Liability Laws" in the July 19, 1983, issue of The Journal of Commerce.

[97] S.1155, 98th Cong., 1st Sess. (1983).

[98] H.R. 330, 98th Cong., 1st Sess. (1983) (Roe); H.R. 1135, 98th Cong., 1st Sess. (1983) (Downey); H.R. 1382, 98th Cong., 1st Sess. (1983) (Downey); H.R. 1961, 98th Cong., 1st Sess.

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(1983) (Daschle); H.R. 2017, 98th Cong., 1st Sess. (1983) (Daschle); S.374, 98th Cong., 1st Sess. (1983) (Specter); S.786, 98th Cong., 1st Sess. (1983) (Pressler).

99/ See S.2631, 97th Cong., 2d. Sess. (1982); S.Rep.No. 97-670, 97th Cong., 2d. Sess. (1982).

100/ H.R. 2115, 98th Cong., 1st Sess. (1983); H.R. 2222, 98th Cong., 1st Sess. (1983); H.R. 2368, 98th Cong., 1st Sess. (1983); S.976, 98th Cong., 1st Sess. (1983).

101/ S. 921, 98th Cong., 1st Sess. (1983); H.R. 1914, 98th Cong., 1st Sess. (1983).

102/ Congress of the United States, Office of Technology Assessment, Technologies and Management Strategies for Hazardous Waste Control, Summary (Mar. 1983), p. 32.

The lame duck Congress last winter enacted an "orphan drug" act containing a provision requiring HHS to develop tables relating radiation exposure levels to the probabilities of diseases.103/ These tables may be intended to serve as the basis for legislation in the radiation compensation area, perhaps similar to Senator Hatch's or Representative Dan Marriott's tort claims procedure for cancer allegedly related to nuclear fallout.104/

I will not attempt to describe comprehensively each of these toxic tort proposals. But I will instead discuss the key details of the "Superfund" and "RCRA" compensation proposals now pending in the House and how they might affect the existing Superfund Act problems I discussed earlier.

A. H.R. 2330 and H.R. 2482 -- The LaFalce Administrative Compensation Plan.

Congressman John J. LaFalce's toxic injury compensation bills, H.R. 2330 and H.R. 2482, are virtually identical to the bill he introduced when the Superfund § 301(e) Report was released late in the second session of the 97th Congress.105/

103/ Pub.L. No. 97-414, § 7(b) (1983).

104/ S.921, 98th Cong., 1st Sess. (1983); H.R. 6052, 97th Cong., 2d. Sess. (1982).

105/ *Compare* H.R. 2330 and H.R. 2482, 98th Cong., 1st Sess. (1983) *with* H.R. 7300, 97th Cong., 2d. Sess. (1982). The only

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There are only two changes from that earlier legislation. First, the federal administration of the proposed "victim compensation" plan has been changed from EPA to the Department of Health & Human Services.106/ The second change is: the new bill requires that a defendant "fail to establish . . . that . . . [a] release was not the result of a failure of the defendant to exercise due care" in order to make out a defense to liability under the Act.107/ This was a change from the requirement in the earlier bill that the defendant must prove that a "release was not the result of a failure of the defendant to exercise due care" to avoid liability.108/

Congressman LaFalce's proposal is, except for the statute of limitations change which distinguishes H.R. 2482 from both H.R. 2330 and H.R. 7300, identical to H.R. 7300 in the last Congress.109/ As such, unlike S.946, Senator Mitchell's

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difference between H.R. 2330 and H.R. 2482 is that H.R. 2330 starts the running of the statute of limitations upon discovery of a disease, and H.R. 2482 starts the running only upon discovery of a disease and its cause. H.R. 7300 was like H.R. 2330 in this respect.

106/ Compare H.R. 2330, 98th Cong., 1st Sess. §§ 3(3), 4(7), 102(b)(7), 201, 301 (1983) with H.R. 7300, 97th Cong., 2nd Sess. §§ 3(3), 4(7), 102(b)(7), 201, 301 (1982).

107/ H.R. 2330, 98th Cong., 1st Sess. § 101(c)(3)(B) (1983) (emphasis added).

108/ H.R. 7300, 97th Cong., 2d Sess. § 102(c)(2)(1982).

109/ See 127 Cong.Rec. H.8490 (daily ed. Oct. 1, 1982) (Cong. LaFalce's remarks noting that H.R. 7300 was drafted "over 1 year ago.").

"301(e)" bill, it has no direct relationship to the Superfund Study Group's recommendations. Although there is some coincidental similarity between the Study Group's recommendations and LaFalce's bill in the creation of an administrative compensation remedy to supplement remedies in the courts, H.R. 2482's approach goes beyond the Superfund Study Group's recommendations in many respects. More important, it creates the federal cause of action for pollution-caused injuries and illnesses which the Study Group unanimously rejected. While the bill creates a liability section outside the Superfund Act, unlike Senator Stafford's and Senator Mitchell's approaches, the identified responsible parties under LaFalce's bill are virtually identical to those in the Superfund Act.110/ They are the owner and operators of a waste site, transporters, and those who arrange for disposal or transportation.

The federal cause of action that would be created includes a broad presumption of causation for a plaintiff who shows that his exposure was "at such a level and for such duration as would be reasonably likely to cause or significantly contribute to the class or type of injury, illness, or damage allegedly suffered . . ."111/ An extremely broad variety of

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110/ Compare H.R. 2330, 98th Cong., 1st Sess. § 101(a) (1983) with CERCLA §107(a), 42 U.S.C. § 9607(a).

111/ H.R. 2330, 98th Cong., 1st Sess. § 102(a)(1) (1983).

evidence is declared to be "relevant" to a causation determination, similar to provisions in S.1480 and Senator Stafford's S.917.[112] The presumptions in these bills go considerably beyond the presumption the Study Group recommended, although I think that our experience with the Black Lung legislation proves that the statutory presumption approach is unwise no matter how narrowly such a statutory presumption is drafted.

Under the LaFalce bill, jurisdiction to make awards would exist "in any court of competent jurisdiction in any State" in addition to the federal courts.[113] It also contains an unusual state law "preemption" provision intended to force state workmen's compensation boards to award claims based on the federal statute's eligibility criteria. For example, one provision states:

> In any administrative or judicial proceeding under a State's workers compensation law, the presumption of cause provided by section 102(a)(1) shall constitute the law of such State.[114]

This provision is premised on the assumption that Congress can constitutionally rewrite state law where it conflicts with federal policies rather than to write a federal law which would then displace state law through traditional preemption. In my view, this approach may be constitutionally defective if anything remains of National League of Cities v. Usery [115] after the Supreme Court's recent opinions in FERC v. Mississippi[116] and EEOC v. Wyoming.[117] I should add that the Superfund Study Group considered this approach but rejected it on policy as well as legal grounds.[118]

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[112] Compare H.R. 2330, 98th Cong., 1st Sess. § 102(b) (1983) with S.917, 98th Cong., 1st Sess § 5(a) (1983) (adding CERCLA § 107(c)(2)).

[113] H.R. 2330, 98th Cong., 1st Sess. § 103(a) (1983).

[114] H.R. 2330, 98th Cong., 1st Sess. § 104(b) (1983).

Turning to the bill's administrative compensation plan (which would exist in addition to the federal cause of action), it is completely federal, located in a federal agency board funded by federal taxes with federal judicial review of agency decisions. Although the locus of the plan's administration now would be HHS, rather than EPA, the federal agency's duties remain the same: (1) certify "releases" of hazardous substances, (2) certify "victims" of exposure to hazardous substances, (3) pay medical and economic loss benefits to certified "victims," and (4) pursue allegedly responsible parties in subrogation actions. The plan would be funded by a Toxic Victim Compensation Trust Fund which draws funds from the "unobligated balance" in the "Superfund" "at the time any benefit is awarded to a victim of a release."119/ The Victims Fund is to be reimbursed by subrogation actions and is also to get loans from the Treasury. The bill authorizes appropriations to Treasury to make these loans.120/ Thus, unlike the 301(e) study group's recommendations, the LaFalce bill establishes no new tax on industry to fund the toxic victims compensation program.121/

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[115] 426 U.S. 833 (1976).

[116] 456 U.S. 742 (1982).

[117] ___ U.S. ___, 103 S.Ct. 1054, 75 L.Ed.2d 18 (1983).

[118] Whether constitutional or not, this "indirect" preemption approach is contrary to the fundamental principles of "our federalism" as I understand them. See note 6 to my separate comments in the Section 301(e) Report at pages 328-329.

B. H.R. 2582 -- The Markey/Florio Bill

Congressman Markey has stated that "this legislation is modeled -- with some changes -- after a proposal resulting from a thorough study of victim compensation mandated in the 1980 Superfund law."122/ But those changes are rather significant. The major one is that the bill creates the federal cause of

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119/ H.R. 2330, 98th Cong., 1st Sess. § 402 (1983).

120/ *See* H.R. 2330, 98th Cong., 1st Sess. §§ 402-405 (1983).

121/ Toxic Victims Compensation, A Speech by the Honorable John J. LaFalce before the Center for Energy and Environmental Management, April 5, 1983, Capital Hilton, Washington, D.C., p. 9.

122/ Statement of Rep. Edward J. Markey (Mar. 24, 1983); H.R. 2582, 98th Cong., 1st. Sess. (1983) (jointly referred to Ways and Means and Energy and Commerce committees).

action which the Superfund Study Group rejected. The federal cause of action explicitly provides for strict, joint and several liability, and damages for pain and suffering. Moreover, it would supplement rather than displace duplicative or conflicting state law remedies.

Turning to the administrative plan proposed in the bill, although it contains some problems which I will discuss later, it is a useful step in the right direction. The proposed administrative compensation plan would permit EPA to award compensation for physical injuries and limited economic loss where a person has "suffered a physical injury or illness caused by exposure to a hazardous substance."123/

What distinguishes this bill from other pending bills in the House and Senate are the limitations which have been built into its "Two Tier" approach. Many of these limitations, which are generally not included in the other toxic injury compensation bills, were also included in the Superfund § 301(e) Report's proposed plan. For example, the liability system is said not to be imposed retroactively in the sense of reimbursement of the government fund by responsible parties.124/

123/ H.R. 2582, 98th Cong., 1st Sess. § 9012(a) (1983).

124/ Nevertheless, the system may in practice operate retroactively unless the provision is rewritten so as to exclude application of the new liability on the basis of activities of the alleged defendant rather than subsequent exposure of the plaintiff.

While a kind of joint and several liability is imposed, liability generally is imposed only for exposures occurring while one has possession or control over a hazardous substance or if one owns or operates a site at which a person is exposed.

The liability is also not imposed on an owner or operator who did not know or reasonably should not have known that a substance was located at a facility when he acquired the site or in which no action or inaction of such a person caused or contributed to an exposure. If an exposure results from a federally permitted release, the defendant is not liable absent, at a minimum, negligence. Further, the results of an administrative proceeding may not be introduced into evidence in any court action. There is also a collateral source offset for any social insurance program such as medicare and medicaid and for private insurance in both systems. Thus, both the administrative compensation and federal cause of action systems are "excess liability" systems only. In addition, any person covered by workers' compensation is excluded completely from recovery under either system.

All of these limitations were discussed in the Superfund 301(e) Report, and most were recommended. Unfortunately, H.R. 2582 also picks up some of the objectionable recommendations of that Report, including a statutory presumption of causation in the administrative compensation system. As the

Study Group recommended, however, the presumption is not applied in the federal cause of action, although health effect documents prepared for use in connection with such presumptions may be introduced in a court action by any party. The administrative system is funded mostly by oil and petrochemical taxes and to a lesser extent by a waste-end tax as is the Superfund Act's "post-closure" fund.

I believe that H.R. 2582 in its present form has three major flaws. For the reasons I gave in my separate statement in the Superfund Report, I believe the special tax and the statutory presumptions provisions are unsound.[125] And like all of my colleagues in the Study Group, I believe it unwise to superimpose a duplicative federal cause of action on to existing state tort law.

One of the reasons I, as an individual member of the Superfund Study Group, was not willing to recommend a federal cause of action in the tort area is that I believe that the present tort system, as distinguished from an administrative system, is enormously inefficient. I have no way of knowing with exactness the proportion of the billions of dollars that are now being spent in the form of compensation payments, insurance premiums or legal fees, or support of the court system, but I have the idea that only $1 out of every $3 goes to a victim. The other $2 go to the system. Therefore, I think an administrative system which can cut down on those transactional costs is a desirable thing. But going forward with an add-on federal system that just builds on top of it all the problems that we have with the present state systems is no real answer.

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[125] Superfund § 301(e) Report, Part 1, pp. 311-314 (special tax), pp. 314-319 (statutory presumption).

I would also like to point out that that is not the only reason I was opposed to a federal cause of action. I also think it was not the only reason that led us as a group not to recommend that you consider a federal tort remedy. Intellectually, some of us recognized that if we were to recommend such an alternative to you, we would have to face up to, and make recommendations to you on, three important aspects of such a system. First, we would have to tackle the transactional costs issue. That is legal fees. That is the contingent fee. Given the composition of our group, we knew that we could never unite in a recommendation to you to abolish contingent fees and substitute court-approved payments. Personally, however, I think if you are going to enhance substantially the likelihood of recovery, the case for contingent fees goes down and the case for court-approved lawyers' fees based on reasonable hourly rates goes up substantially.

Second, we would have had to address and make recommendations to you on subjectively determined damages. By "subjectively determined," I mean damages such as pain and suffering or punitive damages, which really cannot be objectively quantified, and therefore the award of those damages lies largely with the prejudices or inclinations of the person awarding them.

Third, we would need to face up to the issue of preemption. If Congress were to create a federal cause of action, it seems to me you should make it the exclusive remedy and preempt contrary state law.

If Congress wants to enact a federal cause of action provision and it faces up to these key issues, then a revised H.R. 2582 could be the vehicle for sound legislation in this area. Suppose the strict liability cause of action linked to control over a waste, rather than duplicating state tort law, provided the exclusive tort remedy. There would be no costly cross actions in cases where workers' compensation applied, no actions where public or private insurance covered the costs of an injury, no contingent liability in situations where exposure occurred after a hazardous substance has left one's control, no retroactive application of the strict liability system. In addition, liability for hazardous substances exposures would be uniform throughout the United States with access to the federal courts eliminating conflict of law, choice of law, and many jurisdictional questions. This would end the forum-shopping which presently goes on in many toxic tort cases.

Suppose further that the administrative compensation system were limited to "orphan sites" for which no responsible party could be sued (like California's Hazardous Substance Compensation Account),[126] and that anyone claiming against the Fund would be barred from bringing a lawsuit against anyone for the same exposure. Then use of the fund and litigation would be properly limited. And suppose that the tax funding the plan was a fair tax on all the industries responsible for exposures or from general revenues.

With amendments carefully drafted along these lines, the Markey/Florio bill could provide for a two-tier toxic substance compensation approach which might be preferable to the present system.

Another legislative toxic substances proposal that merits serious attention is the recent state-level proposal of the North Carolina Governor's Waste Management Board.[127] In North Carolina at least, that Board felt that the need for any administrative compensation system had not been demonstrated. The

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[126] Cal. Health & Safety Code §§ 25300-25611 (1981).

[127] *See* Attachment C to this statement.

Board recommended, however, a new statutory prospective strict liability system in which generators and those in possession of a substance would be held liable for damages arising out of a statutorily defined hazardous waste occurrence. Generators could avoid liability upon proof that they had transferred the waste to a treatment, storage or disposal facility, with a manifest providing presumptive evidence of that transfer.128/ The North Carolina scheme is a more limited no-fault liability proposal than the Congressional proposals. The North Carolina board carefully assessed the Superfund § 301(e) recommendations and then adopted those recommendations in part, while rejecting some of the Report's other proposals.

C. H.R. 2478 and H.R. 2867 - Toxic Compensation through the Back Door.

Before I leave the hazardous substance injury compensation legislative proposals, I should mention some other proposals, which I alluded to earlier, which would attempt to create a federal hazardous substance exposure cause of action through a route other than one of these "victim compensation" bills. H.R. 2867, the RCRA reauthorization bill, would authorize citizens to sue for damages from hazardous waste disposal in

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128/ Michigan has already enacted a similar provision into law. Mich.Comp.Laws § 299.538.

Federal court by amending the citizen suit provision in RCRA. Former Assistant Attorney General Carol Dinkins has testified against this proposal because such counts would "unduly complicate, confuse, and delay" EPA and Justice enforcement efforts.129/ H.R. 2867 contains provisions allowing citizens to bring "enforcement" actions under an equivalent to RCRA's emergency hazard provision § 7003,130/ even where there are no alleged violations of a RCRA permit or any EPA order or regulation. In addition to making any overall EPA-Justice enforcement policy impossible for them to orchestrate, this provision could result in the transfer of much of the litigation that would otherwise take place in the state courts to the federal courts. In these "citizens suit" enforcement actions, the plaintiff is to be awarded attorneys' fees if he prevails.131/ Moreover, he could append his action for damages under state law to his RCRA suit under the doctrine of pendent jurisdiction.132/ Should he prevail on the pendent state claims, would

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129/ See "Justice Official Opposes Amendment to Allow Citizen Lawsuits Under RCRA," 13 Envir.Rep. (BNA) 2184 (Apr. 1, 1983).

130/ H.R. 2867, 98th Cong., 1st Sess. § 11(f) (1983); H.R. 2478, 98th Cong., 1st Sess. § 12(g) (1983) (amending RCRA § 7002).

131/ H.R. 2867, 98th Cong., 1st Sess. § 11(b) (1983); H.R. 2478, 98th Cong., 1st Sess. § 12(e) (1983).

132/ See United Mine Workers v. Gibbs, 383 U.S. 715 (1966); see H.R. Rep. 98-198 (Part 1), 98th Cong., 1st Sess. 118-119 (1983).

his lawyer be entitled to a contingent fee on top of his court awarded fee? Even if state law were not promising or against him, a plaintiff could avail himself of the proposed expansion of § 7002 to cover past acts and to subject past offsite waste generators to § 7002 "injunctions."133/ In addition to reversing U.S. v. Wade134/ and U.S. v. Waste Industries,135/ H.R. 2867 would apparently provide plaintiffs with a right to order a remedy, such as to pay for a cleanup on someone else's property. The debate over the existence of a private right of action under the present Superfund Act would, in large measure, be mooted, since plaintiffs could seek to order cleanups in advance without incurring any costs initially or concerning themselves with possible inconsistency with the national contingency plan.136/

A second "back door" toxic substance compensation proposal is also hidden in H.R. 2867. Its innocuous looking Section 11(c) states: "Section 7002(f) of such Act is amended by striking out 'section' and substituting 'Act.'"137/ This

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133/ Cf. H.R. 2867, 98th Cong., 1st Sess. § 11(a) (1983); H.R. 2478, 98th Cong., 1st Sess. § 12(b) (1983) (amending RCRA § 7003).

134/ 546 F.Supp. 785 (E.D.Pa. 1982).

135/ 556 F.Supp. 1301 (E.D.N.C. 1982).

136/ These are requirements for liability under CERCLA § 107(a), 42 U.S.C. § 9607(a).

137/ H.R. 2867, 98th Cong., 1st Sess. § 11(c) (1983); H.R. 2478, 98th Cong., 1st Sess. § 12(d) (1983).

amendment to RCRA's savings clause is intended to "create" or "revive" a federal common law of nuisance which has been held to be superseded, to the extent existent, by Congress' enactment of the Clean Water Act, the Clean Air Act and RCRA.[138]

I believe that this attempted "revival" approach is ill-conceived for the following reasons.[139]

The Founding Fathers denied the federal government the *parens patriae* police powers of the British Crown, carefully safeguarding that residuum of sovereignty to the states. The common law of tort and nuisance is part of those retained police powers. This is the "true" common law in that it is the continuum of the pre-American Revolution English "judge-made" law. It was this true common law to which Justice Brandeis referred in the landmark case of *Erie Railroad Co.* v. *Tompkins*: "Congress has no power to declare substantive rules of common law, whether they be local in nature or 'general,' be they commercial law or part of the law of torts."[140]

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[138] *City of Milwaukee v. Illinois*, 451 U.S. 304 (1981) (Clean Water Act); *New England Legal Foundation v. Costle*, 666 F.2d 30 (2d Cir. 1981) (Clean Air Act); *Connecticut v. Long Island Lighting Co.*, 535 F.Supp. 546 (E.D.N.Y. 1981) (Clean Air Act); *U.S. v. Kin-Buc, Inc.*, 532 F.Supp. 699 (D.N.J. 1981); *U.S. v. Waste Industries*, 18 ERC 1521, 1533-1534 (E.D.N.C. 1983) (RCRA and Superfund); *City of Philadelphia v. Stepan Chemical Co.*, 544 F.Supp. 1135 (E.D.Pa. 1982) (RCRA and Superfund); *U.S. v. Price*, 523 F.Supp. 1055 (D.N.J. 1981) *aff'd* 688 F.2d 204 (3d Cir. 1982) (RCRA and Superfund).

[139] *See* Freeman, *Back to Basics: State (the True) Common Law and the Constitution*, 2 Envtl. Forum 27 (July 1983).

[140] 304 U.S. 68, 78 (1937).

In Milwaukee I,141/ the Court did not abandon this fundamental concept that our federal government is a government of limited powers. Instead, it held that states had general access to the federal district courts to seek judicially-fashioned equitable remedies for interstate pollution where Congress has not by statute specified other federal forums. This right of access for federal judicial resolution of disputes between states which prior to the Constitution would have been an international controversy, was held to be an implicit quid pro quo for the original 13 states' ratification of the Constitution. This right was consistent with the express right accorded one state to sue another in the Supreme Court by Article III of the Constitution. Similarly, where Congress has not legislated the substantive standards which govern an interstate controversy between a state and another state (or person or entity outside the plaintiff state), the Court might consider state standards where they exist but the rule of decision necessarily is federal. These ad hoc, judicially-fashioned, equitable remedies for the states in the context of interstate pollution were referred to by the short-hand phrase "federal common law." But apart from this epigram, federal common law bore little relation to "state common law" other than that both were judicially defined rather than legislatively created.

141/ 406 U.S. 91 (1972).

In Milwaukee II,142/ the Court held that the Federal Water Pollution Control Act is now the fixed law which governs the rights of states regarding interstate water pollution controversies, leaving no room for judicial articulation of the "federal common law" to which the Milwaukee I court referred. In Middlesex County Sewerage Authority v. National Sea Clammers Association,143/ decided later in the Court's 1981 term, the Court held that no implied damages remedy exists under any federal law for violations of the Clean Water Act or the Marine Protection Research and Sanctuaries Act. The Court reasoned that because Congress had provided some express remedies in those Acts, "we are compelled to conclude that Congress provided precisely the remedies it considered appropriate."144/ Turning to the "federal common law" claim, the Court summarily dismissed it on the grounds that "federal common law" no longer existed, citing Milwaukee II.145/ Thus the question of whether private persons, as distinguished from states, ever had any standing to sue as plaintiffs under federal common law was moot. But in dicta on that issue the Court noted that the

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142/ 451 U.S. 304 (1981).

143/ 453 U.S. 1 (1981).

144/ 453 U.S. at 15.

145/ 453 U.S. at 21-22.

Court of Appeals in Sea Clammers had gone "considerably beyond [Milwaukee I], which involved purely prospective relief sought by a state plaintiff,"[146] in asserting that federal common law could be invoked by private plaintiffs in an action for damages. Many observers believe that, had the Court reached and decided the moot question, it would have held that no private right of action ever existed because federal common law exists only for states.

Those who advocate creation or "revivification" of federal common law in the area of federal environmental law appear to ignore the fundamental distinction between state common law (the "true" common law in the sense of the continuing legacy of English common law) and the more restricted federal common law (an implied right of states to protect themselves derived from their entry into the Union). Moreover, the amendments to the savings clauses of the environmental statutes which they propose probably would not have their intended effect. I believe that the Court would hold a congressional attempt to delegate the formulation of a federal cause of action for damages to the federal courts unconstitutional on one or more of three grounds. First, private persons have no standing to assert a right of action under federal common law. Second, a congressional attempt to ape the House of Lords by "reversing" the Supreme Court's two decisions as if the court were the merely the Court of Criminal Appeal would violate the cardinal Constitutional principle of separation of legislative and judicial powers. Third, the attempt would also fall under the newly invigorated doctrine of standardless delegation.147/ If Congress desires to create a federal remedy to allow private persons to recover damages, it can do so only by legislating an express private federal cause of action.

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[146] 453 U.S. at 10.

IV. A HUMBLE BEGINNING: FILLING SOME GAPS IN THE SUPERFUND ACT

I hope that this analysis of the various "Superfund" and "RCRA" hazardous substance injury compensation bills pending in the House against the background of our earlier Superfund Study Group deliberations will be be useful to you. I have also included as an attachment to this statement a table analyzing these and other pending bills.148/ Despite my criticisms of specific provisions in specific bills, I would like to emphasize that in my opinion substantial major reform is necessary in tort law generally, as well as in the laws applicable

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147/ *See*, *e.g.*, S.Rep. No. 97-284, 97th Cong., 1st Sess. 164-165 (1981).

148/ Attachment B.

to injuries from hazardous wastes. In my view, the greatest chance for long lasting tort law reform in the hazardous wastes and other critical areas like products liability, asbestos and airline accidents lies in carefully selected and restrained federal preemption by Congress. Reform in all of these areas should be consistent unless there are articulated and impelling reasons for exceptions. Moreover, the extent and substance of federal preemption should, in my opinion, involve a careful balancing of what is best for the economy, what is the least disruptive of existing legal obligations, what is the proper additional burden to put on our federal courts and what is consistent with our federalism.149/ There has been no effort to date in Congress to see how reform proposals in these various fields of tort law relate to each other. It is not surprising that the optimum these reforms should take remains somewhat elusive at this point. "Quick fix" solutions (such as the creation of duplicative judicially-defined remedies in H.R. 2867) or of a federal cause of action deliberately left ill-defined (such as Section 107 of the Superfund Act) are not appropriate Congressional responses to this complicated problem.

This does not mean, however, that Congress should do nothing now in this entire area. The time for action clearly

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149/ *See* my separate comments in the Section 301(e) Superfund Report at 307 and 328-329.

has arrived for it to correct some of the legislative mistakes made in 1980 in leaving many of the Superfund Act's cleanup liability provisions' most important features to so-called "common law development," that is to litigation with its associated unnecessary waste of both the government's and the private sector's resources. While Congress carefully considers meaningful long-term reforms of state tort law regarding identified hazardous substance injury problems such as asbestos and while it continues to search for the parameters of any hazardous waste exposure problem, it should move quickly to fill the gaps in the cleanup statute to prevent future exposure problems through cleanup now.

Let me suggest one modest example of such a gap filling, which would clarify the Superfund Act and make the cleanup program more workable. Congress would in my opinion substantially improve the Act by providing for explicit statutory joinder and apportionment rules for splitting the costs of remedial actions among liable defendants, as has been accomplished in the California and other state superfund laws. I have enclosed a first draft of such a "technical amendment" to Section 107 of the Superfund Act, along with an explanation, as Attachment E to this statement.

ATTACHMENTS TO TESTIMONY OF
GEORGE CLEMON FREEMAN, JR.

July 26, 1983

A. Superfund Section 301(e) Study Group Comments of George Clemon Freeman, Jr. (excerpts from pages 305-348 of the published report)

B. Toxic Injury Compensation Proposals -- Hazardous Waste Bills.

C. State of North Carolina Governor's Waste Management Board, Report and Recommendations on Liability Issues in the Area of Waste Management (January 1983).

D. Schmalz, _Superfunds and Tort Law Reforms: Are They Insurable?_ 38 Bus.Law. 175 (1982).

E. Comprehensive Environmental Response Compensation, and Liability Apportionment Act of 1983 (a proposed Superfund Act amendment).

ATTACHMENT A

COMMENTS OF
GEORGE CLEMON FREEMAN, JR. [1]

I agree with some but not all of the recommendations in the Report.[2] There are also several important matters on which we spent considerable time that are not dealt with adequately in the Report. I address these matters in this separate statement.

1. Judge Breitel's Separate Views

Before turning to the Report, I commend the thoughtful comments of the most distinguished member of our Study Commission, Judge Charles Breitel of New York. I agree with Part II of his statement and his Conclusion. While I also agree with his general observations in Part I, I would like to make two comments.

First, after much thought, I do not put as much distance between myself and the Report. I participated in the give and take of its formulation and suggested substantive points and language changes. Some were adopted; others were rejected. The final Report, like the product of most committees, is incomplete, sometimes inconsistent, and sometimes deliberately silent or obscure. These flaws are usually unavoidable in the elusive quest for consensus. Despite the Report's weaknesses, it focuses on the crucial legal issues in the area of the generation, transport and disposal of hazardous wastes. But I agree with Judge Breitel that these issues need to be resolved with additional views outside the legal profession. As he points out, they merit consideration by a much broader community, including economists, businessmen, political scientists, and medical and scientific experts. Thus I join in urging that before Congress or the States act on any of the recommendations of this Report, they need to hear from these other disciplines, particularly the economists.

Second, I do not regard our decision to limit our focus as a default. It was a practical solution to the otherwise impossible problems posed by our deficiencies in time, personnel and perspective. Moreover, the reasonableness of our early decision to confine ourselves to what was feasible should also be viewed in light of how the Study Group was authorized. Section 301(e) of the Superfund Act was part of a political compromise. It was a substitute for specific provisions in the Senate Bill that would have created a limited Federal plenary action in tort for persons injured by many toxic substances. Opponents of the Senate bill's provisions voiced a number of concerns. Those concerns were addressed in two ways in the enacted legislation. First, the final Superfund Act was limited to provisions for the cleanup of spills and disposal of hazardous wastes. Second, the federal tort action provisions were deleted and our Study was mandated.[3] The remaining concerns were reflected in the charge given our Study Group by Section 301(e).[4] Our study, limited as is, addresses those issues. And in this context, it is significant that none of us thought that creation of a federal tort remedy was necessary or desirable.[5] Instead, we recommend a more limited Federal social insurance scheme, which would be far less disruptive of the economy and existing legal obligations, less burdensome on the Federal courts and more consistent with our Federalism.[6]

At the same time, I agree with Judge Breitel that a much broader reexamination of all the aspects of our legal system governing liability for all toxic substances is needed. Indeed, I would extend it to encompass other facets of "tort" law that also are in ferment: products liability, occupational diseases, automobile accidents and airline accidents. There are Constitutional and legal principles that are common to all of these areas of the law. These principles, as well as their application or nonapplication in any particular area, merit serious review. Any inconsistencies among these fields should be rational and deliberate, rather than the result of political accident, as happens all too frequently. Finally, the composite incidence of all these laws on our society -- the economic, political and other social effects -- needs to be assessed to see where change is needed and which changes would actually be in the public interest.

2. The Report's "Tier One" Recommendations.

A. "Orphan" Sites. -- I join in the recommendation of a limited social insurance system to aid persons who have been injured in the past through the disposal of hazardous wastes at sites that are now "abandoned" or "orphaned." State common law and most present State statutes provide no adequate remedy to compensate victims of improper disposal of hazardous substances at "orphan" sites. Since it is reasonable to assume that most of these wastes originated in past industrial activities in interstate commerce, it is also reasonable to assume that the country has benefited generally from the economic activities that generated them. From hindsight, we can see that improper disposal of these wastes in the past was in part attributable to the failure of our State governments to use their police powers adequately to control hazardous waste disposal or of our Federal Government to do so through its commerce power. Society as a whole should share some of these costs by providing compensation to those injured for actual injuries to health and for loss of wages which have not been compensated by other means. Since we are dealing with past conduct, the government, either at the State or Federal level, affords the only practical means of providing this limited compensation.

Because of the probable interstate commerce origin of these wastes and the Federal Government's greater taxing resources, when we discussed this matter last fall it appeared reasonable to recommend that the Federal Government fund this program. But since then several States have enacted or are considering State laws somewhat similar to our Tier One recommendation. California, for example, did so on September 25, 1981.[7] New York reportedly considered such a law this year.[8] If a substantial number of the States in which most of these orphan sites are located adopt appropriate legislation by the time Congress considers legislation along these lines, the practical need for Federal legislation dealing with orphan sites may have disappeared. In any event, Congress should take careful note of the States' ongoing efforts and take advantage of their experiences under their new statutes.

B. Existing and New Sites. -- I also join in the recommendation for creation of a Federal Tier One System to aid persons injured by hazardous wastes at ongoing or new disposal sites because it may be regarded properly as an incidence of Federal regulation of these sites under the Resource Conservation and Recovery Act (RCRA). Equally important, our current State tort systems generally do not provide a satisfactory means for dealing with relatively small claims whatever their origin.[9] Except where there are a number of similar small valid claims arising from the same situation that may be consolidated into a single trial, the expense of litigation often precludes recovery. Where the injury arises through the deliberate or negligent action of others, leaving the loss totally on the victim is obviously unfair. Also public awareness of this general deficiency of the present tort system may strengthen public opposition to socially needed or desirable economic activities in which the generation, transportation or disposal of hazardous wastes is unavoidable. Thus the creation of a social insurance compensation system would not only be fairer to those who are injured from hazardous wastes through no fault of their own, but also could lead to greater public acceptance of RCRA licensed hazardous waste management facilities that may be essential to our long term economic prosperity.[10]

C. The Limited Extent of Our Knowledge. -- I wish to emphasize that while I support the general concept of a Tier One system, subject to the reservations expressed below, I do not do so because I believe that there are many persons who have been injured by exposure to hazardous wastes and not compensated from other sources. Contrary statements in the Report have no basis in the information available to us. Repeated requests by the Group to the Justice Department for relevant "hard" evidence currently available to it and to EPA as to the extent of this problem produced very little.[11] Our Study Group had no practical means of remedying this lack of information since we were not constituted to be a fact-finding body. The Report correctly indicates the Group had no alternative but to conduct its Study on the assumption that substantial health problems caused by hazardous wastes exist and will occur. But statements to the effect that "some more evidence of hazard has become available" reflect neither a finding by the Group nor my personal view.[12]

D. Funding of a "Tier One" System. -- Based on the information available to the Study Group -- or, more accurately, the lack of information -- I cannot join in the Group's recommendation that the Tier One System be funded by a special tax on the chemical and oil industries, and perhaps other selected industries, rather than from general tax revenues.[13] The Study Group knows virtually nothing about the probable magnitude of direct costs of a Tier One system. Those costs would include funds paid to claimants and administrative costs.[14] Potential claims would include (a) those attributable to exposures to wastes disposed of prior to the enactment of any Tier One system and (b) those resulting from exposures to wastes disposed of after that date. The special tax the Study Group proposes would have to fund the total costs of "pre-enactment" disposal based claims, while subrogation would fund some of post-enactment based claims.

It is reasonable to suppose that most of the pre-enactment activities involving waste transportation or disposal were not licensed under RCRA, while virtually all of the post-enactment activities will be licensed.[15] Little is known about the actual nature, extent and location of hazardous wastes disposed of in the past across this country.[16] Also, virtually no information is available on the nature and extent of any personal injuries or property damage such wastes may have caused.[17] Turning to claims based on future activities, the potential costs of a Tier One system are also equally unpredictable. It is reasonable to suppose that the frequency of claims based on future disposal is more controllable because of the much greater care RCRA licensing will require. But there is always the problem that things viewed as harmless from today's knowledge may prove hazardous from tomorrow's hindsight. The only way to guard totally against the dangers of the currently unforeseeable consequences of economic activities is not to undertake them. But I do not see how anyone could seriously advocate society's prohibiting all activity that has not been proven to be "risk free."[18] The economic and political consequences of imposing a "risk free" standard of care on all economic activity would literally wreck our society.

The Parkinson's Law of bureaucratic programs insures that claims will be made and money spent under any government compensation system. If in such a system cancers which have multiple actual causes are presumed by law to have been caused solely or primarily by exposure to hazardous wastes, the potential for abuse is apparent. Thus while we could be confronting potential claims that aggregate only several millions, these claims might soar into billions.[19] Indeed, any dollar estimates of the costs of a Tier One system made in the near future may prove as erroneous as those made for the Black Lung Fund when it was amended in 1977.[20] The principal incidence of these unknown costs would fall, through the Tier One tax, on the chemical and oil industries. We have no idea what the economic effects would be on these industries and our economy.

In contrast, we know that our American economy is part of a world-wide economy, that many American industries are now being severely challenged by foreign producers and that our economy is in the midst of a severe recession.[21] The day-to-day livelihood, health and the security of our people depend upon the continuing ability of our domestic industries to compete successfully with foreign producers. This reality leads me to conclude that proponents of funding a "Tier One" system through increases in the current Superfund Act tax on specific American industries should have the heavy burden of proving that its incidence would not impede the ability of our domestic producers to compete with foreign producers in our own and foreign markets. If the ultimate result of this Group's proposals and other efforts to internalize the social costs of activities that produce hazardous wastes is to impair substantially that ability of our domestic industries to compete with foreign producers, then not only would these efforts at internalization fail, but also other direct and indirect external social costs would be visited on other citizens.[22] Those external costs could substantially exceed the external costs now being borne by others because of hazardous wastes disposal.

In summary, we must be careful in righting wrongs flowing from incomplete internalization of the social costs involved in the generation and disposal of hazardous wastes not to go overboard and impose a whole new set of non-internalized social costs -- loss of trade, unemployment, the disappearance of useful products -- that are equally capriciously and unfairly imposed on other segments of our society. Two wrongs can not make a right.

E. Statutory Presumptions. -- While I join in the Report's recommendation for a system to create "Toxic Substance Documents", I cannot support the particular statutory presumptions recommended.[23] Good presumptions can play a useful role in a Tier One system, just as good presumptions are an important part of the existing common law. But the statutory presumptions proposed here are not good presumptions.

The principal purpose of presumptions in the first tier ought to be to facilitate expedited payments to "qualified", or "worthy", recipients. They should be persons who have been injured primarily because of the hazardous wastes. Two secondary purposes should be to ease the burden on the administrators of the fund and to hold down overall transactional costs.

However worthy these three objectives, presumptions should be employed only where they are a valid reflection (a shorthand summary) of what is generally true. Since absolute truth is elusive in the areas we have considered, we should settle for the second best -- the prevailing views of the medical and scientific experts. But that is far better than the third best -- legislative fiats that reflect policy biases as to who should win rather than what is most likely to be objectively determined truth. One of the major problems with the three recently repealed Black Lung Act statutory presumptions was that they did not meet this criterion of truth, and they well nigh-wrecked that program.[24] Throughout the Study Group's deliberations Judge Breitel continued to remind us that in the common law the strengths of presumptions vary, and they vary directly in proportion to the reliability of their perceived nexus to the truth.[25]

Diseases such as cancers may have multiple causes. If the Group's two statutory presumptions are intended to make exposure to hazardous wastes the principal legal cause of such injuries and thus to lessen the importance of evidence of other causes, they cannot pass muster for the broad array of hazardous materials and diseases envisioned in the proposal. While these presumptions may well be valid for the rare form of cancer that is closely correlated statistically to asbestos inhalation, the strength of that single causal linkage appears to be exceptional. Cancer may often be the result of multiple factors acting together: heredity, diet, radiation exposures, climate, as well as what we breathe, eat and touch. One of the major problems with the implicit single cause criterion in the now repealed Black Lung Act presumptions was that they ignored this reality. What is called for in formulating reasonable presumptions in these multiple cause areas is probability analysis.[26]

The farther a presumption departs from reality, the more arbitrary it becomes. And the more dubious presumptions that are built into a statutory scheme, the more unfair it becomes.[27] The Supreme Court once said:

> [R]es judicata renders white that which is black, and straight that which is crooked... No other evidence can afford strength to the presumption of truth it creates, and no argument can detract from its legal efficacy.[28]

That should not be said of any presumptions written into any statute enacted pursuant to this report's recommendations or subsequently adopted under the administrative scheme recommended.

Turning to the recommended Toxic Substance Documents, if they are the product of a rigorous rulemaking process where scientific and medical peer group review of supporting studies and their underlying data is possible, then they can substantially aid in expediting recoveries and reducing transactional costs. Thus, preparation of these Documents should be entrusted only to an impartial federal agency, as distinguished from a "mission oriented" agency. The agency should also have no connection with funding of the Tier One system. The best scientific and medical evidence available should be assembled and tested through fair procedures where all concerned could have access and opportunity to participate. This would require going beyond the present bare bones notice and comment provision of the current Administrative Procedure Act to the "hybrid type" procedures that Senate Bill S. 1080, which was passed on March 24, 1982 by 94 to 0 vote, would afford.[29]

Documents produced through such a hybrid rulemaking process would be roughly equivalent to a scientific or medical treatise or journal article. Compiling and sorting the complex evidence in a single, generic proceeding would obviate the need for repetitious marshalling of the same evidence in case by case processing of multiple claims.

There is precedent for such an effort to expedite reasoned decision making in the experience of the Nuclear Regulatory Commission with hybrid rulemaking in dealing with complex generic issues. In the early 1970's the licensing of nuclear powerplants came to a literal standstill because generic health and safety issues under the Atomic Energy Act and generic National Environmental Policy Act issues were being adjudicated over and over again in individual plant licensing cases. The Commission finally broke the licensing impasse by removing each of these issues from those multiple forums and resolving it in a single generic rulemaking. Most commentators and courts applauded this process.[30]

Any discussion of presumptions must also take into account the reality that presumptions formulated for a Tier One system will eventually find their way by analogy into the Tier Two tort system. The Report recognizes that application of the proposed statutory presumptions would be grossly unfair if applied in Tier Two and seeks to avert this result by declaring that the "carryover" should not occur.[31] Such declarations are, however, likely to prove as ineffective as King Canute's order to the tide not to rise. In our Group discussions, Judge Breitel pointed out that legislative determinations are a source of common law principles. They will continue to be so however we opine. I believe that the better remedy to the perceived danger of abuses in Tier Two lies rather in greater restraint in the formulation of the presumptions for Tier One. If there were no statutory presumptions for Tier One and the only presumptions that were formulated administratively were factually sound ones developed through the deliberative process discussed above, it would be difficult to justify their rejection on either tier. There is little social merit in requiring the same factual matters to be relitigated time and time again.

F. Strict Liability. -- Judge Breitel in his comments points out that the analyses of "strict liability" adopted in the Report are flawed.[32] The Report's view is at the outer fringes, rather than in the mainstream, of current thinking on this aspect of the common law both in the courts and academia.[33] I disassociate myself from this aspect of the Report and instead concur in Judge Breitel's views.

G. Retroactivity. -- One topic that recurred again and again in our Group discussions was "retroactivity." Some of this discussion touched on the questions of Constitutionality, which an Appendix to the Report discusses,[34] but the bulk of it centered on questions of policy, which the Report does not discuss in sufficient detail.[35] This glossing over should not obscure one of the Study Group's most important decisions: the recommendation that subrogation for claims paid out of the Tier One system not apply to wastes disposed of prior to the enactment of any implementing legislation. Since I was one of the principal opponents of retroactivity, I believe it important to spell out the policy reasons for our opting against it.

Our discussion of retroactivity centered on the currently active hazardous waste disposal facilities. We decided to recommend imposition of liability for injuries or damages which were unforeseeable only on a prospective basis. Thus, the operator of an existing facility would have the option of staying in business under the new rule or closing it down. Concerns in the insurance industry, where "claims made" and "occurrences" policies still apply for differing sites, might also be lessened. As to past acts, the negligent operator would continue to be liable to the victim (or to those who compensated him) under common law principles, but the risk of unforeseeable injuries would rest on the Fund and not the operator.

In deciding against retroactive application of a "strict liability" subrogation claim, the Study Group looked to the same equitable principles which underlie the constitutional prohibition against ex post facto laws, due process and the general rule of construction that a statute should only be given prospective application.[36] We also recognized that sporadic imposition of strict liability on a retroactive basis poses potentially serious impediments to the long term availability of adequate insurance coverage at reasonable rates.[37]

H. Election of Remedies. -- On this issue, I join in the Anderson, Breitel, et al. statement and dissent from the Report's recommendation.[38]

I was one of the five members of the Group who voted to require a "binding" election between pursuing Tier One and Tier Two remedies. I understood that such a requirement would also have included a limited "safety valve" to avoid substantially unjust or unconscionable results. The proposal in the preceding joint separate statement, which was advanced after our final meeting in Boston on June 28, is an elaboration of that concept.

A limitation on election is needed to make a two tier system work. Relaxation of tort standards posed by (1) the statutory presumptions recommended in Tier One and (2) the Report's extreme views of strict liability if applied in Tier Two, might result in claims in both Tiers that would make predictability of economic risk impossible. Such predictability is needed by industry and insurers. Without that predictability the system would become increasingly costly and economically inefficient. It is also possible that insurance could become unavailable.

Thus, in setting any compensation schedules for Tier One, considerable care should be taken in setting ceilings on specified awards. The amounts of compensation available might have to be limited in rough relationship to the weakening of the causal connection resulting from adoption of the recommended statutory presumptions.

Concern over predictability also leads me to caution that great care should be taken in fashioning schedules for "the psychological, social and related injuries sustained ... geared to tangible events objectively identified as physical and neurological consequences of physical injuries." What is intended is to provide for schedules for specified physical injuries somewhat similar to those available in first party accidental injury policies. The underlined words have been carefully chosen to preclude payments for what is essentially the subjective component in "pain and suffering" awards. That component in its more exaggerated form becomes "punitive" or "exemplary" damages. But whatever it is called, the vengeance concept has no place in a Tier One system.[39]

I. Punitive Damages -- I also believe that punitive or exemplary damages have no socially desirable role in Tier Two when a comprehensive scheme of regulation such as RCRA, encompassing civil and criminal sanctions, is in place. I regret that my colleagues who were divided over this issue decided not to address it one way or the other in the Report. For my part, I believe that where Congress has exercised its commerce power to impose a comprehensive system of regulation over an industrial activity or practice and elects to leave private remedies to State law, it should nevertheless preempt any State punitive and exemplary damages since in essence they duplicate the criminal and civil sanctions provided in the

Federal statute.[40] Vigilantes have no place when the sheriff takes over. In my view, that duplication also constitutes an unreasonable burden on interstate commerce since it may furnish a major deterrent to engaging in worthwhile economic activity. A third reason is that unless class actions are to be required, permitting punitive damages to be imposed on activities regulated under RCRA might result in overcompensation of a few claimants and their lawyers to the detriment of many equally worthy claimants.[41]

3. Federal "Common Law" and Federal Implied Rights of Action

The Study Group received an inquiry from Senators Stafford and Randolph concerning the effect of two recent Supreme Court decisions, the *Milwaukee* and *Sea Clammers* cases on Federal common law in this area.[42] I agree with the conclusions in the Report that Federal common law no longer exists and when existent probably did not afford private parties, as distinguished from States, a right of action in the Federal courts.[43] In addition, I do not believe that Congress can constitutionally create or recreate a private right of action in common law through a "savings clause".[44] Congressional modifications of savings clauses in regulatory statutes, such as those recently considered in connection with S. 1716 and H.R. 6307,[45] would not change the substantive result in *Sea Clammers* or *United States* v. *Price*.[46] They would only serve to produce unnecessary confusion and litigation. It is also clear that the views expressed in one Congress about a prior Congress' intent (e.g., suggesting the Supreme Court's "misinterpretation" of that intent) are entitled to little weight because of the abuses apparent in post-hoc legislative history.[47]

4. Reexamination of the Current Tort System

Concern over social costs and their incidence leads me to emphasize the importance of the general call of the Study Group and Judge Breitel for a reexamination of certain fundamentals of our current tort system.[48] The thoughtful paper submitted to us by Richard Schmalz, which is summarized extensively in Part III-B of the Report, contains specific suggestions on how liability might be "channeled" in generating, transporting and disposing of hazardous wastes, following precedents in the nuclear field both in this country and in Western Europe.[49] His suggestions merit serious consideration and may prove to be one of the most important contributions to current thinking in this general area stimulated by our Study.

Serious questions exist as to whether our present tort system is operating at optimal efficiency, particularly where it requires duplicative insurance against virtually unlimited damages, coupled with ever increasing risks of liability based only on the most tenuous connection with the event actually causing injury.[50] Are looser and looser definitions of "strict liability" in the public interest?[51] Are efforts to extend the concept of "joint and several" liability to allocate costs to the "innocent" as well as the "guilty" in the public interest?[52] Are there fairer, more efficient, less costly alternatives for compensating persons for personal injury and property damage?

These fundamental questions as to the Tier Two tort system, which our recommendations would leave substantially intact and little changed, need to be raised and answered with regard to compensation for injuries and damages from hazardous wastes and the numerous other fields of tort law that are now in controversy.[53]

NOTES

1. Although I was one of the three members of the Study Group named by William Reece Smith, then President of the American Bar Association, I have participated in the work of the Group as an individual member of the bar and not as a representative of that association. I also wish to acknowledge the assistance given me by my associate, Alfred Light, and my research assistant, Karen Donegan, during this Study.

2. Like Judge Breitel, I do not vouch for any of the research papers that appear in the appendices. Some were furnished to us in the course of our deliberations, others only in the last few days of our efforts. Thus, I was able only to review drafts of several of them, which I commented on in letters which appear in the "Comments to the Appendices."

3. The compromise eliminated both a federal private cause of action and joint and several liability; added a third-party defense; eliminated all third-party compensation; and established a limit on compensation for each incident of damage to natural resources. 126 CONG. REC. S14,967-68 (daily ed. Nov. 24, 1980) (remarks of Sen. Stafford); *id*. at S15,004 (remarks of Sen. Helms); Grad, TREATISE ON ENVIRONMENTAL LAW §§ 4A.01(1)(f), 4A.05 at 4A-164); Report at 38; *see* 126 CONG. REC. S15,002 (daily ed. Nov. 24, 1980) (compromise superfund does not provide out-of-pocket medical expenses for individuals) (remarks of Sen. Williams); *id*. at S14,967 (compromise superfund eliminates virtually all third-party compensation, including payment of medical expenses to victims of waste releases) (remarks of Sen. Stafford); 38 CONG.Q.WEEKLY REP. 3435 (Nov. 29, 1980) (deletion of medical expenses "biggest concession" in compromise bill); *cf*. S.1480, 96th Cong., 2d Sess. (1980) (original Senate bill calling for strict liability private cause of action); S.Rep. 96-848, 96th Cong., 2d Sess. (1980); S.1486, 97th Cong., 1st Sess. (1981) (attempt to amend Superfund Act to include private cause of action previously rejected). The proposed provisions in S.1480 reflected some, but not all, of the provisions along the same lines in other bills in that and several other Congresses. *E.g.*, S.1483, 97th Cong., 1st Sess., (1981) (radiation exposure); H.R. 85,

97th Cong., 1st Sess. (1981) (oil spill damage); S.681, 97th Cong., 1st Sess. (1981) (oil spill damage). The Report explains the Study Group's rationale for limiting the study at pages 26-27.

4. *See, e.g.*, S.Rep. 96-848, 96th Cong., 1st Sess. 117-118 (1981) ("The [proposed Federal tort] system of liability . . . is unique and bizarre . . . The problems in such areas of civil procedure as res judicata, collateral estoppel, conflict of laws, and bar and merger have been wholly overlooked . . . [T]his federal alternative available as a partial remedy will seriously retard the development of the common law . . . [T]here has been very little demonstration made in the record as to the financial ability of the victim compensation fund to address the claims which will be honored . . . [T]he fee system is seriously deficient and inequitable.") (minority views of Sen. Simpson); *Id.*, at 119-122 ("S.1480 changes State tort law . . . [and] constitutes an intrusion into judicial processes . . . upon which industries and businesses have relied in assessing their exposure to liability and the availability of insurance . . . The definition of 'hazardous substance' is exceedingly broad . . . [A defendant] would find himself facing a barrage of suits in which he was being held strictly liable for all of the damages regardless of whether or not he had any control over the situation . . . [The bill] does not provide for apportionment in the case where a significant contributor who, for example, was responsible for 60% of damages must bear the full 100% of the costs because the other contributors cannot be found or identified or are found to be insolvent . . . The issue of applying the new standards retroactively remains a troubling one . . . [Subrogation would] shift the balance of litigation . . . [and the law would no longer represent] the drawing of an equitable balance between private litigants . . . . Consequently, it would appear that private sector decisions [would] have to be made [on basis of the worst case]: (1) the defendant faces litigation brought by the Federal government [by subrogation of private claims]; (2) he is one of a few or the single defendant in a case where the release was largely caused by another operator; (3) the more significant contributor is insolvent; (4) actions beyond his reasonable control caused the release; and (5) joint and several liability under S.1480 expose the defendant to the possibility of paying all the damages.") (minority views of Senators Domenici, Bentsen and Baker).

5. This rejection also took into consideration but rejected the arguments advanced in currant literature which called for such a remedy both before and after passage of CERCLA. E.g., Meyer, Compensating Hazardous Waste Victims: RCRA Insurance Regulations and a Not So "Super" Fund Act, 11 ENVIR.L. 689, 719 (1981); Sobel, A Proposal for the Administrative Compensation of Victims of Toxic Substance Pollution: A Model Act, 14 HARV.J.LEGIS. 683 (1977) (similar to Brodhead bill). Note, Inactive or Abandoned Hazardous Waste Disposal Sites: Coping with a Costly Past, 53 S.CAL.L.REV. 1709, 1745 (1980). The Study Group explicitly considered the S.1480 and Brodhead bill proposals and declined to recommend them. E.g., Report, at 215-218, 228-229, 238-239.

6. Younger v. Harris, 401 U.S. 37, 44-45 (1971) (Black, J.) ("[Continued adherence to the principle of Federalism encompasses] a notion of 'comity,' that is, a proper respect for state functions, a recognition of the fact that the entire country is made up of a Union of separate state governments, and a continuance of the belief that the National Government will fare best if the States and their institutions are left free to perform their separate functions in their separate ways. This, perhaps for lack of a better and clearer way to describe it, is referred to by many as "Our Federalism" and one familiar with the profound debates that ushered our Federal Constitution into existence is bound to respect those who remain loyal to the ideals and dreams of "our Federalism." The concept does not mean blind deference to "States' Rights" any more than it means centralization of control over every important issue in our National Government and its courts. The Framers rejected both these courses. What the concept does represent is a system in which there is sensitivity to the legitimate interests of both State and National Governments, and in which the National Government, anxious though it may be to vindicate and protect federal rights and federal interests, always endeavors to do so in ways that will not unduly interfere with the legitimate activities of the States."); see also Friendly, Federalism: A Forward, 86 YALE L.J. 1019 (1977). The Study Group discussed the possibility and potential unconstitutionality of mandating State implementation and administration of the proposed Tier One compensation system. But we noted that the Supreme Court "never has sanctioned explicitly a federal command to the States to promulgate and enforce laws and regulations." Federal

Energy Regulatory Commission v. Mississippi, No. 80-1749 (Jun. 1, 1982), *slip op.*, at 18. The Group opted instead for a "cooperative federalism" or partnership role between the two levels of government based on the model in the Federal Water Pollution Control Act, rather than a hierarchical model "antithetical to the values of federalism and inconsistent with our constitutional history." FERC v. Mississippi, *supra*, (O'Connor concurring and dissenting), *slip op.*, at 1. The national government should not be able "to force state governments to administer unpopular programs, thus transferring political liability for those programs to the States." FERC v. Mississippi, *supra* (O'Connor), *slip op.*, at 14n.19, *citing* Advisory Commission on Intergovernmental Relations, THE FEDERAL ROLE IN THE FEDERAL SYSTEM: THE DYNAMICS OF GROWTH, HEARING ON THE FEDERAL ROLE, p. 32 (October 1980) (statement of Daniel J. Elazar). *cf.* S.Rep. No. 97-151, 97th Cong., 1st Sess. 52 (1981) ("a policy . . . implicit in the institution of federalism created by the Constitution.").

7. CAL. HEALTH & SAFETY CODE, §§ 25370- 25382 (West 1981); *see* Report, at 66-67. I believe that an "orphan site" category includes (a) sites for which no solvent disposal site operator exists and (b) sites for which no other responsible party connected to the site *should* be held responsible. For example, landowners on whose property waste was disposed without their knowledge should not be responsible parties. *See* Report, at 244. Neither should present landowners who purchased property without reason to know the dangerous condition existing on the land. *See* Report, at 34-35.

8. N.Y. TIMES (Jun. 22, 1982), p. B.4. The state Superfund law New York enacted, however, does not provide for compensation of personal injuries. S. 10620 (July 2, 1982).

9. *See* Report, at 178-179.

10. These same factors were evident in the Reagan Administration's decision to promulgate compulsory financial responsibility requirements for licensed owners and operators of hazardous waste management facilities. 47 Fed. Reg. 16544, 16545 (Apr. 16, 1982) (to be codified at 40 C.F.R. Parts 123, 264 and 265). They also have been relevant in discussions at the International Institute for the Unification of Private Law (UNIDROIT) Committee of Governmental Experts, which

is preparing uniform rules for European nations relating to liability and compensation for damage caused during the carriage over land of hazardous substances.

11. Report, Part II; *see also* Grad letter to Assistant Attorney General Carol Dinkins, dated December 18, 1981, and Freeman letter to Chairman Zazzali, dated March 2, 1982 in the "Comments to the Appendices."

12. *See* Report, at 184. The Love Canal study released July 14, 1982 suggests that health problems related to that site may have been overstated. United States Environmental Protection Agency, ENVIRONMENTAL MONITORING AT LOVE CANAL, EPA-600/4-82-030a (May 1982). *See also* H.R.Rep. No. 96-IFC 31, 96th Cong., 1st Sess. 33 (1979) (concluding that early estimates of the number of waste sites were in error); Report, at 21n.19, 23n.34; one puzzling aspect of determining the extent of the abandoned waste site problem has been the suggestion that EPA is experiencing difficulty in finding sites appropriate for cleanup. *See EPA Expecting to Run Out of Sites to Clean Up, Expands Eligibility*, 13 ENVIR. REP. (BNA) 195 (Jun. 18, 1982).

13. The potential for abuse of the Tier One System remains even if the system were to be funded out of general tax revenues. However this potential is greater in the "special fund" situation because the periodic scrutiny incident to the annual budget and appropriations process is less rigorous in the case of special funds. Recent experience with the Federal Black Lung Benefits program and the New Jersey Spill Compensation Fund furnish examples. *See* Lopatto, *Amendments Fight Excesses of Black Lung Statute*, LEGAL TIMES (Mar. 22, 1982); *NJ Auditors Question Spill Fund Management Controls*, HAZARDOUS WASTE LIT. REP. 2,444 (May 18, 1982).

"Superfund" itself is controversial because funds have *not* been advanced to clean up abandoned hazardous waste dumps as expeditiously as Congress apparently intended. It has been charged that this delay has been attributable, in part, to EPA and the Justice Department's preoccupation with pursuing potentially responsible parties. *See* Baise & Bourdeau, *EPA's Implementation of the Superfund Response Program*, 3 ENVIR. ANALYST 3 (May 1982); *Administrative Delays Plague Cleanup Under Superfund, Indiana Congressman Says*, 13 ENVIR. REP. (BNA) 167 (Jun. 11, 1982); *cf*. 47

Fed. Reg. 20664, 20666 (May 13, 1982) (guidelines for using the imminent hazard, enforcement and emergency response authorities of Superfund and other statutes); EPA Guidance Memorandum on Establishing National Priorities List Under Superfund (Dated June 28, 1982), 13 ENVIR.REP. (BNA) 339, 341 (Jul. 2, 1982) ("EPA will consider Fund financed cleanup of [mining] sites only after all available enforcement efforts fail.").

Robert Hershey has reported more generally on rising criticism of the Federal Government's many insurance programs, which even without counting social security or unemployment insurance now cover more than $2,000 billion in risks. He quoted former OMB official Robert G. Penner, now at the American Enterprise Institute, as saying, "The real problem is the lack of careful scrutiny of these things." Hershey, U.S. Insurance; Critics Grow 131 N.Y. TIMES (Jun. 8, 1982), at 1, col. 2; 6, col. 1.

14. One major subcategory of administrative costs virtually overlooked in the Study Group's deliberations (see Report, at 231) are intergovernmental costs, the costs of assessing each State's prospective need for Tier One funds from the Federal Government and the timely periodic transfer of funds between the two levels of government. Because of (1) the proposed relaxation of liability (or "eligibility") requirements through the recommended statutory presumptions in Tier One, (2) the interpretation of "strict liability" advanced by the Report for Tier Two, and (3) because of the generous benefit levels recommended, the potential for the kind of "tragedy of the commons" which the social services block grant program (Title XX of the Social Security Act) experienced in the seventies exists. Derthick, UNCONTROLLABLE SPENDING FOR SOCIAL SERVICES (1977); Hardin, The Tragedy of the Commons, 162 SCIENCE 1243 (1968). This "tragedy" is that each State obviously has an incentive for maximizing its share of the funds, and in the absence of identifiable limiting principles on the group of eligible claimants and the consequent ability of the federal government to estimate need on a rough actuarial basis, the system's overall costs could well soar notwithstanding a modest need for the system to compensate injuries "actually" caused by hazardous wastes.

15. See Report, at 161, 230-232.

16. *See* Report, at 7, 10-11.

17. *See* Report, at 8-17; 24n.47.

18. *See* Leape, *Quantitative Risk Assessment in Regulation of Environmental Carcinogens*, 4 HARV.ENVIR.L.REV. 86, 107 (1980); Ricci and Molton, *Risk and Benefit in Environmental Law*, 214 SCIENCE 1096, 1099-1100 (1981).

19. The direct costs of the Tier One system would be substantially higher because the Group would allow recourse to the Tier One Fund at inactive waste dumps, even though a solvent responsible party is known, as well as at "orphan" sites. This contrasts with the Black Lung Act's compensation of miners only "when there is no responsible mine operator available to provide payments, when there is operator default and when the coal miner's last employment was before January 1, 1970." Baram, ALTERNATIVES TO REGULATION 216 (1982). I would limit compensation from the Fund at inactive sites to "orphan sites" as California did in its recently established state Superfund law. CAL. HEALTH & SAFETY CODE §§ 25370-25383 (West 1981). *See also* Report, at 66, 267n.94.

20. Claims under the Black Lung Benefits Act have exceeded substantially earlier estimated costs. *See* U.S. DEPARTMENT OF LABOR BRIEFING PACKAGE ON BLACK LUNG LEGISLATIVE PROPOSAL (1981). For example, in 1977, benefit outlays for 1978-81 were estimated to be $1,121,100,000. H.Rep. No. 95-151, 95th Cong., 2d.Sess. 27 (1978), *reprinted in* [1978] U.S. CODE CONG. & AD. NEWS 263. Actual expenditures proved to be $2,178,400,000 (a $1,057,300,000 difference.) 127 Cong. Rec. S. 15480 (Dec. 16, 1981). Black Lung benefit expenditures since 1969 stand at over $9 billion. HEARINGS BEFORE THE SUBCOMMITTEE ON OVERSIGHT OF THE COMMITTEE ON WAYS AND MEANS, HOUSE OF REPRESENTATIVES, 97th Cong., 1st Sess. 3 (1981) (Henig, Morton E.).

21. The timing of governmentally mandated expansions of industry costs could well be crucial to the prospect that the additional costs could be absorbed or passed on to consumers and thus internalized. Even where foreign competition is not a significant factor in the U.S. market, internalization is much less likely to occur when the economy is in a recession or depression. *See* Calabresi, *Some Thoughts on Risk Distribution and the Law of Torts*, 70 YALE L.J. 499, 520 (1961).

22. We should profit from our experience in other areas of environmental law. In some areas, such as automobile emissions controls under the Clean Air Act, we have been successful in internalizing pollution costs in the American submarket for automobiles because all cars sold within this country must meet the same emissions standard wherever they are manufactured. But efforts at internalization of pollution control costs have largely failed in U.S. submarkets such as steel, where foreign producers are not required to incur equivalent control costs. Tariffs are one means for offsetting otherwise discriminatory costs on our own industries, but they run counter to other national priorities. Even if tariffs were imposed for this purpose, they could provoke similar barriers to sale of American goods abroad. There are potential international forums for resolving these "internalization" dilemmas through negotiation. *See Legal Committee Completes Draft Covering Hazardous Substances Liability*, 5 INT'L ENVIR.REP. 182 (May 12, 1982) (United Nations Intergovernmental Maritime Consultative Organization); *OECD Waste Management Policy Group Plans Work on Export of Hazardous Waste*, 5 INT'L ENVIR.REP. 183 (May 12, 1982) (Organization for Economic Cooperation and Development). But as yet they are ineffective. A recent OECD report presented at the May 1982 Nairobi meeting on the global environment warned developing countries about the environmental aspects of their planned activities. "These countries are aiming at higher than average growth rates, their environmental machinery is incomplete, the backlog of pollution is large, and consequently the pressure on their relatively fragile environment is already severe." *Nairobi Meeting on Global Environment Looks at Progress Made in Past 10 Years*, 5 INT'L ENVIR.REP. 175 (May 12, 1982) (statement of James MacNeil, Director of OECD's Environment Directorate).

23. *See* Freeman letter to Grad, dated Feb. 19, 1982, in "Comments to the Appendices." In addition to believing that the recommended statutory presumptions in Recommendation Four [Report, at 198-199] are not sound policy here, I find their wording confusing. They represent a drastic modification of presumptions initially suggested by the Reporter [Appendix N, p. 314] which were rejected by the Group.

The Study Group modified the Reporter's suggested presumptions in two major ways. First, recognizing that diseases associated with hazardous waste exposure also may be associated with other causes, the Study Group substituted the words "contributed to" for "caused" in presumption (a) and voted to delete the word "solely" from presumption (b). Second, the Group decided to delete all references to a "person against whom a claim is made" in the statement of the presumptions and the showing which triggers them because it wished to eliminate the necessity for the claimant to identify a potential subrogee once he had identified the source of his exposure (i.e., the waste site or incident).

The presumptions as modified do not resolve the question of how strong a causal connection there should be between an industrial activity and an injury before the loss should be shifted to the Fund. Nor do they preclude the possibility that (a) the amount of a compensation award would be scaled in relation to the strength of the causal connection or (b) reduced if other contributory causes of the disease were present.

What do the modified presumptions actually accomplish? To benefit from either presumption, the claimant must prove by a preponderance of the evidence (1) the existence of a source which at the time of exposure was part of the hazardous waste system, (2) exposure to such hazardous waste, and (3) that the claimant suffered injury of the kind that is known to result from such exposure. Having made these showings, the first presumption that arises is that the waste contributed to his injury. Thus, the claimant is entitled to at least nominal damages unless the Fund rebuts the presumption, so long as the other eligibility criteria have been satisfied (e.g., a public collateral source has not compensated the claimant). The second presumption is more confusing. It states that the waste was responsible for the exposure and the resulting injury. The only reasonable explanation of this second presumption apparent in light of the modifications made to the earlier presumption drafts and the reasons for those modifications is that the Study Group wishes to place the burden of proof with respect to any contributory negligence defense the Fund might have on the Fund rather than the claimant. That is, as between the claimant and the Fund, the Fund bears the burden of proving that the claimant was responsible for the exposure and its consequences.

The Reporter cites the Workers' Compensation and Black Lung disease presumptions of compensability in his discussion of the Fourth Recommendation, indicating that they "serve the purpose of ruling out claimant's own physical weakness or habits as a contributory factor." [Report, at 203] This interpretation is, however, contrary to my reading of the presumptions and the meaning of the modifications made in them by the Study Group. In the Workers' Compensation and Black Lung situations, there are at least identifiable limited target populations, *i.e.*, employees or coal miners, although the Black Lung program has experienced considerable controversy over the revised statutory definition of "miner" which grew out of the 1972 and 1977 amendments to the Black Lung Act. Baram, ALTERNATIVES TO REGULATION 219 (1982) ("[Hazardous waste effects] are not effects that are confined to a narrow constituency like employees in a work place."); BLACK LUNG BENEFITS AND REVENUE AMENDMENTS OF 1981: HEARINGS ON S.1922 BEFORE THE SUBCOMM. ON LABOR OF THE COMM. OF LABOR & HUMAN RESOURCES, 97th Cong., 1st Sess. 119, 128-131 (1981) (statement of Frederick W. Webber). While it may be appropriate for employers to take care of employees who contract diseases which are sometimes but not always work-related, it is not appropriate to expect industry to provide such a service to non-employees. *See* *Diet, Alcohol, not Chemicals, Cited as Major Factors in Cancer Development*, 6 CHEM. REG. REP. (BNA) (Jun. 18, 1982); National Research Council, DIET, NUTRITION, AND CANCER (1982).

24. *See* Comptroller General of the United States, LEGISLATION ALLOWS BLACK LUNG BENEFITS TO BE AWARDED WITHOUT ADEQUATE EVIDENCE OF DISABILITY, HRD-80-81 (Jul. 29, 1981). In 1981, Congress repealed some of these presumptions, in a reflection of widespread dissatisfaction with the program. *See* 127 Cong. Rec. H. 9600-H. 9606 (Dec. 15, 1981); 127 Cong. Rec. S. 15474-S. 15498 (Dec. 16, 1981); 127 Cong. Rec. H. 9788-H. 9798 (Dec. 16, 1981); *see* Appendix to this statement.

25. He does so again in his Comments. *See* Breitel, at 6.

26. *Cf.* EPA's Proposed National Emission Standards for Hazardous Air Pollutants; Policy and Procedures for Identifying, Assessing, and Regulating Airborne Substances Posing a Risk of Cancer, 44 Fed. Reg. 58642 (Oct. 10, 1979); and Comments of the Utility Air Regulatory Group on the Proposed Regulations dated Feb.

21, 1981, in EPA Docket No. OAQPS-79-14. See also Rizzo & Arnold, Causal Apportionment in the Law of Torts: An Economic Theory 80 COLUM.L.REV. 1399, 1426 (1980) ("[C]ourts [are] becom[ing] increasingly aware of the merits of apportioning damages in tort . . . [and should be encouraged] to cast off their traditional reluctance to apportion damages among joint tortfeasors.").

27. See, Breitel, at 6; see also Note, Allocating the Costs of Hazardous Wastes Disposal, 94 HARV.L.REV. 584, 600 (1981) ("Alternatively, a rebuttable presumption of causation could be created in favor of plaintiffs in selected circumstances, with the result that responsible firms of the entire industry would be liable for costs greatly in excess of the demonstrated costs of the wastes. Such a system would be erratically punitive.").

Note that this multiple causation problem would not go away even if one were to eliminate subrogation and the retroactive aspects of a compensation plan completely. Non-subrogation simply shifts the ultimate incidence of a claim from an individual subrogee company to the taxpaying industries collectively. To the extent the taxpaying industries, even collectively, pay costs largely attributable to other causes of disease (e.g., the claimant's own habits, or his exposures to hazardous substances generated by non-taxpaying industries), the system distorts the marketplace for hazardous waste services and in effect creates a forced waste industry subsidy of other parts of society. See generally Stone, REGULATION AND ITS ALTERNATIVES 91-123 (1982); Klemme, The Enterprise Liability Theory of Torts, 47 U.COLO.L.REV. 153, 156-165 (1976). If the Tier One scheme through its presumptions and apportionment rules permits such distortions, then a special industry tax is completely inappropriate. The confusion evident in interpretation of what may be relatively innocuous presumptions in Recommendation Four confirms my view that both the funding and the presumptions recommendations are wrong. See note 23 supra.

28. Jeter v. Hewitt, 22 How. 345, 348, 63 U.S. 352 (1859).

29. See S.Rep. 97-284, 97th Cong., 1st Sess. (1981); cf. Admin. Conference of the United States, Recommendation 82-5, Federal Regulation of Cancer-Causing Chemicals (Jun. 18, 1981).

30. TO ESTABLISH A DEPARTMENT OF ENERGY AND NATURAL RESOURCES, ENERGY RESEARCH AND DEVELOPMENT ADMINISTRATION, AND A NUCLEAR SAFETY AND LICENSING COMMISSION: HEARINGS ON S.2135 AND S.2744 BEFORE THE SUBCOMM. ON REORGANIZATION, RESEARCH, AND INTERNATIONAL ORGANIZATIONS OF THE SENATE COMM. ON GOVERNMENT OPERATIONS, 93rd Cong., 2d Sess. 172, 195-211 (1974) (Statement of George Freeman); *see* Admin. Conference of the United States, Recommendation 82-5 (Jun. 18, 1982), at section V (generic rulemaking on cancer-causing chemicals); Gellhorn, *Public Participation in Administrative Proceedings*, 81 YALE L.J. 359 (1972); Comment, *Public Participation in Federal Administrative Proceedings*, 120 U.PA.L.REV. 702 (1972); Cramton, *A Comment on Trial-Type Hearings in Nuclear Power Plant Siting*, 58 VA.L.REV. 585 (1972); Cramton; *The Why, Where, and How of Broadened Public Participation in the Administrative Process*, 60 GEORGETOWN L.J. 525 (1972); Clagett, *Informal Action-Adjudication-Rulemaking: Some Recent Developments in Federal Administrative Law* 1971 DUKE L.J. 51 (1971); Murphy, *The National Environmental Policy Act and the Licensing Process: Environmentalist Magna Carta or Agency Coup de Grace?* 72 COLUM.L.REV. 963 (1972); Robinson, *The Making of Administrative Policy: Another Look at Rulemaking and Adjudication and Administrative Procedure Reform*, 118 U.PA.L.REV. 485 (1970).

31. Report, at 245, 248-250. Also, the Report is silent concerning whether the presumptions are to apply in the subrogation action. In my view, the proposed statutory presumptions should not since these particular presumptions are inappropriate in either Tier.

32. Breitel, at 4-5; *See* Freeman letter to Grad, dated March 10, 1982, in "Comments to the Appendices."

33. The Report's analysis [Report at 107-108] to the contrary notwithstanding, the New Jersey courts in *New Jersey v. Ventron Corp.* did not pronounce the sort of liability standard described in the Report for cleanup costs under the New Jersey Spill Control and Compensation Act. N.J.S.A. 58:10-23 *et seq.* (1977). The trial court noted in that case that "[i]n this case it was known by the liable defendants that mercury had dangerous potentialities." No. C-2996-75 (Super. Ct., Ch. Div. Bergen County, Aug. 22,1979), at 55; 1 CHEM & RADIATION LIT.REP., at 376 The appellate court in that case found that it could constitutionally apply the statute's liability standard retroactively because

the statute did *not* create a new "substantive liability." Instead, "[i]t was remedial only, providing a procedure for the imposition of costs to pay for the cleanup of prior *wrongful* discharges." 182 N.J. Super. 210 (App.Div. 1981) (emphasis added); *see* Report, at 150n.306. Furthermore, even when a statute appears to adopt an absolute liability standard making the defendant's knowledge or the objective foreseeability of an accident immaterial, foreseeability is still relevant on the issue of causation. The balancing of social costs and benefits of awarding compensation in a particular case at the expense of an industrial activity *always* takes place, at least implicitly, in a tort suit. Kuster, *Toxic Substance Contamination: The Risk-Benefit Approach to Causation Analysis*, 14 MICH.J.L.REFORM 53, 64-65 (1980) ("[P]olicy considerations are . . . manifest in courts' proximate cause determinations generally.").

34. Appendix L.

35. *See* Report at 161, 230-231, 234-235. In addition, I share Professor O'Connell's concern about the multiplicative number of proceedings arising out of the same incident. *See also* Professor O'Connell's separate statement. I would have substantially reduced the likelihood of repetitious proceedings by not only requiring in Tier One that notice be given to potential subrogees [Report at 231] but also by giving them a right, which is direct or indirect under most Workers' Compensation statutes, to participate in such proceedings.

36. U.S. Const., Art. I, § 9, cl. 3 (Congress forbidden to pass ex post facto laws); U.S. Const., Art. I, § 10, cl. 1 (state legislatures forbidden to pass ex post facto laws); the framers of the Constitution intended the clauses to apply to retroactive civil legislation as well as criminal legislation. *See* Crosskey, *The Ex Post Facto and Contract Clauses in the Federal Convention: A Note on the Editorial Ingenuity of James Madison*, 35 U.CHI.L.REV. 248 (1968); Field, *Ex Post Facto in the Constitution*, 20 MICH. L.REV. 315 (1922) but the clauses have been construed to apply *per se* only to criminal statutes. Calder v. Bull, 3 U.S. (3 Dall.) 386 (1798); the following state constitutional provisions prohibit all retroactive legislation: Colo. Const., Art. II, § 11; Ga. Const., Art. I, § 3(2); Idaho Const., Art. XI, § 12; Mo. Const., Art. I, § 13; N.H. Const., Art. I, § 23; Ohio Const., Art. II, § 28;

Tenn. Const., Art. I, § 20; Tex. Const., Art. I, § 16; *cf.* Murrill v. State Board of Accountancy, 97 Cal. App.2d 709, 218 P.2d 569 (1950) (retroactivity violated due process clause); Gayle v. Edwards, 261 Ala. 84, 72 So.2d 848 (1954) (state constitutional violation found in failure to disclose in title of legislation its retroactive effect); SUTHERLAND, STATUTORY CONSTRUCTION, §§ 41.02 - 42.12; Lundquist v. Coddington Bros., Inc., 202 F.Supp. 19 (W.D. Wis. 1962) (statute may not apply retroactively to revive a cause of action already barred by the statute of limitations); Haase v. Sawicki, 20 Wis.2d 308, 121 N.W.2d 876 (1963) (statute attempting to reinstate cause of action objectionable); Massey v. Sullivan County, 464 S.W. 2d 548 (Tenn. 1971) (statute abolishing rule of no contribution for joint tortfeasors cannot be applied retroactively). A law will not be construed as retroactive unless the act clearly, by express language or necessary implication, indicates that the legislature intended a retroactive application. United States v. Whyel, 28 F.2d 30 (3d Cir. 1928); Crawford v. Worth, 447 F.2d 738 (5th cir. 1971); First Nat. Bank in Billings v. First Bank Stock Corp., 197 F.Supp. 417 (D.Mont. 1961); Elliot v. Volpe, 328 F.Supp. 831 (D.Mass. 1971); Douglas Aircraft Co. v. Cranston, 58 Cal.2d 462, 24 Cal.Rptr. 851, 374 P.2d 819 (1962) ("a statute should be given the least retroactive effect that its language reasonably permits."); Mau v. Superior Court, etc., 101 Cal.App.3d 875, 881; 161 Cal.Rptr. 895, 898 (1980) (unfairness of retroactive application to insurers who relied on the previous law in setting premium rates).

Prospective only application of "strict liability" reflected a compromise between those Study Group members who advocated an "industry standard of care" standard for subrogation claims and those who advocated retroactive "strict liability." While the problem of parsing out injuries resulting from past disposal and injuries resulting from post-enactment disposal is difficult, those same difficulties exist under present law (*e.g.*, in determining when an accident occurs for purposes of insurance policy coverage).

37. *See* Report, at 161; Freeman letter to Superfund 301(e) Study Group, dated April 12, 1982, in "Comments to the Appendices;" U.S. Department of the Treasury, HAZARDOUS SUBSTANCE LIABILITY INSURANCE 81 (Mar. 1982).

38. *See* Separate Statement of Messrs. Anderson, Breitel, Freeman and O'Connell. *See also* comments on the Report by Messrs. Anderson, Breitel, Lundquist, O'Connell and Spannaus.

39. Strict liability, apportionment and damage issues are inextricably intertwined, as the Report sometimes fails to emphasize. For example, both the alternative liability and market share theories have been applied or considered only in cases involving *negligent* defendants. *Cf*. Report, at 49. Coupling either with recovery based on a theory of strict liability would go beyond current case law. Justice Traynor has recognized that strict liability situations are different. Limitations on damages, for example the exclusion of pain and suffering, are appropriate to prevent unfairness when liability is permitted without proof of fault. *See* Seffert v. Los Angeles Transit Lines, 56 Cal. 2d. 498, 511; 364 P.2d 337, 345; 15 Cal.Rptr. 161, 169 (1961) (Traynor, J. dissenting); and *see* O'Connell & Simon, PAYMENT FOR PAIN AND SUFFERING: WHO WANTS WHAT, WHEN AND WHY (1972); Levy & Ursin, *Tort Law in California: At the Crossroads*, 67 CALIF.L.REV. 497, 520-530 (1979).

40. *Cf*. Duke Power Co. v. Carolina Environmental Study Group, Inc. 438 U.S. 59, 84-94 (1977) (upholding Price-Anderson Act); Silkwood v. Kerr-McGee Corp., 667 F.2d 908, 922-923 (10th Cir. 1982) ("[P]unitive damages are 'awarded' against a person to punish him for his outrageous conduct and to deter him and others like him from similar conduct in the future.' *Restatement (Second) of Torts* § 908(1)(1979). *See also* City of Newport v. Fact Concerts, Inc., 453 U.S. 247, 266 (1981). . . [Thus where Congress has preempted the field of regulation of the nuclear industry, we are required] to hold invalid any state action that competes substantially with AEC (NRC) in its regulation of radiation hazards associated with plants handling nuclear material. A judicial award of exemplary damages under state law as punishment for bad practices involving exposure to radiation is no less intrusive than direct legislative acts of the state. Thus, we hold that punitive damages may not be awarded in this case.")

The intrusiveness of State punitive damages on the federal scheme of regulation is heightened where the Federal government requires the licensed persons to meet specified financial responsibility requirements, directly or through insurance. *e.g*. 42 U.S.C. § 6924(6) (1976) (RCRA); 42 U.S.C. § 9608 (1980) (CERCLA). Coverage against punitive damages is usually excluded from comprehensive general liability policies since as the Ninth Circuit recently noted "no rule of law limits the amount of punitive damages a jury may award." In re: Northern District of California "Dalkon Shield" IUD Products Liability Litigation No. 81-4648 et al, slip op. (Jun. 18, 1982). And intrusiveness into an intended uniform Federal scheme of regulation is compounded by the wide variations existing among the states as to when punitive damages may or may not be imposed. "[T]he 50 jurisdictions in which these [Dalkon Shield] cases arise do not apply the same punitive damages standards. Punitive damages standards can range from gross negligence to reckless disregard to various levels of willfullness and wantonness. Some states, in fact, do not allow punitive damages at all." Id.

Permitting punitive damages to be awarded in state tort actions for the same actions that are punishable by federal criminal and civil sanctions constitutes a form of double jeopardy -- multiple punishment for the same offense. While this multiple punishment does not violate the letter of the Double Jeopardy Clause of our Constitution, it is alien to its spirit.

41. See e.g. In re Northern District of California "Dalkon Shield" IUD Products Liability Litigation, 521 F.Supp. 1188, 1193 (1981), where the district court ordered certification of a nationwide class of all claims for punitive damages under FRCP 23(b)(1)(B) on two grounds. One was that:

> "It is clear that the amount of punitive damages sought far exceeds the available net worth of the company. This fact poses two very real threats if actions on this issue continue on an individual basis: 1) The company will be unable to respond to claims for punitive damages due to actual or constructive bankruptcy; or 2) At some point in the future, courts could rule that the aggregate sum alredy assessed against the defendant company in punitive damages was such that as a matter of law the company had been sufficiently punished and therefore punitive damage claims would be dismissed as a matter of law." 521 F.Supp. at 1193.

The Ninth Circuit Court of Appeals reversed on this point because:

> "The district court certified the 'limited fund' punitive damage class before requesting or receiving any evidence of Robins' net worth,

> earnings or available insurance coverage. After he ordered certification, the judge received Robins' attorney's affidavit showing the total claims against Robins and Robins' fund of assets but did not reopen discovery to permit plaintiffs to challenge these affidavits." No. 81-4648, *slip op*.

For a summary of the reasons for this overdeterrent effect, *see*, *e.g.*, Parnell, *Manufacturers of Toxic Substances: Tort Liability and Punitive Damages*, 17 FORUM 947, 965-967 (1982) ("Punitive damages, when considered in the setting of multiplaintiff litigation, are counterproductive. . .").

42. *See* letter from Senator Jennings Randolph and Senator Robert T. Stafford to Zazzali, dated April 29, 1982, in "Comments to the Appendices." City of Milwaukee v. Illinois, 451 U.S. 304 (1981); Middlesex County Severage Authority v. National Sea Clammers Association, 453 U.S. 1 (1981).

43. Report, at 70-75 and Appendix F. *See* Freeman letter to Grad dated December 16, 1981, and Freeman letter to Superfund 301(e) Study dated May 17, 1982 [with enclosure] in "Comments to the Appendices." *See* also Grad, TREATISE ON ENVIRONMENTAL LAW § 4A.05 at 4A-173 (Jun. 1982) ("[T]here is clearly no private right of action left under the federal common law of nuisance.") Even prior to 1981, when the Supreme Court made the matter only of academic interest, it was generally recognised that federal common law was "apparently confined to use by governmental entities . . ." Department of Justice, Land and Natural Resources Division, THE SUPERFUND CONCEPT: REPORT OF THE INTERAGENCY TASK FORCE ON COMPENSATION AND LIABILITY FOR RELEASES OF HAZARDOUS SUBSTANCES 16-17 (June 1979). The now defunct doctrine of the federal common law of tort or nuisance was predicated on concepts of State sovereignty and federalism, rather than individual rights, and therefore had no application to nongovernmental plaintiffs. *See* Comment, *Requiem for the Federal Common Law of Nuisance*, 11 ENVIR.L.REP. 10,191 (1981) ("The roots of the federal common law of nuisance reach back to the first decade of this century, when the Supreme Court for the first time acknowledged the jurisdiction of the federal courts to entertain cases brought by the States to abate pollution that has migrated across state lines . . . on the basis of rules borrowed from water apportionment cases and 'principles of federalism.'"); *cf*. In re

"Agent Orange" Product Liability Litigation, 635 F.2d 987 (2d Cir. 1980), *cert. denied sub nom.* Chapman v. Dow Chemical Co., No. 80-1882 (Dec. 14, 1981) (No federal common law tort liability for suppliers of "Agent Orange"). Milwaukee v. Illinois and Michigan held in 1981 that Congress' "establishment of a comprehensive regulatory program supervised by an expert administrative agency" supplanted the possibility of a federal common law available to State plaintiffs in water pollution cases. 451 U.S. at 317. Middlesex County Severage Authority v. National Sea Clammers Association therefore held that it was moot "whether [a] federal common law of nuisance could ever be the basis for a suit for damages by a private party." 453 U.S. at 11n.17. The lower courts have applied this rationale to hazardous waste cases, where RCRA and CERCLA provide the comprehensive statutory scheme which has supplanted any possible federal common law. United States v. Price, 523 F.Supp. 1055, 1069 (D.N.J. 1981) ("The comprehensive nature of the schemes established by the RCRA and the CERCLA require us to conclude that, if federal common law ever governed this type of activity, it has since been preempted by those statutes."); United States v. Kin-Buc, Inc., 532 F.Supp. 699 (D.N.J. 1981).

44. *See* Freeman letter to Orad, dated December 16, 1981, and Freeman letter to Superfund 301(e) Study Group dated May 17, 1982, [with enclosure]. *See also* Comment, *The Supreme Court Closes the Door on Implied Causes of Action Under Environmental Statutes; Middlesex County Severage Authority v. National Sea Clammers* 1 VA. J. NAT. RESOURCES L. 304, 326-327 (1981); Casenote, *Middlesex County Severage Authority v. National Sea Clammers Association; Implied Private Rights of Action for Damages Under the Federal Water Pollution Control Act Amendments of 1972*, 12 ENVIR.L. 197, 223-224 (1981); *cf.* S.Rep. No. 97-284, 97th Cong., 1st Sess. 164-165 (1981) (regulatory reform act).

45. *See* 127 Cong. Rec. S.12249 (daily ed. Oct. 27, 1981) ("technical" amendment to Clean Water Act in S.1716); H.R. Rep. No. 97-570, 97th Cong., 1st Sess. 21-22 (1982) (H.R. 6307, § 10).

46. 523 F.Supp. 1055 (D.N.J. 1981).

47. *E.g.*, Consumer Product Safety Commission v. GTE Sylvania, 447 U.S. 102, 118 (1980) (post hoc statement by conference committee entitled to little weight);

Waterman S.S. Corp. v. United States, 381 U.S. 252, 268-269 (1965) (legislative history of act passed long after another act does not afford any aid in interpreting earlier statute); Davies Warehouse Co. v. Bowles, 321 U.S. 144, 156 (1943) (suggesting post-hoc administrative interpretation of statute litigation inspired); Penn. Mut. L. Ins. Co. v. Lederer, 252 U.S. 523, 537 (1920) (no aid in construing an act of Congress can be derived from the legislative history of another passed six years later).

48. Report, at 240-256; Breitel at 9-10; In addition, even in the description of current law in the Report's Part III, time, budgeting and staff constraints obviously prevented the Group from completing the kind of comprehensive and thorough job typical of the Restatements. While the topics that are addressed in Part III of the Report are generally handled adequately, many other important legal matters were not addressed (*e.g.*, issues of contribution and indemnification, methods of apportionment such as comparative fault or comparative causation, legal defenses such as assumption of the risk and contributory negligence, and many insurance issues). More particularly, nothing in the Report should be construed to imply that hazardous waste companies under current law bear (or should bear), individually or collectively, the health or other social consequences of injuries or diseases to the extent those injuries or diseases are caused by others. Furthermore, where a better method of apportionment exists based on evidence in an individual case, joint and several apportionment may be inappropriate even though the burden of establishing the better apportionment method (*e.g.*, comparative fault, comparative contribution) might properly be placed on the liable defendants collectively rather than on the plaintiffs, according to the Study Group majority report. *Cf.* RESTATEMENT (SECOND) OF TORTS, § 433B(2) (1965) (burden of proof on apportionment); Report, at 69 (California "Superfund" apportionment rules).

49. Schmalz, *"Superfunds" and "Tort Law Reforms" -- Are They Insurable?* 38 BUS. LAW. (Nov. 1982 forthcoming); *see* Report, at 167-169; "claims-made" policies are not inconsistent with this approach, as the Report intimates; *see* Report, at 168, 176n.66. The Post-Closure Liability Trust Fund could be expanded to cover injuries arising out of accidents which occur during the operational life of a facility but which

manifest themselves only some time after closure and to cover those very few unexpectedly severe accidents whose damages exceed the high policy limits which would be required of facility operators under a "channeling" apportionment rule. *See generally* Light, *The "Long Tail" of Liability: Hazardous Waste Disposal Insurance and the Superfund Act's Post-Closure Liability Trust Fund*, VA.J.NAT.RESOURCES L. (1982 forthcoming).

50. See Report, at 165-166; Bardach & Kagen, *Liability Law and Social Regulation* in Bardach & Kagen, ed. SOCIAL REGULATION: STRATEGIES FOR REFORM 237, 257 (1982) ("The point here is simply that liability law reforms designed to overcome very real costs and obstacles to legitimate claims can quickly spill over into the same kind of overinclusiveness, unreasonableness, contentiousness, and perverse consequences that characterize legalistic direct regulation."); Bickard & Novey, *The Hydra-Headed Common Law of Pollution Control*, 1 ENVIR.FORUM 26 (1982) ("[T]he common law of pollution can shatter . . . settled expectations.").

51. Honabach, *Toxic Torts--Is Strict Liability Really the "Fair and Just" Way to Compensate the Victims?* 16 U.RICH.L.REV. 305, 321 (1982) ("In the long run, strict liability might impact quite adversely upon us.") Priest, *A Theory of Consumer Product Warranty*, 90 YALE L.J. 1297 (1981) (recommending a "duty to warn" theory of product liability less burdensome than strict products liability); Posner, *Strict Liability: A Comment*, 2 J.LEGAL STUDIES 205, 221 (1973) ("Since the efficient use of resources is an important although not always paramount social value, the burden, I suggest, is on [advocates of strict liability] to present reasons why a standard that appears to impose avoidable costs on society should nonetheless be adopted.").

52. In this regard, the Study Group's explicit rejection of market share and enterprise liability concepts is notable. Report, at 247. *See*, Fischer, *Products Liability--An Analysis of Market Share Liability*, 34 VAND.L.REV. 1623, 1662 (1981) ("The adversary system was designed to resolve disputes in an impartial manner, and any attempt to convert it into a compensation system will fail because of the enormous cost to society."); Gillick, *The Essence of Enterprise Liability, or the True Meaning of "We're All in This Together,"* 17 FORUM 979 (1981) ("Bending established tort principles, or fashioning a new remedy to assist injured plaintiffs, will create *a de facto* absolute

liability upon producers of products for injuries with which they may have absolutely no connection."); Note, *An Efficiency Analysis of Vicarious Liability Under the Law of Agency*, 91 YALE L.J. 168, 197-198 (1981) ("If the principal lacked a reasonable monitoring capability . . . [vicarious liability] tends to increase the number of torts, perhaps to the detriment of efficiency, by diluting the agent's incentives for precautionary behavior."); *cf*. Parnell, *Manufacturers of Toxic Substances: Tort Liability and Punitive Damages* 17 FORUM 947, 968 (1982) ("Indeed, United States manufacturers' fear of the socialistic welfare state may soon be replaced with a different dread, that of absolute liability for its hazardous waste byproducts."); *see also* Report, at 124n.74.

53. *See* H.R. 6159, 97th Cong., 2d Sess. (1982) (Risk Analysis Research and Demonstration Act of 1982); *THE RISK ANALYSIS RESEARCH AND DEMONSTRATION ACT OF 1982: HEARING BEFORE THE SUBCOMMITTEE ON SCIENCE, RESEARCH AND TECHNOLOGY OF THE HOUSE COMMITTEE ON SCIENCE AND TECHNOLOGY*, 97th Cong., 1st Sess. (1981); H.R. 6307, 97th Cong., 2d Sess. (1982) (Resource Conservation and Recovery Act of 1982); H.R. Rep. No. 97-570, 97th Cong., 2d Sess. (1982); H.R. 85, 97th Cong., 1st Sess. (1981) (Comprehensive Oil Pollution Liability and Compensation Act); H.R. Rep. No. 97-120, 97th Cong., 1st Sess. (1981); S.1483, 97th Cong., 1st Sess. (1981) (Radiation Exposure Compensation Act of 1981).

H.R. 5735, 97th Cong., 2d Sess. (1982) (Occupational Health Hazards Act of 1982); S.2847, 96th Cong., 2d Sess. (1980) (Asbestos Health Hazards Act of 1980); H.R. 25, 97th Cong., 1st Sess. (1981) (Longshoremen's Act Amendments); S.1182, 97th Cong. 1st Sess. (1981) (Longshoremen's Act Amendments); *cf*. D.C. Code 36-301 *et seq*. (D.C. Workers' Compensation Act of 1979); Postel, *The New Act in Town: An Overview of Workers' Compensation in D.C.*, 6 DISTRICT LAW. 24 (May/June 1982).

H.R. 5261, 97th Cong., 1st Sess. (1981) (Standards for State Product Liability Tort Litigation Act of 1982); S.2631, Product Liability Act, 97th Cong., 2d Sess. (1982) (Senate Committee on Commerce, Science and Transportation); *Product Safety: A New Hot Potato for Congress*, U.S. NEWS & WORLD REP. 62 (Jun. 14, 1982); Shea, *Product Liability: A Continuing Process of Change*, 68 ABA J. 576 (1982).

H.R. 1027, 97th Cong., 1st Sess. (1981) (bill to establish a federal cause of action for and federal court procedures with respect to aviation activity).

APPENDIX

Subsection of Statute	Initial Proof Required	What is Established by the Presumption	1981 Legislative Action*
§ 411(c)(1) 30 U.S.C.A. § 921(c)(1)	10 years exposure and evidence of pneumoconiosis	the pneumoconiosis arose out of the claimant's employment	
§ 411(c)(2) 30 U.S.C.A. § 921(c)(2)	10 years exposure and death from a respirable disease	death was due to pneumoconiosis	eliminated
§ 411(c)(3) 30 U.S.C.A. § 921(c)(3)	chest roentgenogram or biopsy showing large opacities or massive lesions in the lung (i.e., evidence of "complicated" pneumoconiosis)	that total disability or death was due to pneumoconiosis	
§ 411(c)(4) 30 U.S.C.A. § 921(c)(4)	15 years exposure and medical evidence of total disabling respiratory or pulmonary impairment	that total disability or death was due to pneumoconiosis	eliminated
§ 411(c)(5) 30 U.S.C.A. § 921(c)(5)	25 years exposure prior to 1971 and death prior to 1978	survivors entitled to benefits	eliminated

*See 127 Cong. Rec. S 15480 (Dec. 16, 1981).

ATTACHMENT B

TOXIC INJURY COMPENSATION PROPOSALS
Hazardous Waste Bills

General	Section 301 Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Sponsors	None	Stafford/ Randolph	Mitchell/ Randolph	Mitchell/ Randolph	LaFalce	Markey/ Florio
Predecessor Bills or Proposals	----	S.1480, 96th Cong., 2d Sess. (1980)	S.1486, 97th Cong., 1st Sess. (1981); Amendment No. 3629 to FIFRA, 97th Cong., 2d Sess. (1982)	§ 301(e) Study (but only in part)	H.R. 7300, 97th Cong., 2d Sess. (1982)	§ 301(e) Study (with some changes)
Preemption of State Law	No	No	No	Yes1/	Yes1/	No
Federal Cause of Action	No	Yes	Yes	Yes1/	Yes	Yes
Administrative Injury Compensation Plan	Yes	Yes2/	Yes2/	Yes	Yes	Yes

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1/ States ordered to adopt federal substantive law.

2/ Through use of present CERCLA claims procedure against the Superfund.

General (cont.)	Section 301 Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Relationship Between Administrative and Judicial Remedies	Can pursue both, but penalty if recovery in court less than 25% more than Administrative Award[4]	Can pursue both, but Administrative Award is waived by settlement with responsible parties or in certain other situations[3]	Can pursue both, but Administrative Award is waived by settlement with responsible parties or in certain other situations[3]	Can pursue both, but penalty if recovery in court less than 25% more than Administrative Award	Can pursue both but Fund has lien on claimant recovery in court	Can pursue both, but claimant waives judicial review of Administrative Award by bringing court action

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[3] Because of present Superfund Act's § 112.

[4] Minority Advocated Binding Election of Remedies for Claimants

Fund Compensation	Section 301(e) Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Special Compensation Fund	Yes	Expands "Superfund"	Expands "Superfund"	Expands "Superfund"	Yes	Yes
Source of Fund Monies	Tax on Oil & Chemicals, Fees on Hazardous Wastes	Expands "Superfund" Tax	Expands "Superfund" Tax	Expands "Superfund" Tax	Borrows from "Superfund" and Treasury	Tax on Oil & Chemicals, Fees on Hazardous Wastes
Payments from Fund limited to Injuries Associated with Industries Taxed	No	No	No	No	No	No
Preemption of State Taxes to Create Similar Fund	No	Yes[1]	Yes[1]	Yes[1]	No	No
Presumptions of Compensability Upon Exposure	Yes, if substance "known to cause" disease	Yes, if "reasonable likelihood" of causation	No	Yes, if substance "known to cause" disease	Yes, if "reasonable likelihood" of causation[2]	Yes, if "high probability" of causation
Must Claimant Pursue "Responsible Party" Before Claiming Against Fund?	No	Yes	Yes	Yes	No	No

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1/ Through CERCLA § 114(c).

2/ Orders States to apply presumption in worker's compensation programs, also.

Fund Compensation (cont)	Section 301(e) Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Social Insurance Offset Against Recovery	Yes	No	No	Yes	No	Yes
Private Insurance Offset Against Recovery	No	No	No	No	Yes	Yes
Claims Covered by Workmen's Compensation Excluded from System	Yes	No	No	No	Yes	Yes
Recovery Mandated in Statute	Medical earnings, loss (2/3 of salary to $2,000/mo.), death benefits	Economic loss, loss due to injury, natural resources loss, loss of income and profits, medical	Medical expenses, death benefits	Medical, wage loss (2/3 of salary to $2,000/mo.), death benefits	Medical and 80% lost earnings up to $50,000 cap.	Medical, lost earnings (2/3 to $2,000/mo), death benefit
Fund Defenses	Unclear	Unclear, see CERCLA § 112	Unclear, see CERCLA § 112	Unclear, "other causation" findings ruled out.	Admin. Findings of no risk of injury, no certification of "Victim," Assumption of Risk	Assumption of Risk

Cause of Action	Section 301(e) Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Jurisdiction	State only	Federal	Federal	State [applying federal principles]	Federal and State	Federal
Standard of Liability	"Strict" (virtually absolute) 5/	Unclear, uses ambiguous CERCLA standard, analogy to FWPCA § 311	Unclear, uses ambiguous CERCLA standard, analogy to FWPCA § 311	Strict, uses ambiguous CERCLA standard but states that this is "strict"	Unclear, but implication of "fault-based" standard in due-care defense and in Congressional findings [§2(6)]	"Strict" (without regard to fault)
Joint and Several	Yes, except for de minimis contributions	Unclear, does not clarify ambiguous CERCLA issue on this	Unclear, does not clarify ambiguous CERCLA issue on this	Yes, except for de minimis contributions	Unclear, uses CERCLA type language	Yes, except that transporters and generators only liable for time they have substance
Causation Presumptions	No. Use of Presumptions in Administrative System forbidden in tort system. But health effects documents may be evidence	Yes	No	No. But no clear prohibition on use of administrative system's presumptions in court actions	Yes	No. But health effects documents may be evidence

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5/ But minority urged more limited, traditional "balancing" approach to strict liability.

Cause of Action (cont)	Section 301(e) Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Contribution Allowed	Yes	Unclear	Unclear	Unclear	Unclear	Yes
Rules to Apportion Liability Among "Responsible Parties"	Channeling or "alternative methods"	None specified	None specified	None specified	Probably comparative fault	None Specified
Recoverable Damages	Present State Law	Medical, all economic loss, injury to natural resources, loss of real or personal property, loss of income or profits, direct or indirect tax loss by governments	Out-of-pocket medical, loss of natural resources	Unspecified	Medical, loss of real or personal property, damage to natural resources, loss of income or profits, pain and suffering, direct or indirect revenue losses by governments, "Illness, Injury, Death" of any person	Medical, loss of income or profits, Pain and suffering
Subrogation of Fund to Claims	Yes, but "strict liability" not applied for past acts	Yes	Yes	Yes, except for exposure before enactment	Yes	Yes, except for exposures solely before enactment
Statute of Limitations	3 years from discovery of disease and its cause	6 years from discovery of disease and its cause	None Specified	3 years from discovery of disease and its cause	H.R. 2330 (3 years from discovery of disease); H.R. 2482 (3 years from discovery of disease and its cause)	3 years from discovery of disease and hazardous substance cause

Cause of Action (cont)	Section 301(e) Study	S.917	S.945	S.946	H.R. 2330/H.R. 2482	H.R. 2582
Defenses	Not Discussed	Present Superfund Act	Present Superfund Act	Present Superfund Act	Due Care[6/]	Owner/ Operator lack of knowledge of problem or lack of causation, Federal permitted release liability only if negligence present
Punitive Damages Allowed	Yes[7/,8/]	Yes[7/]	Yes[7/]	Yes[7/]	Yes, paid to government under statutory provision[9/]	Yes[7/]
Recovery for "Fear" or "Risk" Without Actual Damage Evidence Possible	Yes[7/]	Yes[7/]	Yes[7/]	Yes[7/]	Yes[7/]	Yes[7/]

6/ Confusing wording.

7/ Under State law.

8/ But one member recommended Federal preemption of punitive damages where federal regulation is comprehensive.

9/ Plus under State law through pendent jurisdiction.

ATTACHMENT C

REPORT AND RECOMMENDATIONS
ON LIABILITY ISSUES IN THE
AREA OF WASTE MANAGEMENT

Including

A Report on the Desirability
of Establishing by Statute
a Standard of Strict Liability

Prepared by

The
Governor's Waste Management Board

I. Introduction and Acknowledgements

The Waste Management Act of 1981 created the Governor's Waste Management Board to perform a number of functions relating to the safe management and disposal of hazardous wastes and low-level radioactive wastes in North Carolina. Among these mandated functions is the following:

> The Board shall study and make recommendations on policy issues including but not limited to liability and financial responsibilities within the waste management area. On or before January 1, 1983, the Board shall prepare and present to the Governor and General Assembly a report concerning the desirability of establishing by statute a standard of strict liability for persons involved in storage, transportation, treatment, or disposal of hazardous or low-level radioactive waste in North Carolina.

To carry out this mandate, the Waste Management Board referred the matter to its Legal Committee for study and recommendations. The Legal Committee is chaired by Charles Holt, former State Representative from Cumberland County. In addition to Representative Holt, the Committee is constituted of the two Board members who are members of the legal profession; Heman Clark, former Superior Court Judge and presently Secretary of the North Carolina Department of Crime Control and Public Safety, and William Graham, former member of the North Carolina Court of Appeals and presently Senior Vice President and General Counsel for Carolina Power and Light. The fourth member of the Committee is Melvin Hearn, Deputy Secretary of the North Carolina Department of Agriculture. Glenn Dunn, Attorney with the Solid and Hazardous Waste Management Branch of the North Carolina Department of Human Resources was the Staff Coordinator for the Committee's study.

The composition of the Committee brought an excellent combination of viewpoints to bear on a subject which demands a blend of policy considerations and technical understanding of a complex area of the law. The Committee requested and received valuable assistance from Robert Byrd, Professor of Torts and former Dean of the School of Law at the University of North Carolina at Chapel Hill.

In addition to Professor Byrd's assistance, the Committee benefitted greatly from the viewpoints and expertise of other persons or organizations. The Committee was fortunate to have had available to it since September 1982, a very thorough Report to Congress entitled Injuries and Damages from Hazardous Wastes - Analysis and Improvements of Legal Remedies. This Report is the culmination of a year-long study conducted by a carefully chosen group of lawyers with a variety of viewpoints and considerable expertise. One member of the group that produced the report was George C. Freeman, Jr., of Hunton and Williams, a law firm in Richmond, Virginia, with an extensive environmental law practice. Mr. Freeman, with considerable assistance from Alfred Light, also of the firm, donated much time and expertise to the efforts of the Legal Committee and attended several meetings.

With the aid of the above-mentioned persons and groups, the Committee reached its recommendations and presented them to the Waste Management Board at a public meeting held in Raleigh on December 1, 1982. Comments were received at that meeting from the following: John Runkle, Conservation Council of North Carolina; Dan Stroh, North Carolina Sierra Club; Wes Hart, North Carolina CATCH; and Cathy Markatos, Tri-County Alliance.

After considerable discussion, the Board accepted the recommendations set forth in the remainder of this report for presentation to the Governor and General Assembly. These recommendations were made only in regard to hazardous waste. The Governor's Waste Management Board will soon file a Supplemental Report addressing whether or not strict liability should apply to low-level radioactive waste.

II. Description of the Hazardous Waste Problem

The discussion of legal issues related to liability for injuries from hazardous wastes is necessarily hampered by inadequate factual and scientific knowledge. At this time it is impossible to determine the potential threat of personal injury and property damage posed by the various activities associated with hazardous waste, either in the nation as a whole or in North Carolina. Furthermore, the Board found itself handicapped by its incomplete understanding of what substances the full range of hazardous wastes includes and how hazardous those substances really are.

Despite this uncertainty, there is justified concern over the health consequences of transporting, treating, storing or disposing of hazardous waste. It is certain that the amount and variety of hazardous waste generated in our society is increasing at a tremendous rate. It is also certain that in the past some of this waste has been handled in an improper manner, particularly in areas of the country that became industrialized earlier than North Carolina. Fortunately, large chemical dumps, such as Love Canal, that have caused major environmental or health problems, did not accumulate in North Carolina twenty or thirty years ago.

Today, however, North Carolina has more than caught up with other parts of the nation in the generation of hazardous industrial by-products.

The textile, furniture, agricultural, paper, and chemical industries that are so important to this state all generate hazardous waste. North Carolina has been ranked eleventh in the nation in generating hazardous waste products. A survey indicates that North Carolina has approximately 700 major hazardous waste generators - i.e. generators that produce more than 2,200 pounds of hazardous waste per month. There are approximately 250 facilities that treat, store or dispose of hazardous waste in this state.

North Carolina also has suffered in recent times from improper disposal of waste such as the illegal dumping of PCB laden oil along more than two hundred miles of roadside.

Despite the increasing problems, this state still has suffered relatively little damage from hazardous waste up to this time. Furthermore, the swift and thorough implementation of a hazardous waste regulatory program should substantially reduce the probability of improper waste handling practices. North Carolina has been among the first states to gain authorization to implement the hazardous waste regulations adopted under the Federal Resource Conservation and Recovery Act (RCRA). The regulations require a "manifest" or tracking system which documents the kind and amount of waste produced by a generator and traces the waste until it is properly rendered non-hazardous or disposed of. The regulations also establish standards for design and performance to be followed in the packaging, transportation, treatment, storage and disposal of hazardous waste. And, of particular importance to the subject of liability for injuries, treatment, storage and disposal facilities are required to carry liability insurance of at least $1,000,000 per incident and $2,000,000 annual aggregate for sudden occurrences, and $3,000,000 per incident and $6,000,000 annual aggregate for non-sudden occurrences.

Despite the safeguards provided by the regulatory program, there is still general public skepticism concerning the safeness of hazardous waste management facilities - a skepticism that crystalizes into awesome resistance in any local area where a facility is proposed. Such resistance has thwarted establishment of many needed facilities nationally, and has been equally as apparent in North Carolina in the vehement local opposition to treatment facilities proposed in Mecklenburg and Guilford counties, and to landfill facilities proposed in Warren and Anson counties.

Yet these facilities are needed. As the General Assembly declared in the Waste Management Act of 1981, the safe management of hazardous wastes, and particularly the timely establishment of adequate facilities for the disposal and management of hazardous wastes are essential to the economic growth and to the public health and safety in North Carolina.

In its deliberations concerning liability issues related to hazardous waste, the Board was compelled by the belief that one of the most important considerations in public acceptance of the necessary waste management facilities in North Carolina is the assurance that persons harmed by the generation, transportation, storage, treatment, and disposal of hazardous waste will be fairly compensated. This realization is supplemented by the conviction that it is more just that the cost of injuries caused by hazardous waste be borne by the handler of the waste rather than the injured person. The costs of compensating for such injuries can be passed on by the handler and reflected in the cost of the service or product he provides. However, it is important that there be some limits to liability; otherwise necessary waste management activities would become uninsurable or, at the very least, the cost of liability insurance would be prohibitively high.

Under the present common law, recovery for injuries caused by hazardous wastes is very difficult for several reasons. The effects of hazardous waste on people and the environment are often subtle and may be delayed for many years. This long latency period makes it difficult for an injured person to trace the cause of the injury, and indeed often acts as an absolute bar to recovery if the statute of limitations has run or the potential defendant has vanished.

The effects of hazardous chemicals are often uncertain and affected by many factors and intervening causes. Proving that a waste caused a particular injury is a highly technical matter requiring expert witnesses and other very expensive technical assistance. Requiring an injured person to prove that the waste was handled negligently as a prerequisite to recovery creates additional difficulty and expense. Indeed, proof of negligence may be impossible when the dangers of the waste are unknown, or when the waste combines with other substances or circumstances to cause injury. In either case the particular injury may be considered unforseeable so that the injured party would be denied compensation. The effect of these difficulties and expenses is that some claims, particularly smaller ones, go uncompensated, often because they are too expensive to pursue.

The justness and political necessity of assuring fair compensation for injuries, combined with the barriers to such compensation under existing tort law, convinced the Board that certain liability reforms should be made by statute. Although case law may be moving in the direction of some of the needed reforms, the progress is uneven and unpredictable. Statutory reforms can add predictability to liability decisions which should be to the advantage of hazardous waste handlers and their insurers, as well as injured persons.

In recommending these reforms, the Board realizes that the existing RCRA definition of regulated hazardous waste may be overly broad and includes some wastes that may not be hazardous enough to justify the recommended liability reforms. Nevertheless, in an effort to ensure a liability system that blends with and complements the ongoing regulatory program, it is recommended that hazardous waste be defined as it is under the state RCRA program. Use of this definition ensures that hazardous waste generators, transporters, treaters, storers and disposers will be easily identifiable and on notice that they are subject to any recommended statutory reforms enacted by the General Assembly.

III. Recommendations

A. Application of Strict Liability for Injuries Caused by Hazardous Waste

RECOMMENDATION 1 -- The Board recommends that strict liability be applied by statute to any generator, transporter, treater, storer, or disposer of hazardous waste in control of the waste at the time it causes injury to a claimant, and that strict liability on the part of a generator continue while the waste is transported and until it is accepted by a properly licensed treatment, storage, or disposal facility.

There seems to be a growing consensus that the common law tort system, based on negligence, fails in ensuring that meritorious small and multiple claims for damage are compensated. This is especially true in highly technical areas such as "toxic torts." The reason for this failure is the high cost of litigation necessary in order to prove negligence, such as expensive expert witnesses and high legal fees for protracted negotiation and litigation. These high "transactional costs" have resulted in a burden on the court system, higher insurance costs, and a reduced percentage of the recovery going to the injured party. Strict liability is seen as a way to reduce these costs and to ensure that injuries are compensated by removing the barrier to plaintiffs of having to prove negligence.

Indeed, common law has long acknowledged that certain activities are so hazardous that no degree of care on the part of the actor is adequate to prevent possible injuries. Persons who engage in such activities are held strictly liable for injuries they cause, and the injured party therefore is not required to prove negligence to be compensated.

Opponents of strict liability claim that it is unfair to hold a person responsible for damages when that person has conducted his activities (in this case, the handling of hazardous waste) in a safe, non-negligent manner. They contend that such an approach will discourage useful economic activity or, at the very least, raise insurance costs substantially. The response to these objections is generally that it is more unfair to leave an injured person uncompensated, and that higher insurance costs will be reflected in an increased cost of conducting the activity -- a cost that reflects the true social costs of the activity.

The range of activities to which strict liability should apply was an important issue for the Board to decide. For regulatory purposes, hazardous waste handling activities are divided into generation, transportation, treatment, storage or disposal. The most comprehensive alternative is to impose strict liability for all of those activities. The RCRA regulatory program, however, distinguishes between hazardous waste generators and hazardous waste management facilities, i.e., treatment, storage and disposal facilities. Generators are not as strictly regulated and are not required to carry liability insurance. Treatment, storage and disposal facilities are heavily regulated because the constant presence of large volumes of waste presumably increases the risk they pose, whereas generators must remove any waste they generate within 90 days. It is particularly important to note that treatment, storage and disposal facilities are required to carry liability insurance in the amounts mentioned on page 4 of this report.

In North Carolina, there are approximately 700 large generators of hazardous waste and many more small generators. There are approximately 250 hazardous waste management facilities, over 225 of which are also generators.

Numerous transporters are involved in transporting hazardous waste. Transporting waste probably exposes more people to risk than any other aspect of handling, although statistics show that accidents are rare. While hazardous waste transporters are subject to relatively strict regulatory requirements imposed both by the Environmental Protection Agency and the Department of Transportation, these requirements do not include mandatory liability insurance. Transporters typically require that shippers or receivers indemnify them for accidents occurring during transport resulting from the hazardous nature of the material transported.

Another key issue is the apportionment of liability among the various activities involved in handling hazardous waste. Existing North Carolina law imposes joint and several liability on defendants whose conduct combines to cause an indivisible injury. Under joint and several liability, a single defendant who makes a substantial contribution to the damage is liable for the entire amount of damages. In order to get complete compensation, a plaintiff has to find and prove liability on the part of only one defendant. That defendant, upon payment of the plaintiff's claim, may seek contribution from any other responsible parties.

Joint and several liability is subject to two related criticisms in the hazardous waste context. One is that it is unjust to hold a person who handles a waste liable for any and all damages when he may have acted more carefully than the other parties involved. The other is that insurance costs will become extremely high if every party in the chain of handling the waste has potential liability for all damages, especially if a strict liability standard is in effect.

The Board's recommendation reflects its conclusion that strict liability will help ensure that one of the barriers to meritorious claims, particularly smaller claims, is removed. Strict liability should also cause handlers of hazardous waste to use the greatest possible care to employ practices that minimize the risk of injury.

Continuing generator liability through the transport stage, a limited application of joint and several liability, helps prevent large claims for damages caused by waste in transport from going uncompensated because the transporter, who is not required to carry a certain minimum amount of liability insurance, has inadequate assets or insurance. On the other hand, under the recommended approach, a generator's strict liability does not continue indefinitely because it is terminated when the waste is accepted at a permitted and regulated treatment, storage, or disposal facility. Unlike transporters, facility operators are required under RCRA to carry liability insurance, and it is therefore less likely that they will be unable to compensate for damages.

Under the recommendation, apportionment of damages where strict liability is imposed would be as follows: a person injured by a waste under control of a generator would seek recovery only from the generator under a standard of strict liability; a person injured by waste in the transport stage could seek recovery from the transporter in control of the waste at the time it caused the injury, from the generator, or from both because they would be jointly and severally liable under a standard of strict liability; a person injured by waste at a treatment, storage, or disposal facility could seek recovery only against the owner or operator of the facility under a standard of strict liability.

The recommendation can be implemented by providing by statute that the person in control of a waste shall be strictly liable for damages caused by that waste; and furthermore, a generator of a hazardous waste shall be strictly liable for any damages caused by that waste, except that it shall be an absolute defense if the generator can show that the waste has been properly delivered to and accepted by a treatment, storage and disposal facility properly permitted to receive the waste.

The recommendation blends well with the regulatory program which, among other things, requires that a generator prepare and keep a written "manifest" for any waste shipped by him. The statute could provide that the manifest gives rise to a presumption that the waste was shipped to and accepted by a permitted facility. This presumption would encourage generators to comply with the manifest system.

RECOMMENDATION 2 -- The Board recommends that strict liability apply only to active facilities and ongoing activities.

Although inactive facilities and abandoned sites have substantial potential to cause injuries, a statutory framework already exists at both the federal level (the so-called "Superfund" law) and the state level (the Oil and Hazardous Substances Pollution Control Act) to deal with these spills. Both laws authorize a fund to be used to clean up spills and restore damaged resources. The State statute also establishes a standard of no-fault (strict) liability for damages to persons or property resulting from a discharge into state waters, which would apply in many cases involving damages due to discharges from abandoned sites. While the present Superfund framework does not provide for recovery of personal injury or private property damages, Congress will consider next year a victim compensation scheme recommended by the study group established by Section 301(e) of that law.

The Board was also concerned that imposing strict liability for past acts raises potential constitutional problems and that insurance policies generally covering those acts might not apply to liability that did not exist when the acts took place.

The recommendation reflects the Board's conclusion that existing statutes requiring responsible parties to clean up inactive facilities should be given a chance to succeed in minimizing potential injuries. It is at best harsh, and at worst unconstitutional, to apply statutorily a standard of strict liability for injuries caused by past acts. In such cases the appropriate common law standard should apply.

RECOMMENDATION 3 -- **The Board recommends that strict liability apply only for injuries resulting from risks associated with the characteristics that make the waste hazardous.**

There is no justification for applying strict liability for injuries caused by the normal activities of a hazardous waste handler if those injuries are not caused by the waste itself. It is, after all, the hazardous nature of the waste that justifies application of strict liability.

The following is a somewhat exaggerated application of this recommendation. A worker at a hazardous waste facility runs over a drum lying near the property boundary and sends some fragments of metal flying that injure a bystander. This injury results from an activity associated with hazardous waste management, but does not relate to the characteristics that make the waste hazardous, and therefore, strict liability would not be imposed. However, if it were a corrosive hazardous waste that squirted from the drum and burned the bystander, the injury would result from the hazardous characteristic of the waste, and strict liability would be imposed.

RECOMMENDATION 4 -- **The Board recommends that a handler of hazardous waste be held strictly liable for all injuries caused by the waste, regardless of whether he knew at the time of injury that the waste could cause that particular type of injury.**

The view that liability should exist for all injuries caused by the waste is supported by the argument that it is better that the person that causes the injury pay for it than the innocent victim. On the other hand, the deterrent value of such a policy is questionable because a person can only take precautions against unknown risks by not acting at all. Such forbearance of action can certainly slow economic and technological progress.

The recommendation is consistent with the goal of emphasizing compensation regardless of fault. The Board emphasizes that this does not mean that strict liability will be applied retroactively in the case of a waste that was not considered hazardous at the time of the act that caused the injury. It only applied to an unknown effect from a waste known to be hazardous at the time it causes the injury.

RECOMMENDATION 5 -- The Board recommends that strict liability apply to successors in interest as well as to the generator, transporter, treater, storer or disposer in control of the waste at the time it causes the injury.

To impose liability to successors in interest has the effect of increasing the parties from whom compensation might be sought, thus increasing the possibility that an injured person will be compensated. The former owner of a facility, for instance, should not, by selling the property, be able to relieve himself of liability for injuries caused by conditions he created.

This recommendation would help avoid cases where an injured person is unable to recover damages because the company that created the hazard has passed out of existence or sold a site with a latent hazardous waste problem to another person. In such cases, the new owner may be the only person able to compensate the injured person. Owners of facilities are required to record on the deed that the site has been used as a hazardous waste facility. Therefore, subsequent purchasers should be on notice and have full opportunity to assess the risks they may be acquiring.

RECOMMENDATION 6 -- The Board recommends that punitive damages not be allowed for claims where strict liability is applied.

Damages in tort claims can be generally categorized as compensatory damages such as medical bills, lost property value, lost wages, pain and suffering, mental anguish or similar intangible injuries; and punitive or exemplary damages. The first category focuses on compensating the injured person, but the second focuses on punishing the liable person. Punitive damages are awarded only if the defendant's conduct which causes the damage is intentional, reckless, or in wanton disregard of the safety of others.

The justification for punitive damages is questionable for activities such as waste management that are regulated by a comprehensive statutory scheme which includes criminal and civil penalties for violations of such regulations. Such regulations set the standards for the regulated activities, and the statutory penalties should be considered the exclusive means for punishing violators.

Another reason for not allowing punitive damages is that large punitive damage awards made to initial claimants may leave inadequate funds (or insurance) to compensate others who are injured. Such a possibility becomes more likely when multiple claims arise from the same action. The resulting exhaustion of funds is incompatible with the goal of ensuring that as many injured parties as possible are compensated for their actual injuries.

On the other hand, it is argued that regulatory programs generally are not adequately enforced and penalties are not large enough to deter reprehensible waste management practices, and therefore, the threat of punitive damages is a necessary additional deterrent. However, citizen suit provisions under federal and state statutes permit private initiation of enforcement activities in some cases.

The Board concluded that disallowing punitive damages is consistent with the goal of compensating claimants for injuries without consideration of fault on the part of the defendant, and may help avoid extremely large recoveries based on punitive damages that render a defendant financially unable to compensate other injured persons. Sufficient remedies are provided by state and federal statutes in the form of criminal and administrative penalties for violations by hazardous waste handlers.

RECOMMENDATION 7 -- The Board recommends that although strict liability is imposed, contributory negligence of a claimant will bar any recovery by that claimant.

Under negligence law, a claimant cannot recover damages if his own negligent acts contribute to his injury. However, where strict liability is imposed, recovery is not prevented by ordinary contributory negligence, but will be prevented if the claimant voluntarily and knowingly exposed himself to the risk. The Board concluded that an injured person should not be able to recover, even where strict liability is imposed, if his own negligent acts are an essential cause of the injury.

B. Statute of Limitations

RECOMMENDATION 8 -- The Board recommends that the statute of limitations begin to run only when the injury becomes apparent or ought to become apparent to an injured person, and that the ten-year statute of repose not apply.

The purposes of statutes of limitations are practical ones: (1) to ensure that legal actions are not initiated so long after the time of the acts alleged to have caused the damage that the facts necessary to litigating the matter have "grown cold" (i.e., are no longer capable of accurate determination), and (2) to ensure that persons are not subjected indefinitely to the threat of liability for past actions.

When strict liability is applied, the first reason becomes less important because there is no need to recall facts relevant to whether the defendant's acts were negligent. The main issue of fact becomes whether the hazardous waste under the control of the defendant caused the injury, an issue based more on scientific data than on facts relating to the defendant's actions. Furthermore, statutes of limitations have no deterrent value and in the case of injuries with long latency periods, they provide a shield for waste handlers.

Commencement of the running of the statute of limitations at the time of the defendant's act can be an unfair barrier to recovery for injuries caused by hazardous wastes. Many of the effects of chemical wastes may not manifest themselves for many years. This same problem exists in relation to many other types of injuries in our society today. The fairness of statutes of limitation that run from the time of the act of the defendant has been challenged and most states have statutes that run from the time of "discovery" of the injury by the injured party.

North Carolina's statute [N.C.G.S. 1-52(16)] presently provides that an action must be brought within 3 years "from the time that the injury or damage becomes apparent or ought reasonably to have become apparent to the claimant," but then provides an absolute bar, known as a statute of repose, against any action brought "more than 10 years from the last act or omission of the defendant giving rise to the cause of action." Thus, in North Carolina an injured party has three years after the discovery of the injury in which to initiate an action for recovery; however, no action may be brought after ten years under any circumstances.

The goal of facilitating compensation of injured persons outweighs the practical advantages of putting potential legal actions to rest after a certain period of time. This is especially important in cases where the condition causing the damage may develop very slowly (for example, a buried toxic waste may take many years to reach adjacent water supplies) and where the injury from the condition may not manifest itself for many more years.

C. Causation and Related Evidentiary Requirements

RECOMMENDATION 9 -- The Board recommends that there be no attempt to ease the plaintiff's burden of proving that the injury or damage was caused by the hazardous waste handling activity of the defendant.

As the law presently stands, a plaintiff must prove in each case that there is a causal connection between the disease or injury complained of and the hazardous waste related activity of the defendant. Proof of this causal connection is usually difficult in these cases for several reasons.

First, a plaintiff must show the nature of the exposure - whether by inhalation, ingestion or other contact - in order to show the duration, frequency and intensity of exposure. If exposure is adequately proven, proof of causation will usually require large amounts of sophisticated and thus very expensive, medical and scientific testimony to demonstrate the epidemiologic or statistical correlation between the hazardous waste(s) in question and the injury or disease.

Additional problems of proof stem from the fact that there is often a long latency period before effects of exposure become obvious, and during that period the injured party may have been exposed to a wide variety of substances from other sources. Furthermore, a facility alleged to cause the injury may have handled many types of waste which may interact to cause effects that none of them would cause alone, making it more difficult to determine the exact cause of the injury.

Finally, medical science has simply not been able to determine the effects of many chemicals already on the market and, of course, even less will be known at any given time concerning the effects of new chemicals that enter the market constantly.

The difficulties in proving causation have been eased in Workers Compensation Laws by a "rebuttable presumption" approach.

Under this approach, if the plaintiff can prove that he suffers from certain types of injuries and that he has had a certain amount of exposure to the substance in question that is known to cause those injuries, the burden shifts to the defendant to prove that the substances within his control did not cause the injury.

This "rebuttable presumption" approach is considered fair because a substantial body of information exists concerning certain substances (for example, asbestos) to show a strong statistical correlation between exposure to the substance and the subsequent development of certain diseases [for example, in the case of asbestos, mesothelioma (lung cancer)]. This is particularly valid and useful in worker's compensation cases because employees are exposed day-in and day-out to a known set of substances.

However, in the case of injuries allegedly stemming from hazardous wastes, the plaintiff will less often be subject to constant exposure over a long period of time and the exposure is likely to be to a greater variety of substances or, for that matter, many unknown substances. This should become even more the case now that strict regulatory programs are in effect which tend to isolate the waste in closely controlled facilities. Thus a presumption of causation in the case of alleged injuries from hazardous waste is probably not as valid or fair as in workplace injury cases. Even in the workplace injury context, however, there have been abuses. For example, Congress in 1981, felt it necessary to abolish several statutory "rebuttable presumptions" in the Black Lung Benefits program because many successful claimants under the program did not have adequate medical evidence of black lung disease.

There is at present insufficient indication that there will be a large number of cases alleging injuries from similar types of wastes, which is the circumstance that justifies a presumption of causation. At present, it seems likely that claims for injuries will involve a wide variety of wastes, and that causation must be verified on a case-by-case basis.

D. Administrative Mechanisms for Determining Liability and Compensating Injured Persons

RECOMMENDATION 10 - The Board does not recommend at this time that an administrative agency be created to adjudicate strict liability in hazardous waste cases.

There are two key factors to consider in deciding whether an agency rather than the courts should adjudicate liability. (1) Will there be enough claims to justify establishing a separate system? and (2) Are the cases likely to be so complicated that special judges or panels are needed to resolve them?

Concerning the first question, there is little actual proof yet that there will be a great increase in toxic tort cases, although it is logical to assume that there will be at least some increase due to the proliferation of chemicals in the last 20-25 years and the typically long time it takes to discover an effect. Furthermore, the implementation of a very stringent regulatory program should reduce the potential for injuries. At this point it seems premature to assume that the case load will justify a separate system.

Concerning the second question, the complexity of these cases will certainly be reduced if strict liability is applied, although the remaining issues of causation are highly technical and the use of administrative law judges with particular expertise might promote more consistent decisions.

The Board concluded that it is premature at this time to assume that the volume or complexity of hazardous waste liability cases will be so great that the general court system cannot handle them adequately.

RECOMMENDATION 11 - The Board does not recommend at this time that a fund be established for compensating injured claimants.

The advantage of a fund is that it would ensure that the money is available to compensate a person with a meritorious claim, that the compensation could be immediate and that identification of a specific responsible party would not be necessary. However, the fact that most of the facilities are required to have liability insurance for precisely this purpose certainly reduces the need for a fund. Furthermore, use of a fund entails some administrative costs that will increase the "transactional" costs.

If compensation is made from a fund rather than by the responsible party, then compensation is divorced from responsibility and has no deterrent value, unless the agency administering the fund is empowered to seek recovery from the reponsible person(s). Such a process has potential to increase even further the "transactional" costs. A fund would appear more appropriate in the abandoned or inactive site context, where the identity of a potentially responsible party may be unknown. This is the area Congress will be studying closely in connection with the report required by Section 301(e) of the federal Superfund law. California has adopted an "orphan site" fund to compensate those injured by abandoned sites, but that state is apparently the only one which has found such a system necessary.

Furthermore, any attempt to establish a fund would entail difficult problems of deciding who should provide the money for the fund, whereas direct compensation by the responsible defendant places financial responsibility where it should be. Mandatory liability insurance requirements should be adequate to ensure that the defendant will be able to provide compensation in most cases.

ATTACHMENT D

Superfunds and Tort Law Reforms—Are They Insurable?

By RICHARD A. SCHMALZ*

I

The "Superfund" bill enacted in 1980[1] is a federal law that imposes strict liability on certain classes of persons for cleanup costs and environmental harm resulting from the handling or disposal of hazardous wastes. It is the most recent in a series of federal statutes that promise to become the springboard for major tort law changes. Even without the superfunds, there has been considerable interest in tort law reform within academic circles, and the highest courts of many states seem eager to make their own marks in reshaping tort law according to their own views of public policy, but federal legislation can accomplish deeper changes more quickly than a dozen law review articles or judicial decisions, so superfunds and the tort law reform movement will feed on each other with a synergistic effect.

But are superfunds and tort law reforms insurable? The question is timely because a number of studies are under way, among them the study directed by the 1980 Act itself, a separate study by the Rand Corporation under the auspices of the Institute for Civil Justice, and other studies by various American Bar Association committees. Since studies rarely find the present state of affairs satisfactory, we can expect recommendations for affirmative action to correct the deficiencies as their authors see them.

Are superfunds and tort law reforms insurable? My answer is a cautious and qualified yes. The discussion which follows reflects the perspective of my thirty-five-year association with the property and casualty insurance business. Quite naturally, then, I raise points for consideration mainly from an insurance standpoint, but one should not overlook the broader social and economic aspects.

Tort law reform is inevitable. The only questions really open to debate are how it should be accomplished and how sweeping it should be. I believe a federal statute is the only feasible method of achieving the kind and scope of tort law reforms that are likely to remain insurable in private markets. I outline an entirely nonoriginal federal statutory system that I believe would enable private insurance and reinsurance markets to respond to the very high

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[1] Comprehensive Environmental Response, Compensation, and Liability Act of 1980. Pub. L. No. 96-510, 94 Stat. 2767.

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limits of third-party liability insurance needed to support the concept of strict liability. Let us start with the Superfund bill in its 1980 version.

II

In some of the earlier proposals for superfund legislation, strict liability was also imposed for bodily injury and property damage to third persons. As finally enacted, however, Superfund does not include this feature, restricting itself to cleanup costs and environmental harm. Instead, Congress directed a study to be conducted with the assistance of the American Bar Association, the American Law Institute, the Association of American Trial Lawyers, and the National Association of State Attorneys General. The purpose of the study is to help Congress determine the adequacy of existing common law and statutory remedies in providing legal redress for harm to man and the environment caused by the release of hazardous substances. Accordingly, the study goes well beyond the scope of Superfund, as enacted, or its predecessor federal statutes dealing with clean air, clean water, and oil pollution.

The study group has been directed to submit its report to Congress with appropriate recommendations. The recommendations are to address explicitly the need for revisions in existing statutory or common law, and whether they should take the form of federal statutes or the development of a model code recommended for adoption by the states.

III

There is a certain equilibrium between tort liability-compensation principles and the underlying price structure of goods and products produced by a private enterprise economy. If that equilibrium is disturbed substantially, it may have an effect far beyond that which is desirable from a broader point of view.

Since the potential for disturbing the present equilibrium is great, it is necessary to start at the beginning and reexamine the premises on which a tort liability-compensation system may soundly be based under modern economic and social systems.

IV

Two basic general concepts apply to injury arising out of private or commercial activities:

(A) For every injury there must be compensation, regardless of the circumstances. The source of compensation is of secondary importance.

(B) A loss should be borne by the one who suffers it unless there is a good reason to shift it to another. Loss shifting requires justification. It is the exception, not the rule.[2]

Legal systems generally start from concept B. Concept A is by nature a broad social imperative which does not offer much as a means of resolving the competing claims of individuals.

Concept A implies compensation without fault. It serves as the model for a pure no-fault compensation system. Concept B implies some limitation on eligibility for compensation. There must be a good reason for the loss shifting. Let us call this good reason fault or wrong or tort. Concept B then becomes the model for a tort liability system.

Concept B carries with it a conscious choice not to use it or regard it as a means of compensating every injury. Therefore, it should not be a valid criticism of a tort liability system that some injuries or classes of injuries do not qualify for compensation. The chief exclusions are, almost by definition, those cases where there has been no fault in the legal sense and where, if there is such fault, there has been no causal connection between the fault and the injury in a mixed legal and physical sense.

These are the areas subject to the most pressure from critics of the tort liability-compensation system. A process that continuously relaxes the eligibility requirements for tort liability compensation leads to concept A.

As a general rule, the tort liability compensation system in the United States seeks to provide full damages for injury once the necessary fault and causal connection can be found. This requires putting a monetary value on all injuries, difficult though this may be. For bodily injuries, damages include compensation for medical expenses, lost earning capacity, and pain and suffering. The last element is hard to measure by objective standards and yet, in theory and practice, often overshadows the purely economic losses.

No large modern state has fully adopted concept A in a pure form. The administrative burdens and costs for a large state would be so enormous that such a concept is perhaps untenable for those reasons alone. Concept A has been adopted, in greatly modified form, as a part of legal systems. The main examples that come to mind with respect to the United States are workers' compensation and no-fault automobile insurance systems. Both are third-party liability systems that deal almost entirely with bodily injuries rather than property damage.

In each of these cases where the fault eligibility rule has been abandoned, it has been necessary to set the amount of compensation payable substantially below the level of full tort damages. Otherwise the total cost of the system would rise to unmanageable heights. An initial balancing principle emerges.

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[2] Pfennigstorf, *Environment, Damages, and Compensation*, 1979 Am. Bar Found. Research J. 347, 363.

As we move from the concept of fault to no-fault, we must be prepared to accept lower compensation levels or risk unmanageable costs.

Neither workers' compensation nor automobile no-fault systems have eliminated the second important eligibility requirement of a liability system: the causal connection between the third party's activities and the bodily injury. For no-fault automobile insurance the third party's automobile must be involved in the accident and the accident must be the cause of the claimant's injury. In workers' compensation cases the injury must arise out of and in the course of a worker's employment. For most automobile and industrial accidents causal connection is rarely a problem. In workers' compensation occupational disease claims, however, the causal connection between work-related exposure and injury is often hard to establish with certainty.

There has been a trend toward easing the causal connection burden under workers' compensation laws in occupational disease cases. This has resulted in higher workers' compensation insurance costs. An extreme instance is the relaxation of burdens of proof with respect to black lung disease together with the imposition of retroactive liability. The cost explosion these have brought about has caused serious insurance problems and produced a system which may be running out of control from a financial standpoint.

This brings up a second balancing principle. *If we relax the causal connection requirement for a tort liability system, we risk unmanageable costs even if benefits are sharply reduced below full tort compensation levels.*

To achieve a purpose other than merely compensating every injury, the system must be rational and predictable on both the eligibility for and the amount of compensation. If it appears to be arbitrary or capricious, no message emerges by which individuals can be guided to act in accord with the system's purpose.

Interestingly, the condition of predictability is also essential for a system of private insurance operating under market pricing. When insurance is sold under competitive conditions to individual risks, the losses of each risk or group of risks must be predictable in order to determine an appropriate premium charge. When losses for any insured or group of insureds become unpredictable due to changes in the tort liability system, an insurance crisis usually follows. The recent crises in medical malpractice and products liability insurance are prime examples.

Critics are quick to express dissatisfaction with the tort liability system: it does not compensate every injury; at times it imposes difficult burdens of proof on those seeking to shift losses to others; and it has trouble dealing with the causal relation between certain exposures to hazardous substances and late-emerging injuries. A quick reaction is to set about curing these defects by imposing strict liability and by creating presumptions of a causal connection that cannot be rebutted any more convincingly than the connection could be proved directly. But such a course undercuts the rationale of the tort liability system and its predictability and risks unmanageable costs and insurance crises.

V

The ideal foundation for a workable third-party liability system would be a sound, generally accepted definition of: (1) its purpose; (2) the reasons why it is better to shift losses to some third parties than to leave them on the injured person (or the injured person's insurer); (3) who these third parties are; and (4) the circumstances under which the loss shifting is to take place. Such a definition would enhance predictability and promote efficiency from an economic viewpoint. It is not surprising, then, that several American scholars in the legal and economic disciplines have been devoting considerable effort in recent years to developing a comprehensive theory of accident law based heavily on economic theory.[3]

This approach suggests that the major purpose of liability rules should be to reduce the costs of accidents, including the costs of avoiding accidents. The persons liable for accidents would be the cheapest cost avoiders (i.e., they would be in the best position to make a cost-benefit analysis between accident costs and accident-avoidance costs and to take appropriate action). Making such a determination involves applying economic efficiency and utility theories.[4]

Not all legal and economic scholars who have studied the matter believe that it is practical or sound, even theoretically, to base tort liability-compensation rules primarily on promoting an economically efficient allocation of society's resources. Cost-benefit calculations and interpersonal welfare comparisons are difficult, if not impossible, to make with much confidence in most cases.

England, a lawyer, notes that "[c]areful behavior may be cheap in itself, but the effective avoidance of all the small human errors would probably exceed the totality of society's resources. A truthful cost-benefit calculus of total accident avoidance would therefore in most cases leave the loss where it falls."[5]

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[3] Perhaps the prime mover in this effort is Guido Calabresi, who has written extensively on this subject. His major work, The Costs of Accidents: A Legal and Economic Analysis (1970), is well known. Richard Posner has developed an economic theory of negligence and contributed many scholarly papers. The Journal of Legal Studies, published by the University of Chicago Law School, is replete with articles devoted to an economic analysis of the law. The three-part series of volume 9 (Jan., Mar., and June 1980) is especially useful in gaining a perspective on the state of American legal and economic scholarship in this area.

[4] For a discussion of some conceptual problems and inconsistencies, see Dworkin, *Is Wealth a Value?*, 9 J. Legal Stud. 191 (1980).

> Utilitarianism supposes that individual welfare levels are at least sometimes comparable, so that total or average utility levels can be ordered over various choices of social programs. Economists, as a group, have been skeptical about interpersonal comparisons of utility. If utilitarianism is to be the motor of wealth maximization, then wealth maximizers must forgo that skepticism and move even further from present orthodoxy in economics.

Id. at 216.

Rizzo, an economist, concludes that

> [e]fficiency, as normally understood, is impossible as a goal for tort law. The law cannot and should not aim toward the impossible. . . . Precisely because we live outside of general competitive equilibrium and in a world of unpredictable flux, the efficiency case for negligence must fail. In such a world it is impossible to compare alternative liability systems, in terms of judicial cost-benefit analysis or "fine tuning."[6]

Some forty years ago, Prosser clearly perceived the social engineering aspect of tort law, writing in his classic *Hornbook on Torts:* "Perhaps more than any other branch of the law, the law of torts is a battleground of social theory."[7]

Twenty-five years ago Harper and James observed that

> [i]n the long run, the law tends to respond to its function and there are strong pressures at work toward making accident law serve at least some of the objectives of social insurance. It is in a transitional stage, where professed doctrine and purely logical reasoning from it will not accurately reflect the course of decisions or furnish a safe guide to what courts will do in the future. However little one may be concerned with law reform he cannot understand what is actually happening to the rules in this branch of law today unless he appreciates the trend towards social insurance and the relationship between this trend and the evergrowing institution of liability insurance.[8]

The process of transition has not slowed down. Englard has more recently (1980) noted:

> In general, the cause of the present crisis can be ascribed to a shifting in the aims and functions of tort law. Traditional tort law has been concerned with redressing a certain wrong committed by the tort feasor towards his victim. It is "corrective justice" between two parties based on the concept of fault with a clear moral connotation: the blameworthy injurer must compensate the innocent victim.
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[5] Englard, *The System Builders: A Critical Appraisal of Modern American Tort Theory*, 9 J. Legal Stud. 27, 55 (1980).

[6] Rizzo, *Law Amid Flux: The Economics of Negligence and Strict Liability in Tort*, 9 J. Legal Stud. 291 (1980).

[7] W. Prosser, The Law of Torts 15 (1941).

[8] F. Harper & F. James, The Law of Torts 784 (1956).

> The growing number of accidents created social pressure for the compensation of the victims, which was facilitated by liability insurance. The consequent penetration of distributive goals undermined the deterrent and retributive function of tort law. The traditional concept of fault itself underwent changes. The persistent tendency towards distributive aims weakened the idea of moral culpability by objectifying the elements of fault and causation.
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> ....
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> The acute crisis of conflicting aims has been aggravated by the fact that the dominant elements of loss spreading have developed outside the framework of traditional tort law. In particular, the systems of social and private insurance have been created quite independently from specific rules of liability.
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> The traditional corrective-justice pattern of tort law has proved to be universally inadequate to overcome this conflict of aims and functions. One has only to consider the frequent case where both tort feasor and victim are covered by insurance. The whole tort process in which the court painstakingly labors to elucidate the minute facts of the damaging occurrence appears futile in view of the final allocation of the loss. The actual result of the trial may simply be a mode of accounting between two big insurance agencies. It is little wonder that modern tort law has been described as "schizophrenic, at cross-purposes."[9]

These observations show that, unfortunately, there is not much chance of identifying any single unambiguous, universally accepted purpose underlying the tort liability system. The emphasis will continue to shift toward concept A with less and less importance attached to individual responsibility or fault. Several legal theories now allow a plaintiff to avoid the burden of proving that an individual firm caused harm if the plaintiff can show that the firm is one of many firms marketing essentially the same product. Strict liability is favored over negligence by several scholars.[10] Some would argue against allowing even contributory negligence as a defense on the claim that it promotes economic efficiency,[11] although others do not agree.[12]

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[9] Englard, *supra* note 5, at 27-29.

[10] *E.g.*, Epstein, *A Theory of Strict Liability*, 2 J. Legal Stud. 151 (1973). But note a partial retreat for products liability, R. Epstein, Modern Products Liability Law 28 (1980):

> The arguments just made on behalf of strict liability, even if conclusive in cases involving strangers, to which they apply, are therefore of but limited relevance in products liability situations. They say in effect that strict liability and not negligence should govern whenever an innocent bystander is injured by a defective product made and marketed by the defendant. These same arguments are, however, of no particular relevance to the vast bulk of products liability actions brought by purchasers, users, and consumers of particular products.

Striking a fair and reasonable balance between emphasizing compensation and retaining the concept of individual fault in the traditional sense requires thoughtful consideration of important public policy questions. As we move from concept B (the fault-based corrective-justice system) toward concept A (the pure no-fault compensation system), the scarce resources available to society will be reallocated in different dimensions and directions. American goods and services will cost consumers more, and American industry may become less able to meet foreign competition unless compensation levels are lowered from present tort standards and the distribution costs of the tort system are improved.

One implication of this shift is that lawyers will become less and less involved in pressing accident claims. The system will become more automatic, more like social security. Another is that general damages will have to be reduced or eliminated. All this will bring forth strong opposition from the bar. No doubt claims will be made by some advocates of change that in addition to providing more adequate and more just compensation, society will benefit greatly by a stronger impetus toward accident prevention and "internalization" of costs. These claims are window dressing. The truth is that the more the concept of individual fault is eroded, the less effective the transfer of loss is as a spur to accident-proof conduct; the weaker the causal connection between a tort feasor's activities and an injury that has many possible causes, the greater is the chance that somebody else's costs are being "externalized."

The policy questions involved in an accelerated shift toward more liberal tort liability rules need a solid economic analysis, although perhaps not along the same lines taken by the economic school of tort liability reformers mentioned earlier. I believe that many of these reformers have overemphasized the ability of the tort system to deter unsafe conduct as it evolves to strict liability and more relaxed compensation rules. In any event, it appears that they have not carried the analysis far enough in relation to the ability of society to afford full tort compensation under such conditions.

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11. *See* Rizzo, *supra* note 6, at 317. Rizzo, however, backs into this position. He demonstrates that it is impossible to make the cost benefit analyses needed to determine efficient liability assignment under negligence and foreseeability tests. He prefers strict liability because it is simpler and results in "greater certainty about the locus of responsibility for accidents. This greater certainty promotes efficiency in the basic institutional sense because property rights, in effect, become more clearly or definitely defined." Rizzo, as he of course intended, has abandoned the classic definition of economic efficiency and substituted a new concept of institutional efficiency.

12. Rubin, in *Predictability and the Economic Approach to Law: A Comment on Rizzo*, 9 J. Legal Stud. 319, disputes Rizzo's claim for increased efficiency, even as Rizzo has redefined it, concluding at 334: "Adding a non-economic standard of liability, as proposed by Epstein and defended by Rizzo, would also substantially add to decision-making costs." See also Shavell, *Strict Liability versus Negligence*, 9 J. Legal Stud. 7, n.11 (1980): "It is of course clear that under strict liability without the defense the outcome is inefficient, for victims would have no motive to take care."

Any compensation system is only as good as the financial resources behind it. Heavy reliance will be placed on some form of private or government insurance. If the present mood in Washington continues, there will be more interest in private solutions to the problem of making sure that adequate security is in place. If costs of the tort system escalate, the private solutions may look more like government-sponsored quasi-private funds drawing their resources from special assessments or fees.

Accordingly neither the insurance industry nor industry as a whole can overlook the importance of the economic analysis suggested above. One must ask what kind of a system is rational for America, and in a changing world what kind of system will maximize the development of efficient private insurance markets to support it.

VI

The private insurance market in the United States has developed two kinds of insurance to accommodate the traditional fault-based, corrective-justice tort system. This is the system that applies to almost all commercial activities. For those cases where the loss is shifted from the victim to a tort feasor, third-party liability insurance is available through insurance policies issued to each firm. The policies indemnify against legal liability. Loss is shifted initially from the firm to the insurance company. The insurer in turn shifts it to its other policyholders, reinsurers, and stockholders. Unless the firm is a very small one, however, the firm's own loss experience will have a marked, direct effect on the firm's insurance premiums under the usual experience rating, retrospective rating, and dividend plans applicable to commercial liability insurance. The larger the firm is, the more likely it is that over time its own losses will determine its own premiums.

The wide popularity of experience rating, retrospective rating, dividend, and cash-flow plans demonstrates that firms are almost universally interested in insurance that reflects their own experience as individual productive units whenever that experience measures their true loss potential reasonably well. The cost of at-fault accidents is, then, largely internalized, often even to individual firms, but if not that specifically, at least to rating classes made up of firms conducting similar activities. In the main, group liability insurance policies have not become widely popular.

For those cases where traditional tort principles do not shift the loss from the victim, first-party coverage is widely available through individual policies issued to victims or to their employers.[13] Losses covered include physical

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[13] 13. The Interagency Task Force on the Health Effects of Ionizing Radiation, in the Report of the Work Group on Care and Benefits (June 1979), notes at 34 that:

> Many persons have coverage under a private health insurance plan which pays benefits regardless of an injury's cause. In 1975, an estimated 168 million persons or nearly 80 percent of the population had coverage for hospital care, and a majority had some protection

damage to homes and business property, vehicles, death, disability, hospitalization, medical expenses, and so forth. In this area, group policies are more common and there is less tendency to differentiate finely among the loss potential of individual victims.

A possible partial explanation of the wider use of group policies is that fault is not a consideration in the determination of benefit eligibility. Losses are, therefore, shifted from the individual to larger, less refined rating classes. The compensation available through first-party coverage is generally restricted to economic loss, except for life and accident insurance, and often has sublimits on benefits for medical expenses or work loss to discourage overutilization.

I do not intend to suggest that the combined third-party and first-party systems do not have any gaps that leave some victims without compensation for some injuries or losses. I wish mainly to outline the general scope and nature of the two forms of private insurance that have evolved to accommodate the traditional concept B approach to tort law loss shifting.

The outline points out, however, that the present practice of issuing third-party liability insurance to individual firms under various experience rating plans supports the corrective-justice and accident-deterrence rationales of a tort liability system. And the nature of first-party insurance, with its generally more-limited approach to compensation levels, suggests that if one tries to fill all gaps in the total compensation scheme by expanding eligibility under the tort system, then the possibility of an unmanageable cost explosion becomes real indeed. The tort system has much higher frictional costs and compensation levels than first-party insurance systems.

The private insurance system has been able to accommodate strict liability as a substitute for negligence, and the weakening of certain defenses such as contributory negligence, when there is a clear causal connection been a firm's activities and an accident. For example, aircraft accidents can be dealt with on a strict liability basis without eroding the corrective-justice or accident-deterrence goals of a tort liability system. In this case, the predictability of aircraft accidents and the proper allocation of losses to a single firm is not undercut by a strict liability rule.

The insurance system begins to have serious problems when strict liability is imposed on numerous firms at the same time for the same injury, especially when the injury cannot be causally linked to any particular policyholder, time period, or industrial activity. The chaotic situation with respect to liability and insurance coverage for asbestos-related injuries is a prime example of how marked shifts away from the requirement of clearly

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against the cost of physician's services, surgical care, and prescribed drugs. Most of the growth of private health insurance has been in employee group plans. However, approximately 18 million people do not have any health insurance or access to free care, and more than one-half of all Americans do not have adequate health insurance coverage for the catastrophic expenses which may result from cancer.

identified individual fault and causation as the basis for liability can create havoc for an insurance and industrial system geared to those principles.

The case for moving the tort liability rules for hazardous wastes even closer to liability without fault or specific causation rests largely on characteristics of the disposal of chemical wastes, which are thought to be analogous to the handling of asbestos. There are many firms that have been engaged for many years in producing industrial wastes. Injuries often become manifest long after exposure. And, as far as we know, the injuries may just as well result from natural or other unidentified causes. At best there is a statistical indication of an increased frequency of injury over the rates observed under other conditions.

It can be expected, then, that a general easing of the loss-shifting burden with respect to hazardous wastes will have the same effects on industry and insurance as those we are seeing in asbestos and black lung cases unless great care is taken to control costs and minimize impediments to a sound private insurance system. I believe there are some steps that can be taken to do this, without passing judgment on the wisdom of expanding tort liability remedies for hazardous waste disposal activities.

VII

If a tort liability system is to impose strict liability and still retain any appreciable element of its historical corrective-justice or accident-deterrence function, it should place strict liability on one and only one firm at a time. And where one firm is to be held strictly liable that firm should be the only firm liable to the injured persons.

Spreading strict liability over every firm forever cannot possibly encourage economic efficiency.[14] Nor can an insurance market respond with peak efficiency to such a condition, or to a condition where one firm is strictly liable at one time but all other firms remain liable on some other ground forever.[15]

Fortunately, we do not have to create a new tort liability-insurance system out of whole cloth. There are some well-conceived models designed to maximize insurance capacity under the rules of strict or absolute liability. These models arose in the late 1950s and the early 1960s to deal with the production and use of atomic energy on a wide scale for peaceful purposes.

In the United States we are more familiar with the Price-Anderson Act of

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[14] There is a problem of plurality here. Not every firm at all times can be "the cheapest cost avoider." Note also Shavell, *supra* note 12, at 465: "Under strict liability it is not hard to imagine circumstances where a party decides against engaging in an activity when it would have been socially worthwhile for him to have gone ahead."

[15] This seems obvious to those in the insurance business, but the point has also been recognized by outsiders. Note Pfennigstorf, *supra* note 2, at 419: "The growing tendency to impose strict liability for large-scale industrial pollution has greatly increased the demand for liability insurance protection but has also multiplied the problems of evaluating the risk exposure, defining reasonable limits for the coverages, and calculating adequate prices."

1957.[16] The Paris Convention of 1960 and the Vienna Convention of 1963 covered similar ground. All have common features that would be useful in treating the liability and insurance problems associated with hazardous wastes. All are designed for risks of an exceptional character for which common law rules and practices are not suitable.

First of all, *they are not general purpose systems*. They focus only on the *exceptional risks* associated with a certain type of industrial activity. In their case, the activity is the production and use of nuclear material, including the supply of parts, materials, and equipment; the transportation of nuclear materials; and the treatment, processing, or disposal of nuclear wastes.

Whenever risks, even those associated with nuclear activities, could properly be dealt with through existing legal processes, they were left outside the system. This feature, which was adopted both in the United States and in Europe, requires the identification and separation of the low-hazard activities from the high-hazard risks. The separation has actually been made on several bases, such as the type of production or utilization facility involved, the nature of the particular activity, the quantity and type of nuclear material at hand, and the seriousness of the consequences of an accident.

While there was considerable philosophical debate in some quarters about the appropriateness of a "threshold" concept, the best judgment of the many experts here and abroad who considered the insurance and liability problems was that a reasonable threshold is required as a balance to the imposition of strict or absolute liability and the expanded eligibility for compensation that arises once the threshold is crossed. The threshold concept also allows insurance companies to develop the most efficient means of providing high limits of coverage for the exceptional, high-hazard risks while retaining their normal practices for the less hazardous risks. High-hazard risks are particularly suited to pools, but pools are not the best or most efficient means of insuring low-hazard activities.

The second feature of the atomic energy models is that, directly or indirectly, strict or absolute liability is the rule. The drafting comments on the Paris Convention emphasize that "[t]his does not, however, mean that merely to engage in a nuclear activity or to transport nuclear substances is to be considered in itself as a presumption of fault; but where an incident occurs, the liability of the operator of the nuclear installation concerned is absolute."[17]

The implication of this comment is that the drafters of the convention are not adopting a concept of enterprise liability. There must be an actual causal connection between the installation concerned and the incident giving rise to the injury. This conclusion is strengthened by the third feature. Directly or indirectly, all liability is channeled to one person—usually the operator of the nuclear installation where the nuclear incident occurs.

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[16] Pub. L. No. 85-256, 71 Stat. 576.

[17] The English text of the Paris Convention of July 29, 1960, with the drafting comments, is included in Joint Committee on Atomic Energy, Selected Materials on Atomic Energy Indemnity Legislation, 89th Cong., 1st Sess. 256-90 (June 1965). The quotation is at 274.

The conventions accomplish this by provisions that explicitly state that only the operator is liable for nuclear incidents at installations. Price-Anderson reaches the same result indirectly by requiring operators to maintain liability insurance or other equivalent financial protection covering not only the liability of the operator but also that of any other person.

The Price-Anderson approach is a practical method of channeling exclusive liability onto the operator of nuclear installations because the total number of installations is still rather small (about seventy) and is likely to remain so. Furthermore, the insurance is furnished through two pools which cross-reinsure each other. Together these pools have attracted nearly all of the free world insurance and reinsurance capacity. Possible cumulation of exposure is eliminated under liability policies issued to suppliers and transporters by standard exclusions of the nuclear hazard. However, their liability is covered under the policies issued to the nuclear installations. Strict liability is imposed for "extraordinary nuclear occurrences" (a threshold based mainly on off-site radiation levels, number of persons with immediate signs of injury, and dollar amounts of property damage). This liability does not stem from statutory changes in underlying state tort law, but from compulsory waivers of traditional common law defenses in the financial protection requirements of the Price-Anderson Act, the Nuclear Regulatory Committee's regulations, and the underlying insurance policies.

We probably could not construct today a similar, essentially nonstatutory channeling of exclusive and absolute liability for nonnuclear waste handling and disposal activities. The nuclear conventions, which require statutory implementation at a federal level, will therefore serve as more appropriate models. The Paris Convention is worth study because of its helpful drafting comments and its careful treatment of the key concept that, with rare exceptions, only one operator is liable for the injury resulting from a nuclear incident.

The nuclear fuel cycle can be divided into three segments: (1) the refining, fabrication, and production of nuclear fuel; (2) the utilization of the fuel or radioactive by-product materials; and (3) the handling, storage, and processing of waste. The transportation hazard can arise at several points in the chain, creating another distinct group of firms called transporters.

A similar cycle can be constructed for other toxic or harmful substances which result in hazardous wastes. We might classify the production and utilization facilities as generators, the waste handlers as waste disposal sites, and the remaining firms as transporters. This similarity of higher-risk sources can be kept in mind while reviewing the liability rules for nuclear activities.

Under the Paris Convention the operator of a nuclear installation is liable on proof that damage or loss was caused by a nuclear incident involving nuclear fuel, radioactive products, or waste in, or nuclear substances coming from, the installation. There are some exceptions for transportation accidents and some priorities when more than one nuclear installation is involved.

In accidents involving the transportation of nuclear substances, the direction of transportation becomes important. The operator of the nuclear installation *from which* nuclear materials are being shipped is the focal point. This operator is liable for a nuclear incident outside the installation if (but only if) the incident occurs: (1) before liability with regard to nuclear incidents involving the nuclear substances has been expressly assumed by written contract by the operator of another nuclear installation; or (2) in the absence of such an express assumption, before the operator of another nuclear facility has taken charge of the nuclear substances.

The operator of a nuclear installation *to which* nuclear substances are being transported is similarly liable for a nuclear incident outside that installation only if the incident occurs: (1) after he has expressly assumed liability by contract; or (2) in the absence of such an assumption, after he has taken charge of the nuclear substances.

The operator liable under the Paris Convention must provide the carrier with a certificate issued by or on behalf of the insurer or other financial guarantor furnishing the security required by the convention.[18]

The priorities under the Paris Convention are as follows:

(1) If the nuclear fuel or radioactive products or waste involved in a nuclear incident have been in more than one nuclear installation and are in a nuclear installation at the time damage is caused, no operator of any nuclear installation in which they have previously been shall be liable for the damage.

(2) An exception is made to rule (1) for damage caused by a nuclear incident occurring in a nuclear installation and involving only nuclear substances stored therein incidentally to their carriage. In such cases, the operator of the nuclear installation shall not be liable where another operator or person is already liable.

(3) If the nuclear fuel, radioactive products, or waste involved in a nuclear incident have been in more than one nuclear installation and are not in a nuclear installation at the time damage is caused, no operator other than the operator of the last nuclear installation in which they were before the damage was caused or an operator who has subsequently taken them in charge shall be liable for the damage.

A catchall clause provides for joint and several liability in the rare case where more than one operator becomes liable under the Paris Convention. If it is a transportation accident involving the same means of transport, or, in the case of storage incidental to transportation, in the same nuclear installation, the maximum total amount for which the operators are liable is limited to the ceiling on liability established by the convention, and no one operator is required to pay more than the operator's individual ceiling.

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[18] There are optional provisions under which the carrier, at the carrier's request, can be held liable for transportation accidents in place of an operator who would otherwise be liable.

The liability provisions of the convention expressly state that the right to compensation for damage caused by a nuclear incident may be exercised only against an operator liable for the damage in accordance with the convention or, if a direct right of action against the insurer or other financial guarantor is given by national law, against the insurer or other financial guarantor.

No other person shall be liable for damage caused by a nuclear incident, except that the convention does not affect the liability of any individual for certain damage to nuclear installations or means of transport resulting from an act or omission of that individual done with intent to cause damage.

This summary of the Paris Convention shows the care taken by the drafters to focus liability on one operator, usually the one in charge of the nuclear material by possession or the last possession.

The drafting comments explain that two factors have motivated the channeling of all liability onto the operator in derogation of the rights of an injured person to sue the person causing the damage under the law of torts. First was the desirability of avoiding difficult and lengthy questions of complicated legal cross-actions to establish in individual cases who is legally liable. Second was the need to avoid special insurance which would otherwise be necessary to cover the liability of all those who might be associated with the operation of a nuclear installation as well as the liability of the operator, which would be very expensive and might not be available.

The principles underlying the convention's approach to establishing liability rules seem far sounder from an insurance point of view than many of the superfund-like proposals. These in the main have no threshold, impose absolute liability without limit on a plethora of potential defendants, and are not exclusive of other remedies. Proposals of this nature magnify the insurance problems which are already becoming nearly unmanageable under the present expanding tort liability rules in the United States.

For the purist, the Paris Convention rules will not serve fully the traditional principles of individual corrective justice embodied in the tort law. But because they do require a clear identification of a particular installation, the imposition of strict liability is not nearly as ambiguous as it is under enterprise liability. It is also more likely that costs will be internalized.

How might the Paris Convention rules be used to construct a new tort liability-insurance system in the United States for nonnuclear hazardous substances?

VIII

The new system would require a federal statute, perhaps modeled on the Price-Anderson Act, but with additional features. The major functions of the statute would be:

(1) To identify the area of application, for example, the generators,

disposal sites, and transporters of hazardous wastes

(2) To set the threshold above which strict liability will apply
(3) To set out the rules imposing strict liability on only one firm
(4) To set out the limits and means of providing financial protection
(5) To set an overall ceiling on liability at a reasonable level
(6) To set reasonable limitations on damages recoverable
(7) To establish exclusive liability under the federal statute; that is, to preempt state tort law

It may be necessary to require a higher level of financial protection than the amount of insurance now customary. This would be the quid pro quo for the limitation on liability for all injuries which are the result of a single incident. While the ceiling should not be unreasonably high (perhaps in the range from $500 million to $1 billion), it will probably exceed the amount of private insurance available for some time. It may therefore be necessary to split the total financial protection into two segments. Primary financial protection could be set at the maximum available from private insurance companies. Secondary financial protection could be made up by a participating plan financed by equitable contributions from all generators, waste disposal sites, and, if desirable, transporters.

The primary financial protection serves two purposes. It guarantees that a fairly high level of funds will be immediately at hand to discharge the liability of the firm that is strictly and exclusively responsible. This ensures a prompt response. The finger is pointed at one firm and one insurer. It also internalizes a fairly high level of cost on the firm that is identified as being strictly liable under the statute. The liability rules based on the Paris Convention would do a good job of putting liability on the firm that has perhaps the best opportunity to avoid the accident. This brings both the liability rules and the insurance system into harmony with the more traditional tort law objectives and the newer economic efficiency arguments.

The secondary financial protection operates as a "stop loss" for the firm exclusively liable. At the secondary level the financial burden of exclusive strict liability is spread to the industry as a whole. One might look at it as a form of enterprise liability but at an excess level.

The justification for enterprise liability at an excess level is that strict liability does not require or even make an inquiry into fault. To saddle one firm with both strict and extremely high liability at the same time is unjust. Other potential or actual contributors to the loss, immune from direct liability, should shoulder some of the burden. Excess enterprise liability is a method of providing contribution or subrogation rights which avoids a series of costly cross-actions or "actions over" based on traditional tort principles.

Secondary financial protection lends itself to a pooling arrangement. It can be totally postloss funded as under Price-Anderson, or partially prefunded and partially postloss funded. The legislation should allow private insurers to participate in the secondary financial protection and to have the same tax advantages as would be given to a government-created pool or fund.

Under a statutory structure as outlined above, using the Paris Convention rules, it should be possible for a vigorous private insurance market to develop for all firms subject to the federal system. The features which would strongly encourage a private system are:

(1) For all operations of all industrial firms except those involving materials or activities that fall under the regime, normal insurance markets can be maintained. These markets can respond at lower cost and greater capacity knowing that the higher-hazard activities will not be covered by their normal policies.

(2) For the higher-hazard activities the special coverage will be channeled to a relatively few clearly identified firms. These firms will be licensed and will be required to maintain adequate financial protection.

(3) Primary financial protection will be available from single insurers, but in addition there are likely to be one or more competing pools. These sources can specialize in the insurance of the higher-hazard activities. They can marshall the underwriting, loss-control engineering, and high-capacity reinsurance facilities needed. Reinsurers particularly will be able to participate to the maximum, knowing that they will not be subject to cumulation of liability through their reinsurance of other activities.

(4) The cost of processing claims and handling litigation would be greatly reduced as multidefendant litigation would be eliminated. Today the cost of defending industrial third-party liability claims is becoming almost as significant to insurers as indemnity payments. The astronomical cost of complex litigation is operating as a brake on insurance capacity in many areas.

There are some potential drawbacks in this type of system for insurance companies. The channeling of strict liability to a relatively few target firms carries with it an obligation to provide high limits to those firms. This may require pooling, a device which a number of companies do not view with favor. However, such a statutory scheme as outlined above probably will not provide greater need for pools or more encouragement to the formation of industry captives than will the rapid development of state tort law in the direction of enterprise liability. If the risks are large and the limits of insurance required are high, the sources of insurance also have to be large. This encourages pooling with or without a federal system.

IX

I believe that the trend of state tort law towards strict enterprise liability cannot be stopped. That being the case, it seems wiser to work for a uniform strict liability system that will result in a higher percentage of premiums (or loss dollars) going to the injured persons. The great expense of operating a traditional tort law system that has largely abandoned its traditional loss-shifting premises can hardly be justified.

Strict liability systems, however, can easily run out of control. They can make it impossible for private insurers to provide high-limit liability coverage at reasonable cost. They may also impose such heavy burdens on industrial firms that they will not be able to compete in world markets.

An approach which may have a broader appeal is to adopt strict liability with certain controls and features. These should strike a balance between (a) the eligibility rules for and the levels of compensation to injured persons and (b) reasonable limitations on industrial liability. If this liability is backed by solid financial protection from private sources, including insurance, those injured through industrial activities will be assured of greater total compensation than under the present hit-or-miss tort system.

The argument is often made that any limitation on the liability of private enterprises constitutes an unjustifiable subsidy as well as an infringement of the rights of claimants. This is a simplistic argument. There are three important practical limitations on the liability of private firms—the corporate form of organization, which limits liability to the corporation's assets; bankruptcy; and competition from world markets. Imposing unlimited liability for injuries is hardly worthwhile if there is not enough money to pay for the claims or if it destroys the source of private funds and jobs.[19]

The concept of a ceiling on liability does not imply that no further compensation for injured persons is justified. The concept recognizes that a ceiling is needed in return for expanded eligibility for compensation and that, above a certain point, there is no good reason under tort law to shift the loss to private third parties. Concept B forms the rational basis of tort law. It holds that loss shifting requires justification and is the exception, not the general rule. A formal statutory ceiling on liability (or the de facto ceiling imposed by the practical limitations on liability mentioned above) still leaves room for first-party insurance and governmental assistance.

The most important benefits of such a statutory system are that it will greatly improve the ratio of dollars paid to injured persons to the dollars paid for the system's operating costs and that it will enhance the ability of private industry, including the insurance industry, to marshall the funds needed to discharge the high level of absolute liability imposed.

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[19] Shavell, *An Analysis of Causation and the Scope of Liability in the Law of Torts*, 9 J. Legal Stud. 463 (1980), states in more elegant fashion four sound reasons for limiting the extent of liability: (1) it may help spread risks; (2) it reduces the likelihood of the injurer being discouraged from engaging in a socially worthwhile activity by a crushing burden of liability; (3) if the probability of a high loss is underestimated, holding the injurer liable for the total loss does not increase the incentive to take care; (4) the potential for problems with multiple injurers makes it difficult to place liability too far out in the (unending) sequence of events flowing from the defendant's act.

Id. at 499-500.

ATTACHMENT E

H.R. ____

A BILL

To amend the Comprehensive Environmental Response, Compensation and Liability Act of 1980 to provide for apportionment of liability under the Act.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, that this Act may be cited as the "Comprehensive Environmental Response, Compensation and Liability Apportionment Act of 1982."

SEC. 2. Section 107 of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 is amended by adding the following at the end thereof:

> "(l) APPORTIONMENT - (1) The Response Trust Fund shall be a party in any action for recovery of costs or damages under this section.
>
> "(2) Upon motion and sufficient showing by any party, the court shall join to the action any person who may be liable for costs or damages under this section and any State which may have incurred costs or damages recoverable under this section or in which the costs or damages upon which liability under this section is based were incurred.

"(3) Any party found liable for any costs or damages under this section who establishes that only a portion of such costs or damages are attributable to his or her actions, shall be required to pay only for such portion.

"(4) If the trier of fact finds the evidence insufficient to establish each party's portion of costs or damages under paragraph (3), the court shall apportion such costs or damages, to the extent practicable, according to equitable principles, among liable defendants.

"(5) A defendant whose contribution to the costs or damages upon which liability under this section is based is not significant shall not be liable under this section.

"(6) The Response Trust Fund and the State or States in which the costs or damages upon which liability under this section is based were incurred shall pay any portion of the judgment in excess of the aggregate amount of costs or damages apportioned under paragraphs (3) and (4). The court shall apportion such payment in any manner these governmental entities jointly agree, or, to the extent these governmental entities do not agree, according to equitable principles.

"(m) POST-APPORTIONMENT ADJUSTMENT - After the court has apportioned costs or damages pursuant to subsection (l) of this section, each party who is to make payments pursuant to an apportionment under paragraphs (3) or (4) of subsection (l) may petition the court to reduce the judgment against him in order to alleviate or prevent serious hardship or gross economic inequity. The Response Trust Fund, any State or States in which the costs or damages upon which liability under this section is based were incurred, and the petitioner shall be the only parties to a proceeding under this subsection. The Response Trust Fund and the State or States shall pay any portion by which the court may reduce the petitioner's judgment. The court shall apportion such payment in any manner these governmental entities jointly agree, or, to the extent these governmental entities do not agree, according to equitable principles. In reaching its determinations under this subsection, the court shall consider the need to respond to other releases with Fund or State monies. Nothing in this Act shall be construed to permit a court to consider hardship, economic inequity or the need to respond to other releases with Fund or State monies in apportionment of liability under subsection (l).

SEC. 3. Section 302(d) of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 is amended by striking "Nothing" and substituting "Except as provided in section 107(l) and 107(m), nothing".

Legislative History -

Since passage of the "Superfund" Act in December 1980, there has been much confusion about apportionment of the liability established in section 107 among liable defendants. This confusion results in part from the 96th Congress' failure to explain with sufficient clarity its intent in deleting "joint and several liability" language found in earlier bills. E.g., S. 1480, 96th Cong. 2d Sess. (1980); H.R. 7020, 96th Cong. 2d Sess. (1980). Comments of Congressmen and Senators during floor debate, the only legislative history addressing the compromise bill which passed, differ. 126 Cong. Rec. S14964 (daily ed. Nov. 24, 1980) (Sen. Randolph); 126 Cong. Rec. H11,787 (daily ed. Dec. 3, 1980) (Cong. Florio); 126 Cong. Rec. S15,004 (daily ed. Nov. 24, 1980) (Sen. Helms). EPA and the Justice Department have asserted a broad application of joint and several liability notwithstanding this deletion. On the other hand, academic commentators have contended that "it is . . . clear that joint and several liability was not intended." 1A Grad, Treatise on Environmental Law § 4A.04, at 4A-114. This confusion led to stagnation and litigation in the implementation of the "Superfund" Act rather than to the rapid cleanup of hazardous waste dumps that was intended by Congress.

This amendment establishes the general principles by which courts are to apportion the liability created in section 107. It provides the necessary guidance for courts to fashion the specific federal "common law principles to determine when parties should be severally liable" under the Act. 126 Cong. Rec. S14,964 (daily ed. Nov. 24, 1982) (Sen. Randolph). It is also intended "[t]o insure the development of a uniform rule of law and to discourage business[es] dealing in hazardous substances from locating primarily in States with more lenient laws." 126 Cong. Rec. H11,787 (daily ed. Dec. 3, 1980). (Cong. Florio). The apportionment rules herein enacted are similar but not identical to those applicable under the California "Carpenter-Presley-Tanner Hazardous Substance Account Act." CAL. HEALTH & SAFETY CODE §§ 25360-25366 (1981).

Paragraph (l)(1) states that the Hazardous Response Trust Fund, the "Superfund," is a party in any litigation under section 107. While private parties and federal or state authorities may settle a case without involvement of the Fund, when a dispute reaches litigation the Fund must participate to ensure that expenditures made are "consistent with the National Contingency Plan" and otherwise consistent with the purposes of the Act and to recover costs incurred beyond those for which it is liable under paragraph (l)(6).

Paragraph (l)(2) states that the court must join to any action under this section any party who may be liable for costs or damages recoverable and any State which may have incurred costs or damages recoverable under this section or in which the costs or damages upon which liability under this section is based were incurred. This paragraph is intended to insure that parties responsible for only part of costs recoverable are not singled out for special treatment when other potentially responsible parties are also known. However, where a potential defendant's contribution has not been significant, as described in paragraph (1)(5), he need not be joined.

Paragraph (1)(3) states the equitable principle that a party is liable only for his or her "fair share" of cleanup costs or damages under this section. For example, to the extent a defendant proves that he did not cause a portion of costs or damages, he is not liable for such portion.

Paragraph (1)(4) states the apportionment rule to be followed when the trier of fact finds the evidence insufficient to establish each party's portion of costs or damages. This paragraph applies, for example, where multiple parties have jointly contributed to the creation of an indivisible harm. A court may apportion liability on the basis of the relative lengths of time during which the defendants owned or had control over a site, the relative number and seriousness of releases caused by the defendants, the relative degrees of fault in causing releases, the relative volume of wastes attributable to each defendant, a combination of these methods or other such equitable principles. The governing principle in fashioning an equitable apportionment, however, should be each defendant's relative contribution to the problem, not the relative wealth of each defendant. A party may not be held jointly and severally liable for a necessary remedial action or cleanup costs except where there is no more equitable basis for apportionment.

Paragraph (l)(5) states the de minimis rule that a defendant whose contribution is not significant is not liable. For example, in a case in which more than 250 potentially responsible parties are identified but in which 30 of those parties are responsible for at least 80% of the total waste by volume at the site, it is important that litigation be limited to the more manageable group of responsible parties. Limiting liability to significant contributors, in accordance with commonly accepted principles of joint liability, is also consistent with the efficient use of scarce prosecutorial resources.

Paragraph (l)(6) states the rule of apportionment in situations in which some portion of liability cannot be attributed to any party. In such situations, the Response Trust Fund and the State are jointly liable for the non-attributable portion. The Response Trust Fund thus may bear the cleanup costs for partially "orphan" as well as totally "orphan" sites.

Subsection (m) provides a procedure through which a defendant found liable under section 107 may seek to have the court reduce the judgment against him in order to alleviate or prevent serious hardship or gross economic inequity. Liability is apportioned under subsection (l) on the basis of equitable principles which are not related to the defendant's ability to pay. Rather, the defendant's relative contribution to the problem is the basis for apportionment among liable parties. However, ability to pay is a factor which may properly be considered in deciding whether government or the defendant should pay costs or damages. For example, government must bear the costs assessed a party without any assets or prospect for obtaining such assets. Moreover, the bankruptcy of an ongoing business concern as a result of the payment of its admittedly "fair share" of a judgment under subsection (l) may not be in the national interest. Similarly, gross inequity can exist when a concern's present day customers are different persons than the concern's customers who "benefited" from its polluting activities in the past.

This subsection permits a court to consider ability to pay and economic inequity in reaching a determination as to whether the defendant or government should pay. The amount of money available in the Fund or from the State to respond to other sites and the need for immediate action at these other sites must be balanced against the hardship and inequity which payment may impose on the defendant. The final sentence clarifies that the defendant's ability to pay and the availability of monies from the State or the Fund are not factors to be considered in the apportionment required under subsection (l). Subsection (m) is not intended to permit a State or the Fund to reduce its payments under paragraph (1)(6), but any State or governmental subdivision thereof found liable under this section and whose liability has been apportioned under paragraph (3) or (4) of subsection (1) may petition the court for an apportionment adjustment under this subsection.

Section 3 indicates that, to the extent the apportionment principles stated herein are contrary to principles in other state or federal law, including common law, this section prevails insofar as liability under section 107 is concerned. However, this amendment applies only to the liability created under section 107 and does not affect liability under other federal or state law.

Mr. LEVITAS. Thank you very much, Mr. Light. We appreciate your analysis and submission on behalf of Mr. Freeman and yourself.

Mr. Baron, we welcome you and look forward to your testimony.

Mr. BARON. Thank you.

I am Fred Baron, an attorney in Dallas, TX, with a private law firm. We have an office here in Washington, also. I am licensed to practice law in the State of Texas and I have been associated in various other jurisdictions as trial counsel.

Since 1973 when I first became involved in a group of asbestos cases in Tyler, TX, representing approximately 400 victims, my law practice has been first, partially and now exclusively limited to representing individuals who have contracted diseases because of exposure to toxic substances. Our law firm now consists of approximately 30 employees, with all types of backgrounds, who handle only cases for victims of toxic chemical exposures.

I have served as chairman of the environmental law section of the American Trial Lawyers Association, in fact I was the chairman of that group when we voted to change our name to the environmental and toxic tort law section, indicating a change of emphasis on behalf of the lawyers.

As you know I served as the member of the 301(e) group and I recently published a textbook for attorneys entitled "Handling Occupational Disease Cases."

I would like to draw on those experiences in making my comments to this committee.

It appears that obviously the inquiry here is do we need statutory remedies for these types of problems? It would seem that there are three basic inquiries. First of all are there victims out there? If so, does the legal system provide adequate relief?

Third, if it does, is causation proof so difficult that it presents an insurmountable barrier?

First I would like to address the second question, is the legal system adequate? It has not always been that way but I believe that it is now moving in that direction. Common law shifts generally glacially. The development of the common law is something that has taken hundreds of years in this country and in England. Yet when I was researching my book on occupational disease I decided to try to find all of the cases reported describing compensation for victims of hazardous chemicals.

When I got to before 1973 and eliminated workers compensation cases I essentially found two cases, one involving a polluted river and the other involving a chemical plant that was spewing out hazardous chemicals in the neighborhood. Both were property damage cases and both provided compensation for victims but after a long series of appeals.

So what we are talking about in this area of law is a brand new area, something that has never really been litigated in and something which the common law has not completely developed at this point.

Moreover, I found that prior to 1973—I use 1973 because that was the date of the decision of the famous *Borel* v. *Fibreboard* case that permitted the use of strict liability for victims of asbestos exposure—prior to that time essentially there had not been challenges to these barriers put up by the common law and that in fact no one had really questioned whether or not victims should be able to be compensated.

These barriers are now falling. The statute of limitations has developed into a discovery rule in 43 of the 50 States that have limitation periods. The common law is moving rapidly to develop a type of law or at least rules of law that will now provide compensation for victims of toxic chemicals.

The problem with it is, however, that the cost of the system and the time concerned to make this evolution are not adequate at this time. I would say that if you punch in a statute of limitations, punch in toxic chemicals, punch in discovery rule limitations questions into your West Law machine, whereas 5 years ago you would have drawn a blank screen, the machine essentially goes tilt. There are now law reporters that do nothing but report cases involving toxic chemical exposure. There are now seminars being put on for lawyers all over the country, almost ad nauseum, discussing victim compensation remedies under the common law and there are numerous law review articles being written about these particular problems.

Again the problem with the system is the cost. A victim who is seriously injured doesn't have to worry about that cost because it is probably going to be economical for him to be represented by private counsel. Yet for a victim who has relatively small injuries although substantial to him, the cost of litigating through the third party system may be prohibitive. As this committee knows, plaintiffs' lawyers work on contingency fees which has a habit of making lawyers handle only those cases which are economically feasible. The $2,000 injury case although very significant to the victim, is not economically feasible to go through the common law system. Whereas with the $75,000 case or the $100,000 case, there are several lawyers who certainly are willing to get involved in those cases.

When people have those serious injuries and have a lawyer with a stake in the case, the lawyer is willing to invest in experts to determine the cause of the problem, and to determine the way it should be solved.

That is why the 301(e) committee developed a two-tier system. It was our belief that the common law was in fact adequate or will be adequate as it develops to handle those serious injury cases. Because of the cost of the system there has to be a way for victims to receive some quick compensation albeit limited that will see them through and limit their losses while they pursue a third party remedy if it is required.

Now, the question is, are there a large number of cases? It is obvious to me that there are. Our firm is now representing the downwinders in Dallas near the lead smelter that this committee investigated earlier representing somewhere in the range of 500 potential victims and somewhere in the range of a thousand property owners who have suffered damages as a result of the emissions from the RSR and Dixie smelters.

Our firm represents hundreds of victims of radiation poisoning and asbestos poisoning, we represent large numbers of farm workers who have been exposed to hazarous pesticides. Unquestionably the problem is there and unquestionably the victims are there. It is a question of being able to quantify the numbers. No one has attempted it at this point.

Now, I have serious questions with some answers given by Mr. Roisman, particularly with respect to: Are cases too complex and too difficult and can you win them? These were primarily issues concerned with causation. I take serious issue with him that it takes hundreds of thousands of dollars to work up a causation case. I don't believe that is true. I would say to this committee that airplane crash litigation is very complex for divorce lawyers but not for specialists in aviation law.

I would point out that that there is a new group of plaintiffs lawyers emerging, a toxic tort litigation group, who understand these issues and who are able to focus on the real issues in this litigation. Because of that focus they can now handle these cases efficiently and economically. Moreover, I would point to the Dallas lead case again where EPA spent hundreds of thousands of dollars and perhaps millions to identify whether or not a problem existed over many years. Our firm hired a group of nationally known soil scientists and researchers and after 6 weeks and $12,000 completed a study of the entire neighborhood pinpointing exactly where those lead problems existed.

We believe that the evidence we have now gathered is substantial enough to present the case to a jury. Yet again we could have gone on ad infinitum doing all types of studies that would probably be duplicative and not be necessary to the litigation.

Mr. Roisman was asked how many cases he had settled or had he won. He indicated he only handled one case. Our firm has handled hundreds of these cases and we have settled the great bulk of them. Perhaps 95 percent of these cases that go through the litigation process do settle with an award to the victim.

Moreover, of the cases we have tried, a very sizable proportion of them likewise result in plaintiffs verdicts.

Essentially I believe that the victim compensation scheme as we have outlined in our study, is the necessary way to protect those victims who need immediate limited relief. The study group specifically recommended that the common law remain intact and that it be permitted to develop over a period of years to fashion further and more substantial remedies to the victims of hazardous chemicals. This is essential not only as a stimulus to compensate the victims but as a stimulus to prevent these types of practices that have caused the problem to begin with.

If an industry is faced with open-ended liability they certainly are going to correct the manner in which they operate. Thank you.

[The statement of Mr. Baron follows:]

STATEMENT OF FREDERICK M. BARON

I am an attorney and member of the law firm of Frederick M. Baron & Associates, 8333 Douglas Avenue, Suite 1050, Dallas, Texas 75255.[1] I am licensed to practice law in the State of Texas and before numerous other courts including the United States Supreme Court. Our firm is presently representing claimants, as associated counsel, in several jurisdictions throughout the United States. Since 1973, my law practice has been almost exclusively limited to representing individuals who have contracted occupational diseases and who have retained our firm to pursue claims for monetary damages sustained by themselves and their families because of their impaired physicial condition.

In the course of my practice, our firm has represented hundreds of victims of asbestos disease, radiation exposure, lead toxicity and other similar exposures to hazardous substances. Our experiences have been both in the areas of worker's compensation and third party tort liability litigation. I served as a member of the Section 301(e) study group established under the 1980 Superfund Act. I have recently published a textbook for attorneys entitled *Handling Occupational Disease Cases* and would like to draw on my experiences to make the following observations for this Committee.

Although Ramazzini in the 17th Century wrote extensively of diseases "in the shops of craftsmen," still today many diseases caused by exposures to hazardous chemicals go unrecognized and consequently uncompensated. As stated by John F. Finklea, M.D., former Director of NIOSH:

"No matter how esoteric the causative agent, the diseases usually manifest themselves in relatively conventional forms. The problem is that their origin is frequently overlooked."

Occupational Diseases—A Guide to Their Recognition, p. iii (NIOSH Pub. No. 77-181, 1977).

Similarly overlooked, from a historical point of view, has been the manner in which victims of exposures to hazardous chemicals have been legally compensated for economic losses sustained. Before the Fifth Circuit's landmark decision in *Borel* v. *Fibreboard Paper Products,* 493 F.2d 1076 (5th Cir. 1973) applying the law of strict products liability to an asbestos related disease case, hazardous chemical/disease law amounted to a scattered group of cases and articles discussing the problems of victim recovery for occupational diseases under pertinent state worker's compensation laws. The failing here was not with the "available law" itself, but with the general lack of understanding by the medical and legal professions about chemically related latent injuries.

In the ten years following *Borel,* there has literally been an "explosion" in the number of claims filed and victims compensated. Using tools previously available, a whole new generation of "occupational disease lawyers" has created precedents for compensating victims of such hazardous materials as pesticides, benzene, lead, phosgene and radiation.

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In the area of asbestos litigation alone, thousands of victims have been compensated. The claims have proven so successful that at least three major asbestos manufacturers have attempted to utilize the Bankruptcy Act (despite their admitted solvency) to press for Congressional relief from the payment of these claims.

The national attention that has been garnered by these alleged bankruptcies will have a deterrent effect on some of the questionable practices of other manufacturers of hazardous chemicals and should likewise create an even greater incentive for research on and the testing of the safety of new and old products. The incentive for manufacturers to "clean up their act" in response to this litigation has been a greater stimulus than the various environmental and safety regulations supposedly enforced by EPA and OSHA. The old articles of faith describing the pitfalls of hazardous chemical litigation are, by and large, no longer applicable in common law litigation.

This is not to say, however, that certain problems do not exist. The most glaring deficiency in our tort compensation scheme is in the handling of the case of the victim who has suffered a relatively minor loss. Although the common law damage suit may provide a substantial recovery for a seriously injured victim, it is often costly and time consuming. The tier one scheme suggested by the Section 301(e) study group established by the 1980 Superfund Act provides an excellent solution to this problem.

Under this proposed Federal administrative remedy, claimants could file for limited benefits for economic losses suffered as a result of exposure to hazardous wastes, as defined in the Act. The claim would be handled much like a state worker's compensation claim but the critical difference would be in the utilization of a series of rebuttable presumptions established by the Federal agency. Rather than requiring the injured individual to present expert testimony concerning the etiology of his condition, a rebuttable presumption (if applicable) would be created to establish a prima facia showing that the exposure caused the victim's disease. In the "routine" case, this would eliminate many of the costly procedures otherwise required to establish facts which really should be accepted as true. Because liability or fault is not an issue in these claims, they should be adjudicated in a reasonably expeditious manner.

Perhaps the greatest conflict among the members of the group was on whether or not to make this tier one remedy a truly exclusive compensation remedy for victims in exchange for eliminating the right to pursue common law remedies. Fortunately, the majority of our group recognized the inherent value in protecting the rights of injured claimants to receive substantial recoveries. Because of the deterrent effect of maintaining the unlimited liability of negligent producers and users of hazardous chemicals, the majority of the group voted not to eliminate common law rights.

The trade-off, if it can be called that, was an "exit toll" which essentially acts to penalize the over-litigious claimant and thus deter specious claims from clogging the court system. Although I would have preferred to eliminate this device entirely in favor of free access to the system, I am gratified that no serious consideration was given to eliminating the right of victims to receive common law compensation.

The common law remedy, utilized through our civil jury trial system, guarantees victims of hazardous chemicals their right to seek full and complete compensation for their injuries. Future abuses by those who economically benefit from the manufacture and use of hazardous chemicals will only be deterred when the generators of these materials, and not the public at large, are forced to bear the costs of injury. The asbestos experience has proved that the common law tort system, if allowed to function freely, will bring the injured victim to the doorsteps of guilty parties.

Mr. ROWLAND [presiding]. Thank you very much. I have two or three questions I would like to ask

Do you think the contingency fee system will contribute to the cost over all with reference to poison from the toxic chemicals?

Mr. BARON. Contribute to the cost of what?

Mr. ROWLAND. Over all?

Mr. BARON. I believe the contingency fee system probably reduces the cost of the entire process because I believe the contingency fee system weeds out those cases that are not practical to be brought and permits victims who have serious injury to go ahead and pursue those cases economically.

Mr. Rowland. Do you think it is better and less expensive to have that than it would be for an attorney to take a case and just be given a certain fee for handling this case?

Mr. Baron. I think the discussion we had earlier about the hired-gun theory, that some lawyers, when they are presented with an hourly rate, have a habit of pursuing cases that probably should not be pursued, and they will argue points that probably should not be argued.

The contingency fee system weeds out those types of cases from the system that is already clogged.

Mr. Rowland. I believe you stated that as a toxic tort litigation group, the attorneys are just now beginning to learn about the possibility of compensation for victims outside the workmen's compensation area for injury, and I believe you mentioned that lead poisoning was an example of that, and I believe you said it took many years for that to develop; that lead was indeed a toxic chemical.

Mr. Baron. Not so much that it took many years to develop the knowledge; the medical knowledge has always been there. You can go back in any medical textbook in the 1930's and read all you want to know about lead toxicity. The knowledge of how to develop these cases through the legal system is just now developing. That is where the problem is.

Our profession, the law profession, has been way behind the medical profession in identifying these problems and in dealing with them in terms of victim compensation.

The medicine has always been there.

Mr. Rowland. You don't think it took many years for the fact that lead is a toxic substance, to come about, although you mentioned that we did know this a good many years ago.

Mr. Baron. Mr. Rowland, in reviewing our lead smelter cases, we did a Medline and Toxline, computer-assisted research on the hazards of lead toxicity, and I think that by 1940 we had developed something like 85 articles that were in-depth studies of lead toxicity on children and adults and particularly downwinders from lead smelters.

There is a lot of information available prior to 1940.

Mr. Rowland. You don't think we need to have this evidence now for other chemicals?

Mr. Baron. I think we have to continue to develop the evidence; however, it has been my experience, unfortunately, in reviewing these cases, that the one type of evidence I don't really have a whole lot of difficulty in finding is knowledge of the hazard. That evidence always seems to be available in the literature. That is not the difficulty in the cases.

Mr. Rowland. Mr. Molinari?

Mr. Molinari. Thank you, Mr. Chairman.

I wonder if I could relieve you of a copy of that study report, Mr. Light? I have been trying to get my hands on that.

Could I ask one of the staff members to pick that up? Say yes, please.

Mr. Light. I can't refuse a Congressman.

Actually, Mr. Freeman's secretary, Jane Ketron, had me commit myself to bring this back, but since you have asked me in open hearing, I will release it and make her a copy of my own personal one.

Mr. MOLINARI. Thank you very much. I appreciate that. I look forward to some very intensive reading.

Let me start off by saying that the testimony we have heard from this panel is astounding. It is totally different from what I would have expected, not having read the report of the study group. It is somewhat contrary to that which we have heard over the past few weeks, and perhaps encouraging at the same time.

Mr. Baron, I want to just review with you, if I may, to make sure I understand in my own mind, your firm is a firm that has roughly 30 lawyers?

Mr. BARON. Not 30 lawyers; 30 professionals. We employ researchers and people of that nature. We now have eight lawyers. We have various staff members, too, that work with us.

Mr. MOLINARI. Your specialty is in this particular field?

Mr. BARON. That is all we do. We do handle toxic chemical exposures and disease caused therefrom. I think we are one of two law firms in the United States that do that.

Mr. MOLINARI. That was my next question.

If this is such a lucrative field, I wonder how many firms there are throughout the country that would be similarly situated, of the same size, and so on.

Mr. BARON. First of all, I would say that handling airplane crashes and scaffolding collapses, which is what I used to do years ago, is far more lucrative. We feel this work is far more important. Unfortunately, there are only a couple firms that do it on a full-time basis. There are now several lawyers who spend a great deal of their time if not the majority of their time, working on these types of cases.

Mr. MOLINARI. Couldn't we assume a certain relationship between the paucity of lawyers that are specializing in this field and the ability to recover under existing law?

Mr. BARON. I think that that probably is true. There are many of us who got into this field because of that reason, because we felt there had to be development in this area of law as it developed. As other plaintiffs' lawyers are now aware that recoveries can be made, the shift has been into this area by lawyers who have never dealt with it before. This is a good shift because these people are starting to understand the nature of these problems and, rather than litigate all around the problem, they are learning to focus directly on the issues involved and to fashion recoveries for their clients.

Mr. MOLINARI. Prior to 1973, research indicates there were only two matters that were litigated in this area, and both were property damage cases?

Mr. BARON. Cases where we could find appellate decisions, yes, other than workers' compensation cases.

Mr. MOLINARI. Yes, I understand.

Mr. BARON. I would point out that if you punch into Westlaw, the computerized legal research system, the issues we are talking about, the machine will go tilt with new cases that have been coming out. These issues are now seriously being litigated in appellate courts.

Mr. **Molinari**. Here is the problem that I have. I flew in this morning, and last night I had a town hearing on a local matter that is rather notorious in our area in New York city. It involves a municipal landfill. We had the Regional Administrator of EPA there, we had the Commissioner of Environmental Protection for the State of New York—both very knowledgeable people—and we had a hundred-some people from the area, all of whom were expressing concern for their families. They presented us with information that members of the family were suffering from certain problems, high incidence of asthma, rashes, loss of hair, bladder infections in the case of females, nosebleeds—the symptoms we have heard time and time again.

We are not even thinking about the liability factor. What we are concerned about is trying to determine whether there is a serious problem there, how serious it is, and what remedial steps can be taken.

This has been going on now for 19 months, and there have been two criminal trials in connection with toxic waste dumping at the landfill where there were convictions, one in a Federal court, and one in the State Supreme Court.

Despite the body of evidence indicating substantial amount of dumping, and the identification of about 10 chemicals and chemical compounds, all the medical experts have not been able to tell us how serious a problem we have.

If we look at the medical background here we find that the United States, EPA, the State, and New York City agencies all heavily involved, with New York State generally regarded as one of the forerunners when it comes to health matters. Now I hear that for attorneys with an expertise in the field, those who have particularly specialized, like your firm, it is not all that difficult to establish a legal case.

I have difficulty equating these two sets of facts in my mind.

Let me give you a hypothetical. Suppose we brought you to that area, and said, Mr. Baron, here is the problem. Is there a condition here?

Sure.

Can we provide you with the evidence that, in fact, large amounts of toxic chemical wastes were deposited there?

Sure.

Have there been people injured?

Well, you get an argument there, depending on whom you talk to. But if you talk to the people, as I have up and down the streets, there is no question about injury in my mind.

How do we take that next step? How do we prove that, in fact, the problems are there, health symptoms are there, and they were, in fact, caused by what is in that dump?

Mr. **Baron**. That situation is not analagous to the Dallas lead smelter case that I will have to use as my example. As you know, in 1972, the State and city determined that lead smelters had emitted too much lead, filed suit, and received an injunction. At the beginning of that point, EPA and the Federal agencies and the city and State studied the lead smelter and had not reached a conclusion as to whether or not there was a problem there.

We were contacted by the neighborhood residents, decided to go outside the governmental agencies, where politics were not involved at all, found people that we felt were qualified professionals that did not have an axe to grind one way or the other, and had them inspect the premise and inspect and examine the people.

We were able to establish a case very rapidly through these experts. That is not to say that EPA never does the job properly. Unfortunately, though, we found that in too many occasions, because of political pressures being placed on them at one point or another, their data cannot always be confirmed.

I believe that the outside expert, or someone who does not have any pressure placed on him when he does his investigation, might be the real source of proof for the lawsuit, if there is to be a lawsuit.

Mr. **Molinari.** Let's talk about that for a moment, if we may; the question of medical experts. We have heard repeatedly that it is extremely difficult, if not impossible, to be able to hire medical experts in this area; that there are few of them in the country; and we have heard figures of $350 an hour as being the cost for these high-priced doctors.

Mr. **Rowland.** I am doing the wrong thing.

Mr. **Levitas.** I think I am doing the wrong thing, too.

Mr. **Molinari.** Is there that serious a problem out there in terms of getting medical experts, not just for the lead situation, but in other areas as well?

Mr. **Baron.** It is something of the difference between Dr. Karrh and Dr. Carnow—you want to know who it is you are using—and, yes, if you take the number of physicians practicing in the United States and take the number of doctors qualified to work in this area, it is an infinitesimally small percentage. But there are health professionals out there who are available to do these types of studies and who are capable of rendering a decent, honest, sympathetic opinion.

Now, the issue then becomes how you find them, and that gets to the issue of experience of the attorney and knowing the sources of these individuals.

There are groups—in fact, Dr. Carnow has a large group in Chicago of professionals, health professionals—who are available for consultation on these types of issues. It is just a question of being able to identify them.

I might add, over the last few years their ranks have increased substantially. Again, 10 years ago, it was difficult to find them. Many young physicians are getting into this area, and many older physicians who are interested are now doing it on a more full-time basis.

It is just purely a question of knowing where to look.

Mr. **Molinari.** I think you did say that you disagreed very much with the figures given by Mr. Roisman for the cost of the medical and scientific workup.

Mr. **Baron.** I do, indeed.

Mr. **Molinari.** Let's leave the Dallas lead case aside; more typically we are dealing with somebody who lives near a dumpsite that is contaminated, who is experiencing symptoms such as I described before. What do you think it would cost for the necessary medical workup?

Mr. **Baron**. Again, the cost goes down per person if you have a large number of people. What you can do is bring in a team of health professionals and station them in a local physician's office and start seeing large numbers of them over a period of a week or two at a time.

If you have one person, an individual, I believe a complete medical workup could be completed for less than $1,000.

Mr. **Molinari**. How much?

Mr. **Baron**. Less than $1,000 for a complete medical workup of one individual. If you are talking about 250 people, you can reduce that substantially by the efficiencies of putting everybody in one place.

Mr. **Molinari**. What would that medical workup consist of?

Mr. **Baron**. It would consist of all types of—first of all, a thorough history to rule out any other possible causes for the problem; identification of what problems really are occurring; a routine physical examination, including perhaps x rays, if there is some nature of a suspect tumor or lung disease; blood tests; perhaps pulmonary-function studies; and whatever was necessary to describe the type of physical complaint that the patient was making.

Again, it depends on the type of problem. In the lead case you might want to do one thing, whereas in a trichloroethylene case, you may want to do a neurological exam.

Mr. **Molinari**. I am talking about specifically where you don't know the chemical.

Mr. **Baron**. Well, I think you have to identify generally what types of chemicals are there, and if you know where the source is, or if you have a reasonable belief as to the source, I think the first step would be to hire a qualified scientist to be able to quantify what the exposure essentially is.

Mr. **Molinari**. What would that cost in the case of one individual, roughly?

Mr. **Baron**. I would think roughly less than a couple thousand dollars to do a chemical analysis of the source. Probably substantially less than that, depending on the type of testing you wanted to do.

Mr. **Molinari**. Thank you, Mr. Chairman.

Mr. **Levitas**. Let me say this: We have a series of votes which have just begun on the floor, and it is going to keep us away for a period of time. I thought at the end of the second bells we would leave, and because we are going to be gone for a period of time, that we should at that point break for lunch for about an hour, and let you gentlemen recover your sustenance. I would then like to reconvene, because there are a number of questions I would like to ask all of you, and I can assure you your testimony in this hearing may very well shape the outcome of this legislation, and it is quite important.

So, if you—I notice, Mr. Lundquist, you are looking at your watch. Do you have a plane to catch?

Mr. **Lundquist**. I am not so worried about the plane; and with the hour break that will work out fine.

I have a plane this evening. I have a commitment at 1:30; perhaps I can handle that, or let the person know.

Mr. LEVITAS. If you could, because your testimony concerns the involvement we will have in writing whatever legislation is written, and I think it is very important in order that a proper record be made.

So, with that, why don't we recess.

Mr. MOLINARI. Yes, I think we should break now, Mr. Chairman.

Mr. LEVITAS. Those are the second bells that rang. Let's recess until approximately 2 o'clock.

The subcommittee will stand in recess until 2 o'clock.,

[Whereupon, at 1 p.m., the subcommittee recessed, to reconvene at 2 p.m. this same day.]

AFTERNOON SESSION

Mr. LEVITAS. The subcommittee will be in order.

The gentleman from New York.

Mr. MOLINARI. We discussed briefly this morning the question of the compensation type system administered on the State level—the tier 1 concept. The question I have is, where we have a State administering a program and the dollars that would be awarded would be Federal dollars, would we not run into the problem that since the State is not spending its own dollars, but that of a Federal, such and that there is little in the way of accountability factor the State would be more apt to be overly generous with awards?

Mr. BARON. One thing, they may be mischaracterized as Federal funds, because, as I understand the report we drafted, we would suggest that the funds come from some nature of a tax, very much like the Superfund money and it wouldn't be truly Federal funds.

Mr. MOLINARI. I understand that. I used the term "Federal", to distinguish that awards would be coming from other than a State-supported fund.

Mr. LUNDQUIST. One of the developments which one would hope for is that in dealing with an administrative system, you get a precedential value, so cases wouldn't go askew, so that in one jurisdiction you get $100,000 for one type of award and in another jurisdiction you get 25 percent of that. That is part of it.

Also, the damages that you get are pretty directly linked to special compensation needs. You are not getting pain and suffering damages—that sort of thing. You are really talking about strictly medical costs and a part of your loss of income.

I think that that is not as big a problem as it would be if you were making major awards for the general damage claim you usually get in a personal injury verdict.

Mr. MOLINARI. Is the recommendation limited to out-of-pocket expenses?

Mr. BARON. Yes.

Mr. LUNDQUIST. Yes, it is.

Mr. MOLINARI. No compensation for loss of salary?

Mr. LUNDQUIST. You get loss of salary, a partial compensation for loss of income, but again, that is a fixed amount.

Mr. MOLINARI. So you would have an open area with respect to the period of disability?

Mr. LUNDQUIST. You would have. There is a further limitation, that sees people making more than $36,000 a year wouldn't get full measure of loss. It is rather to give people basic levels. You get the percentage of your loss up to 36,000.

The disability period would be relatively openended.

I think the reason that that won't be a problem is that the case which has a lot of potential, which might get a large damage award, is going to be pursued by the plaintiffs. They will not stay in the tier 1 system for that.

Mr. MOLINARI. Has anyone in this group—Mr. Light?

Mr. LIGHT. Mr. Freeman, in his separate statement on Superfund report, refers to the problem you are talking about, sort of a two-part problem actually, a problem of figuring out what the States will need from year-to-year or from whatever period to whatever period, and that goes to the scope and dimensions of the problem, which you all have referred to earlier, and you really need to talk to actuarial types, insurance company people to get a feel for how that will work. And, second a claims management problem.

But I agree that as the system gears up and you have some experience on how it is operating, you may very well be able to do that more efficiently as time goes on.

Mr. Freeman personally felt that this could be a severe problem, based on the experience Congress has had with other types of administrative special funds, such as the Black Lung Fund.

The Superfund study group had a couple of ways that they tried to address this question, although I think Mr. Freeman would say they didn't address it sufficiently and Congress might want to look further.

One is that there would be a Federal agency which would prepare guidelines for the program. Although the States administer the program it would be pursuant to these Federal guidelines. There would also be a way to review the compensation award and that would generally be through the State court system, but there would be an appeal from the State court system to the Federal agency in charge of the guidelines when there was a difference of opinion between the claimant, say, and the State about the guideline. If there was an award that was not going to be made, you might appeal to the Federal agency.

Also, there would be a Federal judicial review when there was a problem of implementation of the guidelines. The Federal agency also could bring an action itself if it became aware of a problem in the State administration of the program.

The other control they built into the system was similar to what is in Superfund. The State would fund part of the administrative cost of the program. I believe they said it was 20 percent.

Mr. BARON. Ten or twenty, yes.

Mr. LIGHT. So there would be some State responsibility, built-in incentives that way.

Mr. BARON. One of our primary concerns and an area we discussed a great deal, was the conceptual and practical differences. Conceptually, you would like one agency to be able to do this. As a practical matter, looking at the black lung administration and at the Federal Employees Compensation Act administration, and in most Federal systems for giving out compensation benefits, we felt the State workers' compensation system was far preferable as a practical matter, in terms of efficiently doing the job.

Mr. MOLINARI. Dr. Rowland and I had an interesting little chat on the way to the vote. We had the typical conversation that might be held between a doctor and a lawyer.

Mr. ROWLAND. Very typical, yes.

Mr. MOLINARI. Sometimes in discussing whether the law should be enlarged to provide greater compensation, I have tried to, if possible, divorce my own ideas, which may be prejudiced by virtue of the fact that I am an attorney. Perhaps there is a greater sensitivity on the part of an attorney because he has been faced many times with the problem which sometimes has been described as insurmountable at these hearings. I would hope, certainly in my own case, that I am not looking to come up with a solution that would get lawyers fat.

By the same token, I think we all, doctors, lawyers, members of the panel, members of the subcommittee, would like to see some improvement on this system, so that those out there who have been harmed by exposure to toxic waste can seek some type of compensation without being put to the test which, until today, I had reason to believe to be an impossible one.

I just have one other line of questioning.

I find your testimony, Mr. Baron, to be very, very provocative from the standpoint of this Member of Congress. I have until today, regarded the problem of proof as one that is extremely difficult. In fact, referring to our earlier conversation today, we have about 700 lawyers on Staten Island, an area with some very serious environmental problems, but I doubt very much whether there would be a single lawyer out of those 700 who would take any of the cases. Any of the people of Staten Island who have had serious difficulties would have a problem getting a lawyer.

It is that type of case that can make a lawyer go broke. As you know, lawyers have that difficulty of starting out to evaluate a case. Is it going to pay for the time? Are we going to make up any office expenses on this?

I know there are many, many environmental lawyers out there. Yes, in my own area, there are any number in New York City that are highly regarded and well-trained in that field who would handle environmental causes.

But I don't find very many of them gravitating toward the field where they would be handling plaintiffs' causes of action such as your firm.

I wonder why? It would seem to be a natural.

Mr. BARON. That is a function of time. This is truly a new area of law and I emphasize that prior to 1973, really, prior to 1978, there were very few guidelines for lawyers who practice in this area, very few legal decisions that provided precedent.

That is changing now. We don't change that overnight. We don't see the system shift in 4 or 5 years to the extent of 180 degrees. But over that period of time, the natural changes in the law will identify lawyers who are more interested in getting involved in these types of cases.

I do know several lawyers in Manhattan who I believe would be very anxious to get involved in the type of case you have described.

It is just a question of being able to know who it is.

Mr. **Molinari.** Mr. Lundquist?

Mr. **Lundquist.** You have in your area some of the leaders, Mr. Paul Reingold and Norm Landau, I suggest as lawyers, however, there is another thing that Dr. Rowland might take comfort with; as lawyers we know of the phenomenon, when a bus gets in the accident, people don't rush off the bus, people rush on the bus.

Indeed, I have heard in connection with the MGM fire in Las Vegas where the expected normal count of 200 people at that time of day in the Casino came to be 14,000 when claims for smoke injury were received.

What this suggests to me is that when people read about something and get psychologically concerned and worry about it, it is because they also see the opportunity to get some compensation for pain and suffering.

Discerning lawyers for individuals weed out the cases. I tell the client you don't have a strong or just case. Indeed, we tried in this proposal to weed out cases so that, at the basic level, you can't get pain and suffering for the sort of things we see in court today where it is not uncommon in a toxic tort case to have somebody who has been exposed, or has an increased risk of injury, but no provable disability or disease, to put a psychiatrist on the case to testify that the fear of death, is worth compensation and indeed, in California, it is.

But this goes back to the point about the people who you meet and talk to who have asthma and other diseases, which may or may not be disabling. What we need to do is get those who are harmed compensated, and not face a lot of claims for those who don't.

It was an endeavor on our part with the two-tier structure to accomplish that.

Mr. **Molinari.** I think you touched on an area that is worthy of further exploration. That is, in many of these impacted areas, if that is the right term, another subject we were talking about today—the trauma effect—is a factor. How many or how much of the claimed injuries are psychologically induced?

I am one who always believed that the term "nerves" or "nervous condition" is one that is overworked. We have heard, interestingly enough, that one of the symptoms of toxic exposure is nerves.

Doctors have diagnosed health condition as nervousness or nerves. I wonder whether that condition is something that is triggered by exposure to these chemical compounds, or whether it is caused by the fear of the unknown or the fear of leukemia, for example, that might in victims' minds be associated with that exposure?

Is there any way of evaluating that? Do you have any experience with that?

Mr. **Baron.** We have. The only way you can do it is on a one-by-one, case-by-case basis. The real issue for this committee is whether the law should deal with that, and whether the law should provide a remedy for somebody who is psychologically affected.

I question whether that is something we should be attempting to do by passage of any law. Quite honestly, you are going to have to present proof on the issue of some nature which gets you back to the imperfect injury system where maybe the jury one day will believe the doctor who says that this person has a severe psychological problem caused by the chemicals; and maybe the next day, they won't believe him.

That is the imperfection of the system.

Mr. **MOLINARI**. I was somewhat referring not to those who truly have a nervous condition——

Mr. **BARON**. You are talking about psychological only?

Mr. **MOLINARI**. No, not psychologically induced but those whose physical problems manifest themselves in a condition that is diagnosed as nerves.

Mr. **LUNDQUIST**. All the time.

I have been in a courtroom and dealt with situations as follows: One, where someone has an increased risk of mortality, they put on experts to say how that affects his entire life, makes him want to commit suicide and as a result he should be compensated for the psychological injury.

I have also been in a situation where injuries were so severe as to be physically disabling. We have dealt with those situations in the court. To be cynical about it as a lawyer, there is also a respectable body of medical literature which establishes that a lot of this type of injury disappears after the cases are disposed by compensation or if they are lost.

You know, when you don't have that gnawing problem removed, it somehow goes away.

That is beyond my range of—notion of how——

Mr. **LEVITAS**. Will the gentleman yield?

Mr. **MOLINARI**. Sure.

Mr. **LEVITAS**. That is not an unusual medical-legal phenomenon.

Mr. **LUNDQUIST**. It is common, right.

Mr. **LEVITAS**. That is the old automobile whiplash case.

Mr. **ROWLAND**. The name given to that is greenback bogus.

Mr. **LUNDQUIST**. That happens when you have people reading a lot about them and the possibility of making a claim.

Mr. **BARON**. That gets to the di minimus problem, where if you have someone, we have represented clients with severe problems from trichloroethylene and other exposures and in those cases the disability was so severe that we have had toxicologists testify there is a cause and effect relationship and we have had large jury verdicts in them.

In other cases you have people able to function completely but it is just a concern and maybe is more nervous about it. Are we going to set up a system that costs $10,000 or $15,000 to deliver him a couple thousand dollars worth of benefits for that? That is the dilemma. That is the problem.

Mr. **MOLINARI**. It would seem to me that in adopting this recommendation, there would be some type of compensation schedule set up so that you would have to have some idea, beforehand, as to the costs that would be incurred.

I think Mr. Light touched on this before, and the question is are there actuarial studies? Is there any range of anticipated payouts that would occur?

I know it is difficult to estimate but before we could move in that direction I think we would have to have a handle on the projected costs.

Mr. LUNDQUIST. Minnesota has passed a comparable law, as I understand it, Minnesota didn't have a lot of actuarial basis for it. They started with a $5 million general tax input into the fund and a special tax on generators and those involved which will yield about $900,000 dollars a year. That will give us some empirical experience.

But in talking with the Minnesota attorney general's staff and the Attorney General when he was on this committee, they didn't really have that kind of information to do it.

Mr. MOLINARI. They protected themselves by putting a cap on what could be spent while they looked at the experience factor.

Mr. LUNDQUIST. That is right. I think one thing that is essential in this, although our committee didn't directly propose it, is to put some sort of sunset provision in, that after 5 years you look and evaluate the nature of it.

You don't want to build an institution such as Black Lung that has to be cut down to size in a decade or two.

Mr. MOLINARI. Thank you. I have no further questions.

Mr. LEVITAS. Mr. Light wanted to respond.

Mr. LIGHT. Another possible source of experience in this area would be the California experience.

They have had a compensation fund in connection with their State Superfund law for about I guess 2 years now and I have heard varying reports on their experience but I think that that would be worthwhile to find out what their claims experience is under that law.

The other thing is I have talked to a staff person in the Environmental Law Institute and I understand that they recently contracted with EPA to do a study of what happens to toxic tort claims in a couple of geographic areas. That is a study that just was undertaken by ELI for the EPA and that may be of interest to you somewhere down the road.

Third, there is also this study that you have—I would just remind you that there is the study that the Chemical Manufacturers Association is partially funding, the UAREP study, that may offer hints at the scope and dimensions of the problem.

The 301(e) group study, early in its deliberations asked EPA and the Justice Department to compile some information with respect to this aspect of the study and it was about the same time that this committee was undertaking a similar endeavor.

Mr. MOLINARI. I was going to guess that.

Mr. LIGHT. So we didn't get a lot. You will find appendices to the report showing what we were able to come up with. Professor Grad summarizes that in part 2 of the report.

Mr. MOLINARI. Thank you, Mr. Chairman.

Mr. LEVITAS. I have a number of questions, some are more specific, some which are more general but let me start with the specific ones.

Mr. Light, you indicated that Mr. Freeman had objections to the concept of punitive damages and you quoted his language about vigilantes—the sheriff takes over and the vigilantes stop.

But it would seem to me that the role of punitive or punitive-type damages is pretty well established in this area even where the sheriff moves in.

Let me give two that come to mind. In the antitrust field there is the possibility of criminal prosecution for violation of the antitrust laws; there is a provision for compensation, compensatory damages resulting from violating the antitrust laws; and there is in some instances a provision for treble damages which is a statutory punitive-type damage situation.

It is my understanding that the reason is that the standard for proof in a criminal case is not the same as it would be in the civil action; and that in order to provide effective deterrence for certain activities which violate the law the punitive damage or treble damage aspect is written into the law or developed in the common law.

Let me give you another example, a more classic example—fraud. In fraud you can be prosecuted criminally for certain types of fraud, fraudulent activity; obviously you can sue for compensation but you certainly can get punitive damages.

Why would it be inappropriate—why is it inappropriate in the appropriate factual situation in the case of toxic torts to provide a punitive damage or punitive damage-type remedy in addition to the other mechanisms?

Mr. LIGHT. First of all, it may or may not be the case that you would be able to seek punitive damages in the toxic tort case. It depends on what State you are in. Some States provide for punitive damages; some don't.

I think Mr. Freeman's concern in this connection is set forth in brief form in his separate statement in the Superfund report at pages 322 and 323 of the document that I gave Mr. Molinari.

He has several concerns in this area. One is that there is a strict liability doctrine, the doctrine for compensating injuries in the kind of situation where there may be abnormally dangerous activities or an ultra-hazardous substance. There seems to be a move, at least according to Professor Grad and the majority in the Superfund report, to a theory of strict liability in which the standard for awarding compensation is different than the standard you would apply to award punitive damages where you have outrageous conduct and so forth.

When both of those issues, the strict liability and punitive damage issues, are tried in the same forum there may be confusion as to which standards apply. That is one issue.

A second issue that he was concerned about is a situation where—it has occurred in the past in the product liability area—a situation where you have a lot of cases involving the same basic fact situation from the point of view of the defendant. The defendant has manufactured a product; the plaintiff alleges that it is defective in each of the cases and they seek punitive damages.

It is what I guess Jeff O'Connell, who was a member of the commission, would call a lawsuit lottery, everybody is in it for—every plaintiff's attorney is in it for—the possibility of the big payoff. There is also the possibility that in some situations you may have a big payoff for the first few plaintiffs that get to trial in the States that are speedier, and somewhere down the road you may find that there are limits to the amounts of insurance and assets of the corporation that you are suing. Later claimants thus are not compensated.

So there is a possible equity concern in connection with some of the mass tort cases.

The third strand in Mr. Freeman's thought is basically the one I summarized with that phrase "vigilantes have no place where the sheriff takes over" and that is the notion that in connection with some of these environmental statutes such as the Clean Water Act, or the Resource Conservation and Recovery Act, where comprehensive sanctions have already been provided—in fact, if the bill that is coming up for a vote soon, with respect to RCRA passes—there will be enhanced criminal and civil penalties. Under the proposed legislation a citizen would be able to go into court and seek civil penalties under RCRA just as he can now under the Water Act.

This is a law enforcement type function, a private attorney general type function in both cases with one exception, that is where does the award go? In one case, the citizen suit case, to the Government, in the other case the punitive damage case, it goes to whoever has brought the suit.

In the RCRA context you have a provision for awarding attorneys' fees to the prevailing party, so there is no real justification for the contingency fee system in that kind of operation.

So Freeman would put forth the position if he were here that there ought to be a separation of the law enforcement and punitive-type function from the compensation functions, first because of the different standards which are appropriate to awarding compensation under theories of strict liability, and theories for award of punitive damages; second, because of this possibility in the mass tort situation of a limited fund—which can be handled sometimes in a class action suit although our 301(e) group did not recommend class actions for personal injury suits—and the third reason being this notion that law enforcement should be handled by law enforcement personnel rather than private individuals who are in it for their own benefit.

Mr. LEVITAS. I am not sure you have answered my question because I was asking to contrast this to the other examples. Maybe Mr. Lundquist and Mr. Baron would respond.

Mr. LUNDQUIST. There is one very distinct aspect to punitive damages—I should say I am for punitive damages. I don't think that issue is resolved in the courts, particularly in the States. We have statutory ones that you referred to. But I think there is one significant difference in the area of toxic torts punitive damages and that is the latency period. You really are talking about punishing people 20 or 30 years later. In one case I know, the corporation involved, had in its largest shareholder in the State of California the California Teachers Retirement System. The corporation was being asked to be punished for conduct that occurred 20 or 30 years before. I find that illogical myself and it seems to be shifting things to where they don't really belong.

I would prefer to see a civil liability statute make corporate officials liable 25 years later personally. Then you would see the kind of conduct you want, I think.

So there is that problem. There is also the problem of the mass type of tort when the plaintiff with a relatively weak disability claim starts to hold up the system with a $3,000, $4,000, or $5,000 claim with the prospects of hundreds or millions in punitives. This creates an incentive that doesn't make sense and fouls up the system.

Third, as Mr. Light pointed out and the courts haven't gone along with this but one individual may ring the bell and collect everything. In the *Dalcron Shield* cases where there are thousands of people affected, Judge Spencer Williams created a class and said everyone is going to partake of any punitive damages. The ninth circuit, in logic that is not appealing to me, said no, you still have to come along and, under this analysis, the first ones will be lucky and I don't know what will happen to the rest of them.

The other point is that it does sometimes create problems of insurance coverage which again when you are trying to impose liability 20, 30, 40, or 50 years later, it creates a special problem.

Mr. Levitas. Mr. Baron.

Mr. Baron. I would disagree a little bit with Mr. Lundquist here. Punitive damages are absolutely essential particularly in this area. What we are trying to do is compensate victims, but also to determine misconduct. The bad effect of a big payout to one claimant is totally outweighed by the probability of deterring misconduct on the part of other actors. If the law was enforced I might feel differently about that. Unfortunately, vigilantes provide impetus to enforce the law to begin with.

I think those people should be permitted to have this tool to really affect social change on these companies.

As far as the latency period argument, that the companies are being punished for old acts, they committed those acts years ago. The corporation is an entity. The shareholders who now own the stock of that corporation have profited because of those bad acts. Why shouldn't they be punished for it? As long as we continue to provide for punitive damages we make a corporation's liability unpredictable which is exactly where we want it.

If they know what they have to pay they can institutionalize it and pass it on to the consumer. If they don't know what they have to pay, they are going to be much more careful about the way they conduct their business.

Mr. Lundquist. I have yet to see the corporate executive who says if I make a decision today which somebody is going to have to pick up responsibility for in 20 or 25 years, I get a short-term profit against an unknown liability 20 or 25 years down the road who doesn't seek the short-term profit. I just don't see—I wish I did—but I don't see a business responsive to that particular stick and carrot.

Mr. Baron. I would note for the record without wanting to say anything in detriment but Mr. Lundquist is counsel for Johns Manville Corp., which I think is the classic example of where conscious decisions were made by the board of directors in writing 20, 30, 40 years ago to do things that really should not have been done.

Yes, these corporations should be required to pay for them when they manifest themselves.

Mr. Lundquist. I have also handled plenty of punitive damage cases on the other side of that, and just to get in a jab at my good friend, Mr. Baron, essentially, my law firm operates under the auspices of his direction, we are retained in effect by the bankruptcy estate to sue the insurance companies, he is one of the people who is responsible and I trust approves of what we are doing on behalf of the many people affected.

Mr. Levitas. Let me——

Mr. Molinari. This is getting interesting.

Mr. Rowland. You and I are not going to get into that sort of thing.

Mr. Levitas. We are very fortunate, I want to make this a matter of record, to have Dr. Rowland on this subcommittee.

Mr. Molinari. Indeed.

Mr. Levitas. It brings an unusual perspective to our discussion. I am grateful for it.

Mr. Rowland. Thank you, Mr. Chairman.

Mr. Levitas. Those last couple of comments about the punitive damages and unpredictability raise some interesting questions to me which you may be able to respond to.

It is possible that we need accountants to respond to it. But my guess is, since I stopped practicing law actively, acquisition and merger agreements are written differently today than they were 10 years ago, and that generally accepted standards for accounting are probably now more focusing on potential contingent liabilities from environmental torts or environmental exposure.

We know of one situation already in this committee—which I won't identify by name—but in which a major Superfund settlement was made by a responsible party after years of conversation, because they were about to have a public offering, and their accountants or bond counsel would not quantify the exposure and they couldn't get an underwriter to take it, so they said, whatever the amount is, it is better than, as you say, the unpredictability, not knowing what it is, so they entered into a settlement agreement after years of discussions with EPA.

To what extent would a statutory solution to this type of situation, victims' compensation, provide a benefit to business, even by having some sense of certainty in terms of their exposure in situations of this sort?

Mr. Lundquist. I think it makes sense, but punitive makes it hard, first of all, it is against public policy to insure for that in most States. But you can quantify and obviously one of the interests of the insurance industry in our proposal was that it can quantify and can underwrite and provide compensation as kind of an internalized cost which, I think, is helpful to the business and hopefully to the people who get compensated.

My own view is that while punitive damage arguments are fascinating, they are kind of off to the side when we are talking about masses of people who simply are not being cared for. They should be regarded in a separate light.

Mr. Molinari. Would you yield?

Mr. Levitas. First, Mr. Baron would like to say something.

Mr. Baron. One of the things we did when we were on the Superfund, the attorney general of New Jersey, Jim Zasallie, was one of our members, and he made available to us staff members from his office and a couple of helicopters. We visited a couple of waste sites, and one example why we must leave punitive damages in these cases was a dump site owned by—I will change the name—I think the Burrelli Brothers was the right name. They had purchased a dump site, insured it for a known quantity, and then permitted it to be used for improper purposes, declared insolvency and after the suits came, the insurance people paid the money, and there was nothing left.

Without punitive damages, you won't be able to get to the Burrelli Brothers, you won't be able to get actually through the corporate veil and go directly to the source and deter this type of conduct.

It is absolutely essential that that remedy be left available.

Mr. LEVITAS. Mr. Molinari?

Mr. MOLINARI. I just wonder—do you want to respond, Mr. Light?

Mr. LIGHT. I just wanted to respond to Congressman Levitas' question about the possibility of a statutory solution increasing certainty, and that would be of an advantage to both the defendants as well as to plaintiffs.

I think we tried to deal with that in our prepared statement, knowing of your special interest in the Superfund statute.

I think that there is a possibility that it would be the case. In the Superfund Study Group, the group, by implication in its report and explicitly in the statements made in the Commission proceedings, rejected a Federal cause of action approach similar to the one that was set forth in the Senate Superfund bill in 1980, notwithstanding the possibility that there would be increased certainty if you had one uniform liability system.

The problem with that, one of the problems, is preemption. And there are two aspects to that. One is can you preempt State law or force the States, if you are worried about the burden on the Federal courts, can you force the States and the States alone to administer the newly passed Federal law without allowing access to the Federal courts?

The other aspect of it is that since you are limiting rights of the plaintiff or affecting rights of the defendant under State law by preemption by the Federal Government, won't that in the immediate future, the next few years, increase uncertainty?

I am not sure where Freeman comes out on this right now. I think he is a little unsure about where he would come out, except to say that the Superfund liability regime under sections 106 and 107 was very poorly drafted in 1980 for whatever reasons, and is a poor example to follow.

There are gaps in the statute, and that does create uncertainty. What issues are not resolved in the Congress are passed to the courts. You end up with a system which gets mired down in litigation when the stakes are high.

If you have a Superfund site, people want to resolve the situation, but if there are a lot of dollars involved, and nobody really knows what the law is on a particular issue, then you know, you probably on balance have a worse situation than you do if you just relied on previous State law.

Mr. **Molinari**. We discuss these questions in a theoretical vein, and you hear one argument on one side and you are persuaded by that and then somebody gives a counterargument, and you find yourself leaning in that direction. But when you start to look at actual experiences, I think the reaction of most of us is somewhat different.

I would like to share with you a couple of personal examples. Exxon, a rather big corporation. Exxon has a plant in New Jersey which, I found out through an informant, would retain the waste gases built up within the refinery for a period of up to 24 hours—that was the maximum period of time—and only release them, when possible, when the prevailing winds were coming toward Staten Island.

Now, I took that information and submitted it to EPA several years ago. They conducted an investigation and, indeed, found Exxon guilty of the practice. Exxon was fined $50,000, not a very large sum of money for Exxon, but when you consider the fact that Staten Island has one of the highest age-adjusted respiratory cancer rates in the country, your blood starts to boil a little bit. When you hear that, you wonder what is an effective penalty?

Obviously, you will never be able to prove that somebody got cancer because of that kind of occurrence.

Or take another situation, Mr. Chairman, something that floored me last week. Staten Island is across from a large petrochemical complex in New Jersey, and there are always complaints about different types of odors, cat urine, rotten eggs, and a whole host of other identifiable odors.

I took my congressional newsletter and utilized a questionnaire to pinpoint the experiences that my constituents had.

About a year or so ago, we sent the workup of that questionnaire's results to the State Department of Environmental Conservation and said, "This is what we learned from this process. Would you now try to do a better job professionally, as follow up, and see if you can identify who the real responsible parties are?"

Last week, I received a shocking report from the Department of Environmental Conservation of the State of New York. They traced back those specific odors to specific companies, again including some very large names—MERC company and others.

I was flabbergasted that we got such an excellent result where in the State was willing to identify the companies in those cases. Now, some of the odors may not be very harmful, but there are others that are, and you must speculate whether those companies would have known that they were guilty of, in some cases, releasing into the atmosphere some contaminants that were very harmful to the public?

What should we do about that kind of a problem? It is one that we have to consider, even if it is coming from one State and going to another State.

I ask you, what do you do in situations like this?

Mr. **Lundquist**. I have a couple of notions that would be, one at the Federal level, we should not permit as a nation the exporting of anything that you can't use in this country. That is a starting point which I don't believe has been done.

I think we are exporting problems abroad. You would like States to regulate themselves similarly so you don't tolerate one thing in one State that you can get rid of in another State.

That system of federalism, in a system of federalism, that is more difficult.

Probably the ultimate difficulty in these things, and it gets down to the reason why we ought to be eliminating toxicity, get the types of things they are using and hauling with the incinerator ships, because to make things benign is most important.

But the best example of what you cite now is I don't know what I would do if I lived in Tacoma, WA and worked there and had to now vote on whether or not to shut down that copper smelter which is the largest employer in town because they know it impacts on the lives of everyone there.

In my own—the point has been vividly made to me in looking at major industries in California because they have been shut down because of regulation, and the steel industry causing smog problem and all, seeing a picket line of workers in the plant carrying signs that say you can't eat clean air, which is a dilemma that I don't know quite how we resolve on any short-term basis except to compensate those that are hurt and get better and better controls over creating the problems.

Mr. MOLINARI. There is only one comment I would make to that: Anybody who had a member of their family who died as a result of those kinds of activities would not have any problem making a decision on how to vote.

Mr. LUNDQUIST. My experience with jurors is quite to the contrary in those cases. The older jurors say when we grew up, every carpenter had two fingers off their hand, the folks who lived near the plant or worked in the plant that my dad did, all knew they had silicosis because of things in their lungs. They accepted it as the way the world was in those days.

Indeed, these gentlemen here smoking are accepting the fact that they are, every time they do that, cutting off ascertainably probably 15 percent of their lives.

People make a lot of decisions that you don't expect. I found jurors, when you get that kind of situation before them, make that sort of judgment and again, it does not mean I think we should tolerate it.

I think we shouldn't. But I suggest to you that that is not necessarily a consistent objection.

Mr. BARON. Might I respond to that somewhat? I think Mr. Lunquist is a superb lawyer, because he is now talking about closing down the largest employer in a city as opposed to whether Exxon is going to release holding gases at night when the wind is blowing the other direction. They can't be compared, really.

My assistant Mr. Wodka, who used to be assistant safety director of the Oil Workers Union, reminded me that in 1969 and 1970, the OCAW provided testimony about holding those gases by Exxon in New Jersey, and that there was a problem that was identified then.

I suggest to you that fining Exxon $50,000 is probably a little less than requiring me to buy Mr. Lundquist lunch today. This is not an effective remedy. I further suggest to you that the effective remedy would be that if those citizens who are aggrieved and who have proof of the case of the problem band together and file a lawsuit against these companies, seeking punitive damages for these emissions, that they will stop.

Mr. LUNDQUIST. I agree with Fred on that. I didn't mean to take Mr. Molinari's example, I just tried to put the problem in a larger context.

Mr. LEVITAS. That is the way I took your answer.

Mr. LUNDQUIST. I think it is more of a dilemma, and I agree that you, Congressman, you and Fred were right in the Exxon situation or any situation like that. My comment was not meant to turn that around or condone that.

Mr. LEVITAS. I would also say, not in defense of the fact I smoke cigarettes, but——

Mr. ROWLAND. That is indefensible, Mr. Chairman.

Mr. LEVITAS. Also free-fall parachute jumping and hang gliding and a lot of other things I choose to do, it is another thing for Exxon to do it to me.

Mr. LUNDQUIST. I agree.

Mr. LEVITAS. Which is a lot of difference.

Mr. MOLINARI. I was just interested in Mr. Lundquist's acknowledging that as a practice that should not be tolerated. Would you sanction punitive damages in a case like that?

Mr. LUNDQUIST. That is certainly one way. That is a situation where I don't have much trouble with punitive damages. To defer current manipulative conduct they may be effective. My definition of punitive damages is basically outrageous conduct.

If I found out that I lived in your constituency and they were dumping stuff on me, I would be pretty outraged.

Mr. MOLINARI. Thank you.

Mr. LEVITAS. Let me go into another point with Mr. Light that Mr. Freeman had trouble with.

He suggested that there might be a good reason to force a choice to a potential claimant into going into tier 1 or tier 2 remedies.

Mr. Freeman expressed himself as being concerned that if you gave a claimant the opportunity to pursue both they would in fact use the tier 1 compensation as a means of financing the litigation in tier 2 that might occur.

Now without taking a position myself on this thing, but I think fairly characterizing what I understand Mr. Molinari's position to be, he would say precisely that is what ought to occur. You ought not preclude a person from exploring and pursuing full recovery simply because he doesn't have the economic means to do so, and that if a person is given living expenses and is compensated minimally that if the damages are actually greater than that that would be one of the great social benefits resulting from a tier 1 compensation to let this individual pursue tier 2 recovery.

I think I have characterized Mr. Molinari's view on that matter.

Mr. MOLINARI. Right.

Mr. LEVITAS. How would you comment on that?

Mr. LIGHT. Well, I think that in the Superfund Study Group if there was one issue on which it spent more than hours, indeed days, it dealt with this issue of the relationship between the tier 1 and the tier 2 remedies. And the debate is laid out within the 301(e) report which Mr. Molinari has, in some detail there.

On the one side there were those within the study group, for example at one end would be Professor Jeff O'Connell, who suggested that if we were really going to a no-fault type of system of compensation, for that class of claims in which you are providing no-fault compensation, you necessarily have to provide for an exclusive administrative remedy because of this problem that if you add to the costs of the administering the administrative system the costs of allowing the parties to also go in and seek further relief in the state tort system then the overall system will just be too costly to administer.

I think what was behind Professor O'Connell's notion—and he has written on this quite a bit and in fact he has a separate statement in the Superfund report you can look at—is that when you go to these administrative type systems allowing some kind of relaxed burden of proof either through a presumption or through some other mechanism allowing evidence that would not be admissible in a trial, then you are going to overcompensate. You are going to provide some relief to people who probably would not have recovered under the traditional standards of proof.

That cost has to be picked up somewhere and on balance as a cost control mechanism, you need to just prohibit, from O'Connell's point of view, access to the State tort system.

On the other side we have the proponents of the view of free access, which I take it is Mr. Molinari's point of view, which is that even though there may be some overcompensation in the tier 1 system, in that system we are talking about compensation and we want to get the dollars out. In the area of tort law we have other objectives seeking deterrence of evil or bad conduct, and that those are both valid purposes. So there is no real rationale for denying, putting up some barrier between the tier 1 and tier 2 remedies, in their view.

The Superfund study group itself adopted after much deliberation a compromise proposal which Freeman did not favor—he did not vote in favor of it—which was to allow access to the State tort system, but if the recovery in the State tort system, was less than 125 percent of what one got in the administrative system, then the judge would have the authority to assess costs against the plaintiff, both expert witness fees and attorney's fees.

I think that was probably on that particular day the only compromise that would have prevailed. At the last meeting of the group, a group of the attorneys, a subgroup, adopted a second compromise position. This is the position that Freeman adopts in his separate statement and also in a joint statement which is appended to the Superfund report. The four members that signed the separate statement being Professor Anderson, Professor O'Connell, Judge Breitel, the retired senior judge of the New York Court of Appeals, and Mr. Freeman.

In their separate remarks, Weyman Lundquist and Warren Spannaus, one of the attorneys general from Minnesota said that Congress ought to look at this as well.

Well, that proposal on balance would have shifted more claims into the tier 1 system by enhancing the benefits that were available in the tier 1 system to allow some recovery for what we might not—what Tony Roisman might not—call objective damages—for some limited pain and suffering award. In exchange for enhancing the tier 1 system this group of four—including Mr. Freeman—would have tried to systematize the payments for pain and suffering through some scheduling of benefits similar to procedures that operate, I understand, in some workmen's compensation systems in other countries.

That trade was made and then there was the barrier that would be created between the tier 1 and tier 2 system which was as follows: Instead of having an exit barrier, an after-the-fact determination of whether you can go into tier 2, you would have an entry barrier in which a plaintiff who wanted to go to the State tort system would, after having received his benefits in the administrative compensation system, go to a judge. And the judge would determine whether the evidence which was available at the time he had filed the administrative claim and the subsequent evidence which he had discovered, demonstrated that there was a reason to allow this guy into the tier 2 system.

He could allow that. There could be an exception made in the specific circumstances of that individual. But on balance the normal process would be one in which the claimant would choose a quicker, more limited kind of benefit in tier 1 including some pain and suffering award, foregoing the punitive damage claim, for example, because he might not see that there was that kind of outrageous conduct in the case. So he would go for quicker recovery; or go for the tort law recovery with the higher risk but with the possibility of greater recovery at the end of the line.

So that is where Freeman came out. It really was not a pure binding election there. There would be exceptions and it became an entry rather than exit barrier approach. In fairness to the study group I think the proposal in its ultimate form, and as pictured in the joint statement at the end of the report, was not considered because of time. The study concluded 6 months after the legislation called for it, and it was time to get the report out.

Mr. LEVITAS. Let me ask you this, Mr. Lundquist.

You raise the question of the problems associated with long latency periods. We have had a good bit of testimony about that. That creates a whole host of problems. Not only the problem of the exposure of the responsible party and potential defendant, but also enormous questions as to proof, cause and effect, and who is around, not just the defendant but the plaintiffs, that whole question.

It is particularly true in the question of toxic chemicals—carcinogens, mutagens and things of that sort.

How do you feel that this subject of long latency, given the problems of proof deficiency, or impossibility, on the one hand; and the open-endedness on the other hand? How should it be addressed in terms of proof, evidentiary proof, presumptions, and statute of limitations?

Mr. LUNDQUIST. To reverse the order, the statute of limitations I don't think is troublesome. I think if there is one thing that myself and every member of the committee conceded is the statute of limitations begins when you find out, reasonably should have known. I think that there is no problem with that.

Mr. LEVITAS. Known of the injury?

Mr. LUNDQUIST. The fact you are suffering——

Mr. LEVITAS. Not exposure.

Mr. LUNDQUIST. That is right. Not the exposure, the injury. We provided 3 years thereafter within the system that we proposed.

Second, I think this is where the idea of presumptions is extremely important and it is something we don't know enough today to define these precisely. Indeed I have tried to, in evaluating this to talk to scientists not retained by plaintiffs or defendants and indeed in California we have some that are prohibited from taking compensation, those are members of the university system. I have sat down with particularly Mr. Ed Schmuckler, head of pathology at the University of California, San Francisco. He is a leading pathologist nationally known. There are very few areas where there is the precise definition you could start out with today and again any individual might have a lot of problems to do it.

So there we dealt with the situation if you have exposure and if you have an injury that comes from that exposure that is enough. Now, to get to that level, presumptions are based on something happening more likely than not. They are based on credible evidence. Obviously in the initial instance we are not going to take the common cold and the fact somebody caught a cold and worked in a wet, open building. That isn't the sort of situation we are talking about.

But we are talking about a known toxic and an injury that would flow from it and you presume that somebody—somebody in tier 1 has the injury or disease that does that. Then you have to evaluate the disability.

You have that. That is one of the presumptive factors.

The other thing you do is that you allow the Federal Government through the development of toxic guidelines if you will, toxic documents I think we called it, to make tests and say that in this industry if you are exposed to certain things it is known that this exposure will cause a disease and you will be able to use that by way of proof and use that level of scientific knowledge in the case so you don't have to go to the individual proof.

The reason I think this is so important and this speaks to a personal view of mine, I think the system has failed the victims in that the transactional costs have really benefited the lawyers. On the plaintiffs side you have to figure they have gotten 25 percent to a third. The system has paid on the other side, I suspect the defense bar across-the-board collects as much as the plaintiff's bar. It is an enormously expensive thing for injuries which generally have not shown themselves as yet to this day to be severe.

Again I say that because we are talking about the mass of claims now and I am not saying that the person who comes down with cancer and can establish it, doesn't have great injury but we leave that to the existing system. What we were concerned with was to get a quick, efficient and economic system. That was the reason for the presumptions and the approach taken for eliminating those transactional costs that underlie that sort of approach.

Mr. LEVITAS. Now, Mr. Baron, let me turn to you for a moment. I am sure I am oversimplifying but essentially you think the existing tort system works with some modifications and finetuning. Yet I think it is fair to say that this whole question of toxic torts, environmental health damage, the whole business of chemicals and people and environment is a relatively new phenomenon. I grew up in an era when we used to listen to the DuPont Co.'s radio program "Better Living Through Chemistry."

If I said that to my kids today they would scoff and laugh at me even though there is still truth in it.

In every major period of history, either our technology has changed or our communications and transportation have changed, or our commercial institutions have evolved, and the law has created essentially new systems to deal with them; the whole evolvement of the common stock corporation to deal with the massive investments of the mercantile period, and the expansion into the new world during the colonial period; Lord Mansfield's development of commercial law concepts to deal with the whole new system of interchange of that period; the question of workers compensation supplanting the fellow servant doctrine that I remember; the development of a concept of equitable mortgages to protect people who were being foreclosed in an era that you had seen this tremendous mushrooming of middleclass land ownership.

Buick v. *McPherson* was in the 1930's, wasn't it? We didn't get a system of negligence evolved until the 1930's because we had a new system, and in recent years the whole idea of no fault insurance. I was in the State legislature when we passed our first no fault insurance. So the law has revolutionized itself to meet with revolutions going on in the economy and in the technology.

Now, what is wrong with saying, look, we have now discovered toxic torts, chemicals, we have problems of proof, we have problems of long latency, we have problems of cause and effect. We have these variety of problems and notwithstanding the fact that we still may not have the ultimate proof in terms of certainty scientifically. I think if you asked the members of this panel who have been listening to testimony for 2 years now, that we would say there is a problem out there and people are suffering.

Why shouldn't we develop a no fault type system for compensation of these victims and recognize, like in the case of workers compensation, it is a trade-off, or no fault insurance, it is a trade-off? You either provide an efficient system of some degree of certainty of compensation or you bet on the outcome, that there is a good system out there, that the effective lawyer and the skilled expert witness can capitalize.

Why shouldn't we simply go to a sort of no-fault system or workers compensation system?

Mr. BARON. For the same reasons we have not with all the other problems you have described.

The *Buick* case threw out privity, this was done by the common law. The California Supreme Court back in the early 1960's adopted strict liability. That spread. It was done by the common law.

Comparative negligence systems in many States have been implemented by the common law in response to a problem.

Most all of the case law now on the statute of limitations in latent disease cases adopt a discovery rule, again done by the common law.

I believe that the common law is a moving flexible body of law that will deal with these problems in an efficient manner and will permit victims to be able to really and truly have their day in court, where they can receive full compensation without creating some nature of an administrative monster that is going to gobble up the funds in the administration of the system that essentially provides very sparse benefits anyway.

The workers compensation system, unfortunately, is the classic example of that because, as probably everyone in the room knows, for a serious injury case, workers compensation benefits just about anywhere, with some limited exceptions, are inadequate. They overcompensate people with a sore back syndrome or cervical strain, but a victim who has a serious injury doesn't receive adequate compensation through workers comp.

The only way they will truly be compensated will be through movement of the common law which is occurring now. There are exceptions. New York is one of two States where the highest appellate courts have refused to adopt a discovery rule for the statute of limitations. The other 42 State high courts have adopted one in one form or another, and, definitely, legislation is necessary on that issue in that State. That doesn't mean we should throw out the whole tort system.

I believe generally it will evolve to a very workable, efficient system.

Mr. LEVITAS. Well, you lead me into my last question unless either Mr. Light or Mr. Lundquist wants to comment on that, but my last question is a more general philosophical question which I address appropriately to my brother lawyers.

One of the problems and criticisms I have of the 301(e) study is that it was essentially a lawyer's study. And I do not denigrate that aspect of it. The amount of expertise and knowledge and insight into the whole subject matter, not just the legal side of it, is irreplaceable. There is no other place you can go and find that much broad expertise not just from the fee-getting-fee earning business, but just from involvement.

Yet the problem of victims compensation, toxic torts, in today's world is not just what is the best legal remedy. It really isn't. It is a question of spreading the social cost of the world we live in, and we are going to continue to live in, and the social cost to the people who will pay for it and that means everybody, because we are all receiving the benefits from it.

For example, people who are suffering from PCB exposure are people who also benefited from the electrical power generation that came about as a result of that insulation material. Rightly or wrongly, I am just saying we are not talking about the traditional situation.

There may be other important significant contributors to how we resolve this matter. So, I suppose what I am asking is how do we divorce the vested interests of the plaintiff's bar, or the defendant's bar, or the insurance industry and their legitimate interests, from the legitimate social interests.

Mr. Rowland commented to Mr. Molinari—if you don't mind me saying?

Mr. ROWLAND. Go ahead.

Mr. LEVITAS. He said: "A lot of this sounds to me like just another way for lawyers to make some money one way or the other, you know, defending these cases, being plaintiffs' attorneys, and so on, and that is not what this ought to be about."

That is certainly not where we are coming from. So how do we deal with it? I realize this may sound like a nebulous question, but believe me, that is the last question that has to be answered before we make decisions, whatever they may be.

So I wonder if any of you wish to comment.

Mr. Lundquist?

Mr. LUNDQUIST. First of all, among the things that I pointed out that we need are—I had a list of things that if you were looking to the future, what we really need and what I feel is, lacking to some extent, were independent scientific medical-legal-insurance, actuarial people to give us the dimensions of the problem.

I really don't think that we have complete understanding of the dimensions of the problem. Further, we probably need philosophers because the person who dies at 77 and has lost 5 years of his life but when he was born had a life expectancy of 51, raises philosophical questions in there. So, we really need that input.

Now, what business do we have as lawyers in this?

I think one of the things that lawyers do or try to do is communicate. To the extent we communicate diverse points of view from science, from business, from insurance, from personal injury clients, we do bring ideas which reflect the different interests of others. So I think to that end we are helpful. But I am not satisfied that we have come to the solution.

Indeed, if I might stand up, I am concerned and I have thought about it a lot. Perhaps we have done what the fellow did who wanted to buy a suit. He didn't have a lot of money and he went to the store and said, "I want a suit," and the salesman said "I got this one." He said "Put it on. That looks nice."

How much is it?"

He said "A hundred dollars. That is a terrific suit."

"I'll take it for a hundred dollars. But this pants leg is a little long. Can you alter it?"

He said "No, we don't alter suits that cost only a hundred dollars. Just put your hand in your pocket and hold it up and it will be all right."

The guy said "All right, but the waist is a little loose."

He said "Look, stick your hand in your jacket and hold it up and it will be fine."

The guy said, "Look, the shoulder is falling off."

He said "Get your chin over there and hold it." And the guy buys the suit. He goes out down the street past two elderly ladies and one says "Look at that poor man, all crippled up like that." The other said, "Yes, but it isn't all bad, the suit fits beautifully."

End of comment.

Mr. LEVITAS. Mr. Baron?

Mr. BARON. Essentially my profession, the plaintiff's bar has been maligned probably as much as any group in the world and probably for very good cause under some circumstances.

Quite honestly, I would far prefer handling aviation cases or divorce cases if there were no toxic chemical cases to be involved in. I think that too often we are judged only by our economic interests in the case.

There are many of us who practice this type of law who feel very strongly about it and who feel strongly that there are two purposes to be served here, certainly victim compensation is No. 1. But No. 2, I learned in law school from Dean Keaton years ago that the purpose of the tort law was to deter conduct and unless and until someone comes up with a better way to deter conduct I believe that the tort system is the only functional way to do it.

We are dealing with the conceptual and practical. Conceptually it would be nice if the victim walked in, the doctor said yes, you have this disease and he was given a check and off he went and there was no transactional cost. That would be a wonderful system.

Unfortunately, that is a pie-in-the-sky thing. It is not going to happen. The Black Lung Act has proved that over and over and over. It is not a situation that is feasible.

There is a need for attorneys to handle these cases. There are bona fide disputes. No one has come up, including our group who studied this issue thoroughly, with a better way of doing it that actually in practicality works. That is the bottom line.

There are wonderful concepts of how it should occur, but when you get right down to it, how do you serve these two functions, we can't find a better way to do it.

Mr. LEVITAS. What do you think about the suggestion of simply shifting the burden of proof to the defendant?

Mr. BARON. I would like to speak to that because I think everyone on the group was very amazed at my position when I took it. They assumed I would like to have a presumption in favor of compensability in any case where it is tier I or II. My practical experience, however, teaches me otherwise. If you develop a presumption that chemical A causes disease B and C, and then your claimant comes in and he has disease D. You have really lost the whole shooting match because the judge or whoever the trier of fact is going to be says, hey, the presumption is that this chemical causes B and C, you have D, it can't be related.

It is a double-edged sword. It is a difficult proposition. If we knew all there was to know about these toxic chemicals and about their effects, then perhaps that would be a rational approach. But the fact of the matter is we don't and because of that fact I think that the presumption probably hurts the victim more often than it helps.

Mr. LEVITAS. Mr. Light, if you would take that last question to which Mr. Lundquist and Mr. Baron responded, that rather general question I asked, we will get it for you from the reporter so I won't have to repeat it, if you would ask Mr. Freeman to respond for the record I would be grateful.

[The information received follows:]

HUNTON & WILLIAMS,
Washington, DC, August 16, 1983.

DEAR MR. LEVITAS: Thank you again for your courtesy to my associate, Dr. Alfred R. Light, at your hearing of July 26th and your kindness in making my prepared testimony a part of the record despite my unavoidable absence. I have had an opportunity to read the rough transcript of those hearings and will respond to your invitation to offer a few thoughts on the concerns you, Mr. Molinari, and Dr. Rowland expressed at the hearing on July 26th about how we might "divorce the vested interests of the plaintiff's bar or the defendant's bar or the insurance industry and their legitimate interests, from the legitimate social interests." A fear expressed was that toxic tort litigation may become "just another way for lawyers to make some money," especially in light of the increasing number of attorneys in this country.

First, let me say that I concur with your view that the Superfund Section 301(e) Study was handicapped by being composed only of lawyers. Judge Breitel and I in our separate statements in the printed report pointed out the need for broader perspectives from outside our profession. The study suffered, as do most of the law review articles in this field, from that "lawyers only" limitation of perspective. But we suffered under other limitations as well. As Dr. Light pointed out in response to a question from Mr. Molinari at the hearing, the Study Commission also suffered from the same difficulties which this subcommittee recently experienced in trying to obtain information from EPA and the Justice Department as to the scope or dimensions of the hazardous waste exposure problem.

But, on balance, I think the 301(e) Study still provides you a useful first step to thoughtful consideration of the compensation issue. What is now needed is for Congress to insist that others outside the legal profession come forward with information directly relevant to the scope and dimension of actual or potential injuries and how persons actually injured are or are not being compensated under the present system. We also need some sort of objective evaluation of the efficiencies and inefficiencies of the present system and porposed changes to it. The recent Rand Report showing that only $1 out of every $3 paid out in connection with the current number of asbestos related tort claims goes to compensate those injured illustrates the need for equivalent analysis in this area of tort law. But to urge caution does not mean that we should turn our backs on the concerns that have been expressed in these areas and be content with the status guo. Quite the contrary. A growing number of people are concerned with these problems. Various studies are underway involving lawyers and non-lawyers that should permit a more reliable assessment than is possible given the current state of knowledge. They are also seeking what may or may not be the best way to improve or change the present system. For example, one of the most interesting projects, jointly sponsored by the Yale Law School and the ABA's Ad Hoc Committee on Tort Law Reform, is a conference of lawyers and non-lawyers in the Spring of 1984. Its objective is to see how the present system is actually functioning and then to see if reforms would be efficient and equitable. The outcome of this and other studies now underway will be directly relevant to where change is needed and whether the final changes would actually make things better rather than worse.

With this preliminary caution, from my perspective as a member of the Superfund Section 301(e) Study Group and my subsequent study and analysis, there does appear to me to be some general principles, which if adhered to in a federal legislative proposal, would make a compensation system serve legitimate social interests to a greater extent than the present system.

1. In areas where there are major limitations of scientific and medical knowledge as to (i) existence of injury, (ii) extent of injury, and (iii) causation of injury (i.e., the nexus between exposure and injury, particularly where there may be multiple causes), any federal legislation should assure that the principles and procedures governing determinations of injury and causation be fair and objective. That fairness and objectivity must be perceived by both those injured, on the one hand, and industry and consumers, on the other. We should not have a tilt in either direction. A tilt in one direction would be unfair to victims; a tilt in the opposite direction would impose economic burdens which are not only inequitable but which amount to a misallocation of resources which the nation can no longer afford.

These considerations argue in the first instance against any statutory presumptions. There is a line to the effect that it is not what we don't know that hurts so much as the things that we know that are not so. Freezing into a statute a set of presumptions which may well be disproved, at least in part, by later advances in knowledge is highly unwise. The Black Lung experience on presumptions is instructive.

I believe it appropriate to provide for the preparation of health effects documents which might be considered in a proceeding to recover compensation. If such documents prove useful, they could reduce substantially the waste apparent in case-by-case repetitive adjudication of the same scientific or technical issues. This preparation must be subject to three essential safeguards against abuses which only benefit lawyers and other intermediaries.

First, the documents should be prepared by a body totally independent of the agency or agencies charged with regulatory functions or with the function of administering compensation. An appropriate body to prepare such documents might be the Center for Environmental Health in the Centers for Disease Control or the National Institute of Environmental Health Sciences within the National Institutes of Health.

Second, such documents should be prepared and issued in accordance with a rulemaking process governed by the Administrative Procedure Act and should be subject to judicial review under that Act.

Third, the weight to be accorded such documents should not be prescribed in advance but should be determined by the agency administering compensation on the basis of the document's own internal quality and of its relevance to the circumstances of the particular claim.

2. Any Federal statutory remedy should be exclusive and should supersede all concurrent state tort law rights and remedies, particularly if the statutory remedy is to provide for recovery of non-economic damages. Different and overlapping Federal and state law rules as to liability (causation, fault, defenses), statute of limitations, punitive damages, and measure of compensatory recovery would lead to delay, confusion and waste harming both victims and defendants, and benefiting only lawyers whose need is based on such unnecessary complexity.

3. A statutory remedy, particularly if it is to dispense with proof of fault, should put reasonable controls on recovery for pain and suffering and on other types of non-economic, subjectively determined damages. In particular the Federal administering agency should be called upon to develop a uniform schedule prescribing ranges for types of non-economic recovery, along the general lines suggested in the joint Anderson, Breitel, Freeman, O'Connell Statement in the Superfund § 301(e) Report.

4. There should be reasonable controls on litigation expenses. This could involve replacing the contingent fee with a system for separate award of a claimant's attorney's fee above the amount of recovery and a concomitant prohibition against applying any part of the recovery to legal fees. Consideration should also be given to mandating settlement offers, to financial disincentives for groundless claims or defenses, and to financial disincentives for unreasonable litigation tactics (claimant or defense).

5. A statutory system of no-fault recoveries should not be coupled with conventional tort litigation for punitive damages. The Solid Waste Disposal Act currently provides both criminal sanctions and civil penalties with a citizen's right of action to seek injunctive relief. This statutory punishment/deterrence system should be exclusive and should expressly supersede any common law right of action for punitive damages.

6. Claims for compensation under the system should be directed to the owner/operator of the facility at the time the exposure occurred; the owner/operator should be solely liable for payment of such claims ("channeling"). The owner/operator should be required to prove financial responsibility to meet claims, through liability insurance or equivalent. The aggregate liability of the owner/operator should be limited to the required amount of financial responsibility. Any owner/operator's right of recourse against generators or others should be based on rules of comparative responsibility to be determined and applied as a matter of Federal law, if there is to be such a right of recourse. The Federal Government should be responsibile only with respect to (i) so-called orphan sites or (ii) sites where post-closure financial responsibility coverage has expired, or (iii) amounts above the required level of financial responsibility. Adoption of this proposal could substantially reduce the aggregate social costs of the system through reductions in legal costs and elimination of duplicative insurance premiums.

This system would (i) minimize Federal involvement in defense of claims, (ii) assure private financial responsibility to meet claims, (iii) permit workable private insurance arrangements by avoiding pyramiding of coverage through rules of joint and several liability, (iv) to the extent practicable, assign responsibility to the particular activity causing the harm, (v) involve use of Federal funds only where private sector resources are not available at all (such as orphan sites) or where those resources have been first applied (up to the level of required financial responsibility), and (vi) substantially reduce aggregate social costs of the system (which would ultimately be borne by the general public once the system is in place).

7. A number of factors indicate that any such system be administered through an administrative agency rather than through the courts. State agencies, either worker compensation boards or other bodies, should be preferred if they qualify under Federal guidelines. The guidelines should be developed by a separate office within a Federal agency that is not "mission oriented" (such as EPA, for example). That office should assume the claims administration responsibility in any state where a state agency has not applied or has not qualified.

8. Where a state agency administers claims, appeal should ordinarily be through the state courts. Federal courts should have appellate jurisdiction only where the federal agency finds that the state agency was in error or where the federal agency has administered the claim.

I should emphasize that those ideas represent my current preliminary thinking as to a more efficient compensation system. By decreasing the need for lawyers and other intermediaries, it should substantially reduce aggregate transactional costs and, at the same time, assure that a higher percentage of the total social costs of the system actually go out as direct payments to compensate those injured.

Sincerely,

George C. Freeman, Jr.

Mr. Levitas. Dr. Rowland?

Mr. Rowland. Thank you very much, Mr. Chairman.

I do not like to find myself in an adversary position with attorneys, but sometimes I do, even with my colleagues here. But we are all looking for the same thing, I think, we want to compensate those people who have been harmed by dangerous chemicals and I believe there are people out there who have been harmed by these chemicals. I do not know at this point how severe the damages have been, what the long-term effects will be. There are many, many things that are undetermined.

I am particularly interested in the study group and the Superfund idea and the things you talked about, Mr. Lundquist, as a possibility of dealing with the problem.

Let me say why I am concerned about it. I know, as a physician, that we have been saying in this country for many years we need more doctors and this would make the quality of health care improve and costs come down.

So we have more physicians and we are going to get even more and just the opposite of that has happened. There are doctors out there doing things they ought not to do, because there are too many of them, and they are hungry.

So the quality of medical care is coming down, and the cost of medical care is going up. I don't impugn or even question the motives of the toxic tort litigation group because I don't know.

As you say, this is a new area of law. Toxic effects of chemicals, as we look at them now, is an entirely new area. Also, we talked about lead poisoning and how long we have known about lead poisoning. We have known about it for a long time.

In fact, I think we may have established that the Romans had lead poisoning and that may have been the cause of the fall of the Roman Empire. But the fact is, we are learning more and more about lead poisoning.

At one time, we thought it was neurotoxic; we are learning that it causes mental retardation. We are learning more and more about it. So I think that it takes a long time to really determine the effects of these chemicals on individuals.

The toxic tort litigation group—and we talked about this going out—causes me a great deal of concern because I am concerned that more and more suits may be filed in the coming years. It doesn't cost much to file a suit, of course.

But a suit does cause a great deal of anxiety and it can cause a great deal of cost and this cost will simply be passed along to the people that receive whatever it happens to be that this company is producing.

In fact, it could go to the point that the company could be put out of business with a lot of dislocations. There are all kinds of things that could happen in that area. Things are getting cheaper, you even refer to the fact that while one person said it might cost $200,000 to get a case together, you indicated it could cost much less than that, and maybe if we did this in large numbers, it would be even cheaper than that so far as getting medical examinations and Don and Mr. Roisman earlier this morning said that, I believe, that the faster gun is the one that wins sometimes.

So, I have a lot of concerns about what is taking place in this area. I want some compensation for the people who have been injured, but I think it is going to take a long time to prove that, and in the meantime, perhaps we need to look very closely at what has been taking place with Mr. Lundquist, not ruling out the possibilities of the victims' compensation law.

I think we need to continue to look at many things. I just wanted to express my feelings about this, Mr. Chairman, and any response that anyone may have, I would be delighted to hear it.

Mr. **Baron**. I would like to respond briefly, if I might, Mr. Congressman.

As far as forming groups of lawyers handling this, the purpose was to solve the problem Mr. Molinari pointed out. There are not enough lawyers now who are familiar with these problems, and when a client comes into your office and you know nothing whatsoever about the problem, you have got to either find somebody who does or learn about it yourself and, therefore, that is the reason for this group.

There is no profit motive involved as such. There is a professional efficacy motive involved in teaching other lawyers how to deal with these types of cases so they can recognize and handle them.

Second, I am glad it doesn't cost much to file suit. I disagree with the Chief Justice. I would prefer seeing more lawsuits filed for valid claims. It certainly keeps people off the streets with guns and knives.

If people have been injured and they can prove in court who caused their injury, they should be entitled to access to the courts. We shouldn't create barriers to keep these people from asserting the rights that they have in our country.

Finally, if companies are put out of business because they have harmed other people and because they have to pay for that harm, so be it. I don't think that there is anything that is so absolute that it should never be attacked.

If a company is engaging in practices that cause a substantial harm to other people, then indeed they should pay for it. I firmly believe that.

Mr. **Rowland**. Let me make one comment about what Mr. Lundquist said, that after it, why more people try to get on the bus than before the wreck. I appreciate your statement.

Mr. **Lundquist**. I think that—I said this last week to the ninth circuit, judicial circuit. I think lawyers are part of the problem. The explosion of big law firms—and I come from a big law firm—is due to the fact that lawyers get paid by the hour. It is thought the more you can do on behalf of the client, the more hours you put in.

I do think that that element is there. I think that the whole notion of too many lawyers from unaccredited law schools, indeed someone has said that in California, with our current rate of admission by the year 2007, everyone in the State will be a lawyer.

That leaves us only ourselves to sue. Obviously, that isn't a good thing. At the same time, I think lawyers and particularly the jury system have functioned very well. We have caused a lot to change, and I think we need to preserve that.

But my own philosophy in this two-tier approach was to try to get rid of some of the legal costs on both sides, defense and plaintiffs, and get the money to individuals. I think if you could pull lawyers out of that, or limit the fees on either side by way of plaintiffs of defense, you are doing a good thing. You should do that.

So, lawyers are part of the problem.

The other thing is, I keep coming back to it, my 30-year history with environmental movements from living in Alaska to California to Massachusetts, is to prevent things. I think acid rain or any type of mass pollution, this is where we ought to be looking, because we have a problem now.

Hopefully, in 20 years, we won't be looking at the same problem.

Mr. LEVITAS. Thank you very much.

I should really send each of you a copy of my Law Days speech this year to Emory University. You have all addressed it in your final comments. There is a Parkinson's Law at work in the legal profession, the amount of business expands to subsume the amount of lawyers.

I know you have all heard the one about the lawyer who got out of law school in a small town and was starving to death. A few years later, he saw a friend of his, a lawyer who had been starving to death, who had a nice suit on, and looked prosperous driving his BMW and the friend says, "How is it you had such a great turnaround economically?"

He said, "Another lawyer moved to town." And until then, he didn't have any business. I am sure you have all heard that story.

I think there is a real problem. This is part of it. This is what Dr. Rowland is talking about. That is why I was so pleased he mentioned that. There is a feeling in this new area of the law we are addressing where the lawyers come out on it.

I don't just say that to plaintiff's bar at all, really. It is a question of "Lawyer, heal thyself," I suppose, and we as a profession have to be worrying about that more and more as a profession, which we are still, I suppose.

But anyway, gentlemen, I can assure you that your testimony has been most provocative as well as enlightening. I learned a great deal today from what each of you had to say, and the differing views that were so well and articulately expressed by each of you will, I think, give us a basis for a much better understanding of this whole question of toxic tort and victims' compensation as we look to the future.

I have a feeling that your contribution will help make our final report and recommendations to Congress of benchmark value.

I want to express my appreciation to you.

Tomorrow, we continue with these hearings. We have Khristine Hall, cochairman of the Toxic Program of the Environmental Defense Fund; Leslie Cheek, III, vice president, Federal Affairs, Crum and Forster Insurance Cos.; Sheila Birnbaum, associate dean and professor of law, NYU Law School; and Dr. John Beljan, representing the American Medical Association.

Mr. Rowland, maybe you would want to come back and watch that?

He is going to explain the position of the AMA on dioxin, as I understand.

Mr. ROWLAND. I will be here.

Mr. LEVITAS. So, tomorrow, we have a very full agenda, and I hope the schedule on the floor of the House will permit us to get through it with a minimum of interruptions.

Again, gentlemen, thank you very much. I appreciate your being here.

The subcommittee stands in recess until 10 a.m., tomorrow morning.

[Whereupon, at 3:50 p.m., the subcommittee recessed, to reconvene at 10 a.m., Wednesday, July 27, 1983.]

HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

WEDNESDAY, JULY 27, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 11:10 a.m., in room 2167, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Mr. Levitas. The subcommittee will please be in order.

First of all let me apologize to everyone here for the late start of these hearings. Congressman Molinari and I were involved in preparation for some other matters that had to be done this morning before we could begin these hearings and it was unavoidable. I express our apologies to everyone who may have been inconvenienced by it.

We are very much looking forward to the testimony today. Yesterday we heard from a panel of attorneys as well as a former official of the Justice Department who has been active in the area of litigation in the toxic tort field. It was a hearing that demonstrated once again that there is great diversity of opinion and approach in this entire matter. At times acrimonious.

But there still seems to be a realization that we are talking about a new area of law and a new area of concern, of public policy concern, that requires the attention of Congress to address in an objective way, based upon a full record of facts. That is where we begin today, to continue to make that record.

We will be hearing from Khristine Hall who has had experience in the field of victim compensation on behalf of environmental organizations, and will describe the problem of victim compensation as viewed by the environmental community. Her specific activities in this area, in fact I think I recall reading an article or a panel that you participated in on environmental matters not too long ago.

Following Ms. Hall will be a panel of individuals reflecting the views of the insurance industry. Since the whole question of victims' compensation and the problem of toxic tort as well as hazardous waste has created an entire new dimension for insurance carriers, we feel it is very important to shed some light on the subject from that perspective.

Then we are finally going to hear today from Dr. John Beljan appearing in behalf of and representing the American Medical Association. He essentially will talk about dioxin, agent orange, and the AMA's position on this matter about which there has been a great deal of controversy swirling for a number of reasons including resolutions and rescission of resolutions that occurred at AMA's recent meeting in Chicago.

I am looking forward to a very interesting day of testimony.

One other point; the House will be going in session at noon today. My guess is that we will have interruptions during the course of the day and they are to be expected. It is par for the course. It is also possible—and I will confer with my colleagues later—that we will take a short break for lunch at some appropriate time.

With those few comments I inquire whether my colleague, Mr. Molinari, has any statement.

Mr. **Molinari**. No, Mr. Chairman, thank you.

Mr. **Levitas**. Mr. Rowland.

Mr. **Rowland**. No, Mr. Chairman.

Mr. **Levitas**. If not, may I ask you to raise your right hand, Ms. Hall.

[Witness sworn.]

Mr. **Levitas**. Thank you very much, Ms. Hall.

We appreciate your being here today. I would request of you and the other witnesses to confine your opening remarks to approximately 10 minutes so that we can get into questions.

We welcome you and look forward to your testimony. You may proceed as you so desire.

TESTIMONY OF KHRISTINE L. HALL, COCHAIRPERSON, TOXIC CHEMICALS PROGRAM, ENVIRONMENTAL DEFENSE FUND

Ms. **Hall**. Thank you, Mr. Chairman, and members of the subcommittee.

My name is Khristine Hall, I am a staff attorney with the Environmental Defense Fund. I thank you for the opportunity to give my remarks today. I am with EDF's Toxic Chemicals Program which for the last 10 years or so has concentrated primarily on regulatory aspects of toxic chemicals.

During the course of that work we have come into contact with many people across the country and this contact has led us to be concerned with victim compensation, that is compensation for people who have injuries resulting from exposure to toxic chemicals.

Over the last several months I have had occasion to meet periodically with members of other environmental organizations, with citizen groups from across the country and other people who are also interested in the issue of victim compensation. I can unequivocably state that the environmental community strongly supports the need for the victim compensation legislation. We want to see the glaring loophole of Superfund plugged wherein damage by toxic chemicals for natural resources is compensated but people injured by toxic chemicals are not.

Our support for victim compensation legislation is premised on the fact that there are real victims and secondly that there are insurmountable legal barriers preventing them from being compensated.

Although I know you have had panels with people who are victims, I would also like to add a few more examples to the record. These are examples that have come to my attention in the last 6 months or so.

Out of 112 blood samples taken by the Center for Disease Control from people living in dioxin-contaminated areas of Missouri, all 112 samples showed biomedical evidence of liver dysfunction. These are very sick people.

The rate of heart defects in infants born to mothers in San Jose, CA, who consumed water contaminated with trichloroethane is significantly higher than the rate of such defect in the general population.

Third, many children living near a lead smelter in Dallas, TX, have severely elevated blood lead levels, well above levels that the Public Health Service defines as lead poisoning.

Fourth, scientists have found that children in Michigan with a high body burden of polybrominated biphenyls in their fatty tissue have decreased intellectual capacity as measured by school performance.

Lastly, a significant number of the rural population of Hardeman County, TN that drank chemically-contaminated drinking water have symptoms including liver damage, enlarged livers, or optic atrophy, early onset of blindness.

These are a few examples, it is not a complete list. A complete list is impossible to compile since we have only begun to research the issue of injuries produced by toxic chemicals and because many of the diseases produced by toxic chemicals have very long latency periods and we would not expect to see them for some years hence. This is enough of a sampling however to indicate that there are very real victims.

In addition, we know that there are many people who have been exposed to chemicals that in either epidemiology studies or laboratory animal studies have adverse effects ranging from decreased IQs to infertility, to cancer, and respiratory and other problems. We cannot assume that these exposures will not lead to some sort of problems in these human beings.

It will be many years before we know the full extent of injury from toxic chemicals. Those studies have just now begun and it will take some time to document them. Hasty studies done in the near term are not terribly useful for documenting the extent of the problem. I want to emphasize however, that we feel strongly that we know enough to know that there is a problem that needs to be addressed.

This is not a solution in search of a problem as those who created the problem so glibly assert, but a serious problem in search of a thoughtful answer.

Once the premise is accepted that there are victims one must ask if they are being compensated for their injuries? Compensation is important for two reasons, fairness and economic efficiency.

It is a basic tenet of our common law that it is fundamentally unfair that people who are injured are not compensated for their injury. In addition, it is economically inefficient if the costs are borne by others than those who reap the benefit from some social activity. It leads to a situation where the social costs outweigh the social benefits. In addition, when an entity pays the full costs of his activities it should lead to changes in his behavior.

The two most authoritative documents on the issue of victims compensation have concluded that there are very real legal barriers to compensation. These are of course the studies done by the Environmental Law Institute and the study done by the section 301(e) panel under the Superfund law.

They have documented a range of problems including antiquated statute of limitations, proof of causation requirements that fit traditional torts but not toxic torts; standards of liability; joinder defendants and just the general complexity of this type of litigation.

Several proposals have been put forward in reaction to the studies. I will not comment on them specifically but rather address some basic concepts in them.

First of all, we feel strongly that the scope of any legislation should include compensation for all injuries from environmental contamination, not just injuries from hazardous waste facilities.

As appealing as the hazardous waste issue has been over the last year there are also some very real problems with contamination from other sources as the example I have of the children in Dallas, TX indicates.

The Superfund legislation now on the books is not confined to hazardous waste facilities nor should any potential victim compensation legislation.

Second, the section 301(e) panel put forward a proposal for a two-tiered system, an administrative system of compensation and a judicial approach. The environmental community generally supports the concept of a two-tiered system but has some reservations about methods of implementing it.

The administrative step, the first tier, would provide limited recovery of out-of-pocket expenses and lost income up to $2000 a month.

We generally support that concept.

It must however, be accompanied by an easing of the burden of proof that is in traditional tort law. From our point of view, three requirements should be placed and claimants in that burden before the law would provide that the claimant has met his burden of proof.

One is that there is a reasonable likelihood of exposure of sufficient dose and duration to have caused or contributed to the disease.

Second, that there be some link scientifically between the disease and the exposure. Some have proposed this be based on hazardous substance documents. While these documents may be useful to the debate and to any judgment we do not feel they should be relied upon alone since they will undoubtedly be very, very controversial documents and probably will be outdated by the time they are even issued.

Additional evidence in the form of epidemiological studies, statistical studies, animal studies, tissue culture studies should also be admitted. There are problems now admitting this type of evidence.

Third, there should be proof of injury.

This is still, contrary to some assertions, a very large, expensive burden of proof. Proof of exposure alone is a very costly proposition as I know from my days with the Department of Justice when I was trying to put together hazardous waste cases. It entails very expensive, extensive monitoring to find out what the concentrations of pollutants are or were, often after the fact when the evidence has been changed substantially. It may also require very sophisticated computer modeling to forecast where some of that chemical matter has gone.

Proof of the link between exposure and the subtance is also very costly. It will entail very detailed studies, very detailed analysis of very complicated scientific studies. In addition, the medical expenses of proving injury, some of which take sophisticated diagnostic skills, is also very large. These are not common, every-day injuries.

Because of this and because of the fact that the funds that you can get from the administrative approach are very limited, we think that the first tier may not often be used. It is simply too expensive a proposition for too small a benefit.

Therefore, we endorse it but endorse it as a limited remedy and certainly not the exclusive remedy and feel that the second tier, the judicial remedy proposal still remains the most important aspect of victims' compensation.

The section 301(e) study recommended that the second tier be left in the hands of State courts. We feel strongly that a Federal cause of action ought to be created. However, to ensure that the resources of State courts are still used, we recommend that the policy followed in the Federal Employers Liability Act wherein a Federal cause of action may also be brought in State courts be followed.

In addition we feel strongly there should be no preemption of existing State remedies, either legislative or common law.

The Federal cause of action should squarely address the issue of proof of causation and the difficulties in it. The burden of going forward should be premised on the three elements that I outlined in regard to the first tier, that is the plaintiff's burden of proof should be counted as having been met when there is a showing of exposure that produces a likelihood of causing or contributing to the disease; the scientific link between the disease and the exposure; and the injury.

As with tier I this is still a very difficult burden of proof, very expensive. It does not release the plaintiff from having to prove by the traditional test—a preponderance of the evidence that he was injured. Our traditional standard in common law tort cases would still be there. What this does is allow the plaintiffs to introduce relevant evidence that is now often excluded from hearings, and avoid the requirements of traditional theories of tort. One can easily trace a bullet out of a gun into the victim's body. You cannot do that with toxic torts. You cannot trace the molecule out of the smokestack or out of the toxic dump into the victim's body, resulting in disease.

You have to be able to use in these cases some of the more non-traditional types of evidences as epidemiological studies and animal studies.

Liability should be as in Superfund, joint, strict, and several. I know especially the joint and several is controversial, but it is necessary to be sure that plaintiffs have workable cases and that they don't have to identify every last possible defendant before they sue. In addition we part company with many of the proposals that have been put forward that would limit liability to diseases that are caused by exposure that occurs after the date of enactment of any legislation.

We do this for two reasons, first of all we think this may be a fundamentally unworkable provision. How do you distinguish with diseases that have long latency periods whether they arise from exposure before or after enactment? I suggest that that is rather an impossible provision and would lead to protracted litigation.

Second, the sums that have been suggested for victim compensation, particularly in the administrative stage, the fund is rather small. The bulk of the problem right now and for the near future is going to be from exposure that occurred before the date of enactment of any legislation. Unless there is a significant increase in the amount of money in these funds you are going to create a problem by saying that the fund cannot be subrogated to the rights of plaintiffs if the state of exposure predates the legislation.

With that general overview I will again thank you for the opportunity. I would be happy to answer any questions.

[The statement of Ms. Hall follows:]

TESTIMONY OF KHRISTINE L. HALL

Mr. Chairman, members of the Subcommittee, my name is Khristine L. Hall. I am an attorney with the Toxic Chemicals Program of the Environmental Defense Fund. Thank you for inviting me to testify on the issue of compensating victims of exposure to toxic substances.

The Environmental Defense Fund (EDF) is a national not-for-profit environmental organization with a nationwide membership of approximately 47,000. The goal of EDF's Toxic Chemicals Program is to minimize or prevent exposure by people and the environment to toxic chemicals. Over the past decade, EDF has worked to insure proper implementation of a number of regulatory programs concerning toxic chemicals: the Food, Drug and Cosmetic Act, the Clean Water Act, the Clean Air Act, the hazardous waste provisions of the Resource Conservation and Recovery Act, and others. Most recently we have been actively involved in implementation of the Comprehensive Environmental Response, Compensation and Liability Act (Superfund). Our work on these programs, especially of Superfund, has aroused our concern about the ability of people who are injured by exposure from toxic substances to be compensated for those injuries.

Discussions of the important issue of providing a better mechanism that now exists to insure compensation to those people who have been injured by toxic chemicals has recently resumed after being curtailed by the passage of Superfund in 1980. EDF was among those organizations that worked for inclusion of a victim compensation provision in Superfund. While we understand the political necessity of dropping such provisions from the final bill to ensure passage, we remain convinced of the necessity of such a provision.

Over the past several months, I have been meeting periodically with representatives of environmental organizations, citizen groups, and other public interest organizations to discuss victim compensation. While I am not prepared today to lay out a detailed formal position on behalf of those organizations. I can state unequivocally that the environmental community strongly supports enactment of victim compensation legislation in this Congress. To allow the inconsistency of Superfund—which provides for compensation for natural resources damaged by toxic chemicals, but not people who are injured by toxic chemicals—to stand for much longer is not good public policy.

Our support for legislation is premised on (1) the fact that there are people who have been injured by toxic chemicals and (2) the current legal system presents a number of difficult and sometimes insurmountable barriers to recovering compensation for those injuries.

PEOPLE ARE BEING INJURED BY TOXIC CHEMICALS

Out of 112 blood samples taken by the Centers for Disease Control from people living in dioxin-contaminated neighborhoods in Missouri, all 112 samples showed biomedical evidence of liver dysfunction.

The rate of heart defects in infants born to mothers in San Jose, California, who consumed water contaminated with trichloroethane (TCA) is significantly higher than the rate in the general population.

Many children living near a lead smelter in Deere Park, Texas have severely elevated blood lead levels—well above levels the Public Health Service defines as lead poisoning.

Scientists have found that children in Michigan with a high body burden of polybrominated biphenyls (PBBs) in their fatty issue have decreased intellectual capacity, as measured by school performance.

A significant number of the rural population of Hardman County, Tennessee that drank chemically-contaminated drinking water have symptoms including liver damage, enlarged liver, or optic atrophy.

These are but a few examples of where toxic chemicals have caused injury to human beings. They are examples that have come to my attention in recent months. This is not a complete list—nor could it be, since we have only recently begun to study and document the effect of toxic chemicals on people and since some diseases caused by toxic chemicals have long latency periods.

It is enough of a sampling, however, to indicate that there are, in fact, real victims of exposure to toxic chemicals. In addition, we know that some people are exposed, through the air they breathe, the water they drink or the food they eat, to a broad range of substances that have been shown in animal tests and epidemiology studies to cause cancer, birth defects, infertility, lowered IQs, respiratory problems, and other serious illnesses. Dose levels for some of these exposures are relatively high. We cannot assume that there have not been and will not be human injuries from such exposure.

It may be many years before we can reasonably expect to have scientifically documented the full magnitude of injuries caused by toxic chemicals. Hasty studies undertaken with an eye toward pending legislation will not produce such documentation. I want to emphasize, however, that the environmental community believes we have enough evidence in hand to indicate that people are being injured by toxic chemicals. This is not a solution in search of a problem as those created the problem so glibly assert, but a serious problem in search of a problem as those who created the problem so glibly assert, but a serious problem in search of a thoughtful solution.

LEGAL BARRIERS PREVENT RECOVERY OF COMPENSATION

Once the premise is accepted that people have been or will be injured from the toxic chemicals now found in our environment, two questions should be asked: (1) What can society do to minimize or prevent exposure to these toxicants and (2) Are the victims being compensated for their injuries?

The answer to the first question lies largely in more effective regulation of the way toxic chemicals find their way into the environment. Considerable thought has been addressed to this issue over the past decade. Although improvements are in order for many of our efforts (e.g., toxic water pollution amendments pending before this Committee, the pending reauthorization of the Resource Conservation and Recovery Act, and new amendments to the Clean Air Act and the Toxic Substances Control Act), action has at least been initiated.

Unfortunately, the second question—concerning compensation—has not yet been adequately addressed. The starting point for any inquiry is the basic question of why we should be concerned about making sure that victims of toxic chemicals are compensated. It is a basic goal of our tort system that each victim be compensated by the entity inflicting harm on him or her. This goal rests primarily on the principles of fairness and economic efficiency. It is fundamentally unfair if a victim is not compensated for injuries received as a result of activities carried on by and benefiting another. Furthermore, injuries resulting from an activity are part of the social cost of that activity. If those costs are borne by someone other than the entity reaping the benefit of the activity, and more important, making the decision as to the amount of the activity, then an economic inefficiency occurs. That inefficiency could result in a level of activity at which the social benefit from the activity does not outweigh its social costs. A variant of this theory is that if a polluting entity is made to pay the full cost of the damage it causes, such economic costs may change the manner in which business is conducted.

The issue of whether a victim has been adequately compensated is thus important—both to the victim and to society. Delay in solving problems in compensation cannot, therefore, wait the many years it will take to thoroughly document the extent of the problem.

The two most authoritative documents to address this important issue of whether current remedies are adequate—the Environmental Law Institute's report entitled "Statutory Reform of 'Toxic Torts' Relieving Legal. Scientific, and Economic Durdens on the Chemical Victim" and the Superfund Section 301(e) report. Injuries and Damages from Hazardous Wastes—Analysis and Improvement of Legal Remedies,"—both conclude that our present system or tort law is inadequate to insure compensation to victims of toxic chemicals. To quote from the Section 301(e) Study Group:

"The Study Group concludes (1) that there are causes of action on which to base a claim for damages for such injuries or damage; (2) that there are many substantive and procedural barriers to recovery; and (3) that existing courses of action and existing barriers to recovery will allow some recoveries in a number of highly selected cases. Available, remedies are inadequate in view of the substantial number of claims that may arise, and the factual and legal complexities that will be involved in their litigation."

It is fair to say that our present tort system is behind times in dealing with modern society and the injuries produced in modern society. Od rules which worked well when industrialization was just beginning have been slow to adapt to the complexities of our technological age. Problems such as antiquated statutes of limitations, proof of causation, standard of liability, and joinder of defendants, as well as the general complexity of toxic tort litigation, have blocked recovery or made it so difficult as to discourage seeking it. Although progress has been made in some states on some issues, major problems remain, as a general matter, that deserve attention and solutions.

WHAT ARE THE SOLUTIONS

Descriptions of the problem is the easy part of this controversy. Finding a meaningful, fair solution is not so easy. Several specific legislative proposals, incorporating different approaches and concepts have already been put forward in both the House and Senate side. Rather than commenting specifically on these bills, I would like to make a few suggestions and to address concepts incorporated in the bills generally and in the ELI and 301(e) studies.

A. Scope of coverage

Victims compensation legislation should cover injury from all environmental exposures—not just from hazardous waste. Although the hazardous waste problem is dramatic and exposure to it significant, exposure to toxic chemicals through other environmental media is also important, as illustrated by the blood lead levels in children close to the lead smelter in Texas, cited above. It makes little sense for a person whose injury arises from exposure to lead from a smelter to have to follow different, more stringent rules for recovery than the person whose injury arose from a hazardous waste site. We note that Superfund coverage is not confined to hazardous waste sites and believe that victims compensation legislation likewise should be broad enough to cover all environmental exposure.

B. Two-tiered system

The section 301(e) panel proposed a two-tiered recovery system, with the first tier being an administrative mechanism and the second tier a judicial approach. This approach has been adopted in some of the bills that have been introduced. We endorse the general concept of the two-tiered system, with reservations about details of its implementation.

1. Tier I—Administrative mechanism

As laid out in the Section 301(e) study, Tier I would establish an administrative mechanism for taking evidence from potential victims and making awards from a Fund, financed primarily by a tax on industry. We would propose that EPA administer the compensation system, but that the board or panel making findings also have representation from Health and Human Services.

We propose that recovery from Tier I be limited to lost income up to $2000 a month and reasonable out-of-pocket expenses (i.e., medical expenses, cost of relocation). This differs from the recommendation of the Section 301(e) panel only in allowing other reasonable out-of-pocket expenses (such as the expense of a forced relocation) to be recompensed in addition to medical expenses.

To make this process work, the burden of proof now generally required of plaintiffs would have to be eased. We recommend that legislation state that a plantiff has met his burden of proof, subject to rebuttal, by showing the following: (a) reasonable likehihood of exposure of sufficient duration and dose to produce injury; (b) scientific link between injury and exposure to chemical; and (c) injury. This is not unlike the rebutable presumption recommendation of the Section 301(e) panel for Tier I.

In establishing the second element, the scientific link between injury and exposure, plaintiffs may rely on Toxic Substances Documents, if available. As recommended by the Section 301(e) panel, these documents would provide evidentiary data showing that specific hazardous substances or wastes cause specific diseases or illnesses. Because preparation of these documents is likely to be protracted and the subject of intense debate, they will undoubtedly be produced only late in the process. In addition, because of the rapid rate that scientific discoveries are now being made about toxic chemicals and their effects, these documents are also likely to be outdated when finally produced or shortly thereafter. We therefore recommend that plaintiffs be allowed to rely on other scientific evidence available tending to prove cause-and-effect or correclation between exposure to a chemical and disease. This would include epidemiological studies, animal studies, tissue culture studies, and micro-organism studies.

This approach leaves in place a very considerable burden of proof on plaintiffs. Proof of a reasonable likelihood of exposure of sufficient dose and duration to cause injury is very difficult. In many instances, it will require extensive and expensive sampling and sophisticated modeling and analysis to prove the fact of exposure and the concentrations of the pollutants—often after-the-fact when circumstances have changed or evidence altered.

Establishing the link between exposure to the chemical and disease is also an expensive proposition, given that we doubt the widespread avialability or usefulness as the ultimate resource of the Toxic Substances Document. Expensive research and analysis will also be required for this step.

Finally, establishing the medical fact of injury with certainty of diagnosis may also be expensive. As everyone is aware, any visit to the doctor is expensive. Medical attention to unusual conditions or diseases caused by toxic chemcials requires special expertise, which is even more expensive.

In short, although this proposed approach eliminates some barriers in the current system and allows the introduction of more evidence than often allowed by courts now, it still presents a very high hurdle to recovery. Hiring the necessary technical people, analysts, and expert witnesses is still very expensive—in many cases more expensive than the small amount of money proposals would allow to be recovered under Tier I.

This is an important point. What it means is the Tier I may not be used with success very often. It is for this reason that we view Tier I as a very limited remedy of limited utility. In fact, the Tier I does not even begin to address the bigger, more complicated issues associated with injury from exposure to toxic chemicals, such as pain and suffering, compensation for loss of intelligence, and compensation for loss of fertility. We support establishing Tier I as a way of dealing with very small injuries and as a means of gaining experience in this area with administrative compensation mechanisms. Given its limited utility and scope, however, it is clearly not a substitute for Tier II. We therefore vigorously oppose any efforts to limit compensation to Tier I or to force a binding choice between Tier I and II.

2. Tier II

The second tier is resort to the judicial system. The Section 301(e) panel essentially left Tier II up to the state courts, while making recommendations on ways in which state legal requirements should be changed to enhance recovery possibilities. This approach ducks the issue and is unacceptable, given the continued importance that judicial remedies will play in any viable compensation system. While some states have already adopted and other states will adopt, either through court decision or legislation, some of the reforms recommended by the Study Group, reliance on the state court system will result in a patchwork quilt of remedies, when viewed from the national perspective. Just as it has been necessary to establish minimum federal standards and guidelines as a floor for pollution control, so too is it necessary to establish minimum federal rules governing compensation systems.

It is for this reason that we commended establishment of a Federal cause of action, incorporation reforms recommended by the Section 301(e) Study Group. To insure that the resources of our state court system continue to be used, we suggest that this federal cause of action may be brought either in the state courts or in the federal courts. There is precedent for this approach in the Federal Employers Liability Act (FELA—45 U.S.C. § 56). Plaintiff should also be allowed to use state courts and causes of action.

There should be no limits on the amount or type of recovery. In addition, legislation should make clear that the plaintiff's burden of going forward has been met by showing the same three elements of the rebuttable presumption in Tier I: (a) reasonable likelihood of exposure of sufficient dose and duration to cause injury: (b) link between exposure and injury and (c) injury. This approach does not relieve plaintiff from proving his case by a "preponderance of evidence" as is now the usual standard in civil liability cases. Rather, it allows plaintiff to introduce more types of evidence (such as epidemiology studies, animal studies, etc.) without challenges to relevency and relieves plaintiff of the burden of tracing a molecule of the pollutant from the source into the body to produce the injury. This burden, which traditional tort law may require, is and will remain impossible to meet. This approach allows plaintiff to take evidence relevent to modern injuries to the jury. Defendant, of course, still may introduce countervailing evidence.

As discussed above, this is still a very considerable, expensive burden of proof. Defining the burden of going forward in this manner does not mean that plaintiff will always prevail; it does mean giving plaintiff the opportunity to present his case in the only way relevant to modern injuries to the jury.

C. Liability issues

In accordance with the recommendations of the Section 301(e) Study Group, liability for incident that causes or contributes to the injury complained of should be strict, joint and several. This standard should apply both to direct judicial actions by injured plaintiffs and to claims by the Fund subrogated to plaintiffs' claim. Strict liability is necessary so that plaintiff does not have to prove fault—an impossible burden of proof in most of these cases.

Joint and several liability is necessary because of the difficulty of locating all defendants who may have caused or contributed to the injury. Delays in the onset of disease as well as, for instance, the fact that many companies may have used the dump from which a toxic substance leaked mean that, in the words of the Section 301(e) Study Group:

"It is impossible here, as in other instances of environmental pollution to attribute and allocate responsibilities and liabilities with specificity. Substantive and procedural rules that require such specific allocation will defeat the plaintiff's claim."

For this reason, the Section 301(e) Study Group recommended the use of the common law concept of joint and several liability. To soften the potentially harsh effects of joint and several liability, the common law excepted those whose contribution to the injury was de minimus. This should be incorporated, as well as a specific provision that liable parties may seek contribution from other parties.

The Fund should be subrogated to all plaintiff's claims, whether based on present or past action. The Section 301(e) Study Group had recommended subrogation only for claims arising out of exposure after the date of enactment. This restriction is unworkable—for long latency diseases there may be no way of determining whether exposure occurred before or after the date enactment. In addition, for some time to come, most claims will arise from exposure occurring before the date of exposure, thus placing a severe drain on the Fund. Since the allocations to the Fund provided for in various bills are very modest, we doubt that this is good policy.

CONCLUSION

I want to thank you for the time you are taking to explore this important question. The issues are complicated and deserve in depth consideration and thoughtful discussion. They are resolvable, however. We urge that resolution occur in this Congress. I would be happy to answer questions.

Mr. LEVITAS. Thank you very much, Ms. Hall.

Perhaps you can tell me just so we would have a matter of record, have you handled any litigation personally involving hazardous waste sites or toxic torts?

Ms. HALL. I have not handled toxic tort litigation. When I was at the Department of Justice I helped put together the cases against *Hooker Chemical Co.* concerning *Love Canal, Hyde Park,* and a couple other hazardous waste cases. So I know the difficulty just from that perspective of trying to put together a case that is going to argue that there has in fact been some effect from the hazardous waste.

Mr. LEVITAS. In your statement you mention that you believe victim compenation legislation should cover injury from all environmental exposures; however we have received testimony and read other materials from experts who believe there is insufficient scientific data to conclude that injury results from exposure to hazardous wastes. How would you reconcile these two widely divergent positions?

Ms. HALL. As I tried to outline in my statement, I think it will be quite some time before we do know the full magnitude of injuries from toxic chemicals generally. That process of studying the effect of toxic chemicals on human beings has just begun. In fact the mechanism that the Public Health Service is now using for documenting exposure and resultant injuries is something that began as a result of an EDF lawsuit about 3 months ago.

It will be many years before we have conclusive results from that study.

At the same time I think one can point to enough instances across the country—I have tried to outline a few of them in my testimony—where there is scientifically provable, clinically provable, instances of injury from hazardous chemicals.

As I stated, this is not a full catalog—it is probably impossible to compile a full catalog—but it is indicative of the fact that there is a problem. We also know that many people are now being exposed and have been exposed to toxic chemicals that we know from epidemiological studies and from animal studies lead to a range of results of injuries, and those two combined leads us to believe that there is a large problem that demands a solution.

Mr. LEVITAS. You made an interesting comment about the question of admissibility of evidence as one of the major problems. I used to teach evidence in law school and have been very interested in the artificial restrictions on denying triers of fact full information as a result of these rules of evidence.

In this particular situation is there a special reason why there should be a change in the rules of admissibility as opposed to other types of tort litigation?

Ms. HALL. Well, the type of evidence we are talking about, statistical evidence primarily, arises preeminently in this field of toxic torts. I am not aware of other areas where it might also arise. Epidemiology studies are, however, relevant to toxic torts whether they occur in the workplace or from environmental exposure. There is also a long line of cases on relevancy and admissibility on epidemiology studies with respect to HEW's Swine Flu program several years ago.

So there are related areas where it is also an issue.

Many courts have been very reluctant to admit this type of evidence or if they admit it to give very little weight.

Mr. LEVITAS. Can't you get that evidence in through an expert witness using it as a basis for formation of his opinion?

Ms. HALL. From the cases I am aware of, specifically on epidemiology studies, in some cases that is possible. In other cases courts have concluded that since an epidemiology study does not address specifically the issue of whether a particular plaintiff contracted the disease but goes to a general population, it is not relevant. You can also get it in in some instances, in some cases, as being relevant circumstantial evidence.

Other courts have excluded it because they feel it is confusing and too technical for a jury to understand.

There have been a range of reasons. The courts are beginning to open their minds to this but it has been a problem. I think you have even more of a problem when you move from epidemiology studies which use real human beings to animal studies and you have an extra leap to translate the animal data to human beings.

Mr. LEVITAS. I have just a couple other questions, but one of them is on the subject that you raised about injuries occurring or manifesting themselves before or after enactment.

A more serious ongoing question would be on the question of statute of limitations. We heard yesterday that there is pretty much consensus that there should be a 3-year statute of limitations based on the discovery of the injury.

Ms. HALL. Yes.

Mr. LEVITAS. Now, I was surprised to hear everybody seemed to agree on that when there seems to be a whole can of worms there since most of the types of illnesses or injuries that are connected with toxic chemicals are the same type of illnesses or diseases that people might receive from other sources, liver problems, kidney dysfunction, skin rashes, respiratory problems, all of these things you can get without ever being exposed or certainly not as a causal factor being environmental reasons, how would one know when you got—given the long latency period as well—how would one know or be in a position to make a judgment that when they develop a kidney dysfunction or a respiratory illness that that is a result of exposure *x* number of years ago to toxic chemicals?

Ms. HALL. First of all addressing the statute of limitations problem. I didn't include it in my testimony because I, too, perceive there is general consensus that it should be changed.

I don't think the statute of limitations issue is the way of getting to the problem you have just raised.

One of the ways of addressing that issue is by reliance on epidemiology. for instance, the people at Hardeman County, Tennessee and the mothers in San Jose, CA—by surveying through scientific methods a community one can determine whether in fact the incidence of a disease or injury is elevated above naturally occurring levels of disease.

If there is a statistically significant elevation that suggests that the cause is the environmental factor and not just some natural background.

Mr. **Levitas**. That deals with the question of proof, once someone made the judgement I got sick because of the environmental exposure.

If I became ill next week with a somewhat common ailment, how would I be able to make a judgment that that was caused as a result of my living near a hazardous wastesite when I was in the Air Force in Maine back in the 1950's?

Ms. **Hall**. Common ailments are the biggest problem in this, skin rashes, respiratory diseases, minor infections, they are the biggest problem because those are the things that occur with more frequency in the background.

Other diseases such as heart defects, liver dysfunction, certain types of cancer, are not so common and one can through these statistical methods distinguish populations.

It is somewhat ironic that the section 301(e) panel thought that first and foremost—from what I read of their deliberations—they were providing remedies for the small problems when they are probably scientifically the most difficult to distinguish and probably be the ones that will take many years to be able to develop a basis for.

My understanding is that—and being an attorney I am hesitant to comment on it—is that there is a growing body of knowledge on immunology where you can now do tests on immunological deficiencies to determine whether or not because of deficiencies in your immune system that you might not be more subject to infections, respiratory problems, that sort of thing, and that can be done biomedically and scientifically to determine what shape your immune system is in.

Mr. **Levitas**. Well, I won't belabor this point because we want to address a broad range of issues, but I can see now if I were a defense attorney representing a defendant in a toxic tort situation under a 3-year statute of limitations of the sort we are talking about, that in the course of discovery I would try to find out what symptoms or illnesses this individual manifested over the last 5 or 6 years and if I could spot one 5 years ago or 3 years and 1 day ago, that could also be related to that exposure to the same chemical, I would suggest that the injury had manifested itself 3 years, 1 day ago and even though you are suing about another injury now, also possibly related, that you have been caught by the statute.

As I was listening to that testimony yesterday that seemed to be a real problem to me.

Let me go on to the final point. I notice as I was listening to the lawyers testimony yesterday I began to feel—maybe I am in the wrong line of business now.

Ms. **Hall**. I am a lawyer, too.

Mr. **Levitas**. We have this whole deal of contingent fees, if you find the right case you have a pretty good horse to ride and I know, I know Mr. Molinari and Dr. Rowland and I were talking about it, we are creating a situation maybe in which lawyers are going to come out pretty well on this thing and how much of the resources we devote will actually go to the victims and how much will go to the attorneys is something we have to look at as we approach a Tier I system or a Tier II system.

But you recommend not only a special legislation matter and Federal preemption——

Ms. **Hall**. No, not Federal preemption. I want a Federal cause of action.

Mr. **Levitas**. You want a Federal cause of action in Federal courts, all right.

Now, the witnesses yesterday all seemed to believe that the existing common law tort system was an adequate means of addressing the compensation issue in the judicial context; once you got above a Tier I situation.

Also, I would have great concern—just as a matter of policy—of creating any new cause of action in a Federal court that could be handled appropriately in a State court that was not inherently a Federal or interstate commerce type of matter for a lot of reasons which I am sure you have heard as other lawyers have heard, all the policy reasons.

Why do you think you need a Federal cause of action. What is wrong with the tort system in the State courts? They seem to be at least as competent as Federal courts in many instances.

Ms. **Hall**. The basic reason for wanting a Federal cause of action—and I also added that we would have no problem with seeing a Federal cause of action that could be brought in State courts as the Federal Employers Liability Act now provides—is to establish some baseline rules which you cannot do for State courts, to address some of the legal barriers that have been documented in various studies such as statute of limitations. A number of States still have very antiquated statutes of limitations and to go State by State to try to change that could take a number of years.

A number of States still do not recognize strict liability as the appropriate standard of liability in these cases. Having to go under a theory of negligence places a very, very high burden on plaintiffs to try to prove their case. They have to prove some sort of fault. In the big cases, for instance the ongoing litigation on agent orange I think it is going to be fairly easy to show some sort of negligence. But in the run of the mill cases a showing of fault is very difficult.

Some States recognize joint and several liability, other States, for instance, Missouri where I just filed a brief under Superfund legislation, do not. Missouri is out of step with most of the rest of the States, but there are still several States that do not recognize what I regard as a minimum legal requirement for bringing these cases.

So our main concern is making sure that there are some minimum changes in rules that now exist in some places. Just as it has been necessary to put a minimum Federal floor on pollution control standards we feel there should be a minimum Federal floor on some of these rules that govern compensation.

Mr. **Levitas**. Thank you, Ms. Hall.

Mr. Molinari.

Mr. **Molinari**. Thank you, Mr. Chairman.

Ms. Hall, I, too, have problems with the question of statutes of limitation.

I think in this room we probably have a lot of people who have a great deal of familiarity with the kinds of symptoms that people exhibit when they have been exposed to chemicals such as we are discussing.

But I guess most of us relate to personal experiences to understand the real world out there. Back in my community there is a dump where very large quantities of different chemicals were deposited systematically over a period of, a year-and-a-half or 2 years.

The people in the neighborhood were complaining to their doctors of a variety of symptoms and really didn't know nor did the doctors, about the source of the problems. In fact there were different diagnoses until 2 years later, during the course of a Federal trial in Pennsylvania, that there was this dumping.

As soon as that became public, the people started running back to their doctors and the doctors had a better handle on the possible source of their problems. It may have been that they might still have been able to start a lawsuit under the statute of limitations in New York, but I am not sure.

Why do we suggest having the statute of limitations toll from the time when the people have evidence of injury rather than discovery of the source?

Ms. HALL. It can be formulated either way and legislative——

Mr. MOLINARI. I understand that but I thought it was your recommendation or the recommendation——

Ms. HALL. No, I have made no explicit recommendation in my testimony on exact wording. I believe it should be formulated from the—it should run 3 years from the date of exposure or from the date of knowledge of what caused it.

Mr. MOLINARI. Constructive or otherwise?

Ms. HALL. That is right.

Mr. MOLINARI. On that score I would like to say that I would like to see the attorneys making more money—I don't mean a percentage more—but more total earnings in toxic tort litigation because I think the net result of that would be that more people out there who are being injured would be getting compensation; I think too many of them are not under the present system. That is why we are talking here today.

Ms. HALL. I agree with that obviously.

Mr. MOLINARI. I have just a couple other questions I want to ask you.

I was surprised at the testimony of Mr. Baron yesterday—were you in the room?

Ms. HALL. I was not unfortunately, I was busy writing this testimony for today.

Mr. MOLINARI. He kind of shocked us, I guess, by suggesting that the state of the art today is such that lawyers, particularly like those in his firm who specialize are able to develop a case. There has been great disagreement as to what resources it would take to properly develop such a case, but he believes that existing law is adequate, despite the fact that I think he mentioned there is only one other firm that he knew of that is doing this on a regular basis.

Mr. Baron was also a member of the 301(e) study group, so he has some rather impressive credentials. I must say that it made me stop and consider for a moment: if this is a man that has had actual experience—and he probably has had more than just about anybody else that we know of—and after that experience he has suggested leaving the system alone, perhaps it can be handled as it is, in that people that are being harmed have adequate remedies at law today.

Would you comment on that?

Ms. Hall. I think you have put your finger on the difference between Mr. Baron and other points of view of lawyers. He has at his disposal extraordinary resources to put these cases together and he is used to dealing with them. But he is one of a kind or one of a very small handful.

We deal in our work on Superfund with communities across the country, small communities across the country who don't have access to the Mr. Barons and who have access only to the local lawyer down the street. I am not suggesting that a lot of the cases will be brought by the local lawyer down the street but even in cases, for instance, of working with a fairly good-sized firm the lawyers are struggling with these cases. The ordinary case in a lot of States is going to run into legal barriers that have been well-documented by studies including the one Mr. Baron participated in.

I did some consulting with a firm out in the San Francisco area, a large plaintiffs firm that didn't know where to start and is really having some problems with putting together a classic toxic tort case. And it seems to me to address the full breadth of the problem and not the really big and sexy cases, you are going to have to have some easing of some of the barriers that are now contained in the law.

Mr. Molinari. My own impression is that the average lawyer who has a prospective client walk in and relate that they believe they have a physical problem due to a dump that is two or three or four blocks away, may talk to them for a brief period of time, to look at the case as a loser and suggest they go to the bar association. I don't know that the average lawyer is, in his own personal knowledge, aware of firms specializing in toxic tort litigation.

Ms. Hall. It is a big problem. I get several phone calls a month from lawyers wanting to know where they go and what they do.

Mr. Molinari. What is your answer to them?

Ms. Hall. I fill them in as best as I can and point them in the direction of people that I know who have handled some cases and generally wring my hands over it.

Mr. Molinari. Do you have a catalog of attorneys that handle these matters?

Ms. Hall. I don't make formal referrals, no.

Mr. Molinari. You don't?

Ms. Hall. We do not. That is a service that is needed, however.

Mr. Molinari. Is it something you are considering or is it against policy of EDF to do so?

Ms. Hall. It is controversial within the organization now.

Mr. Molinari. Sorry?

Ms. Hall. It is a controversial issue within my organization now.

Mr. Molinari. I see. I just have one last question, Ms. Hall.

You make reference to the Missouri problem and the blood samples showing liver dysfunction. Were you able to determine from your own inquiry whether that liver dysfunction was reversible?

Ms. Hall. You need to ask the scientists that, not me. But my understanding is that it shows that the liver has fundamentally been damaged and that is all I know.

Mr. **Molinari**. I have no further questions, Mr. Chairman, thank you.

Mr. **Levitas**. Thank you very much.

Dr. Rowland.

Mr. **Rowland**. Thank you, Mr. Chairman.

Ms. Hall, what do you think is the most important thing that Congress needs to do now with reference to hazardous waste dumps and toxic chemicals that people are exposed to and things of this nature. What is the most important thing Congress needs to do in your opinion?

Ms. **Hall**. Well, I really have a two-pronged answer to that, sorry. One is to tighten up regulations. You have a variety of means to do that: the Clean Water Act pending before this committee, specifically the provision on toxic water pollutants, the RCRA reauthorization that will hit the floor sometime later this week, amendments to a variety of issues to make sure that we tighten our control and regulation of toxic chemicals to prevent future injuries.

Second, providing a better system to ensure that people who are injured are compensated.

Mr. **Rowland**. I have had concern—and I expressed this yesterday—that allowing suits to be filed in this area when we are not even sure exactly what detrimental effects of these chemicals may be, the fact that there is a new area opening up in the law and only two firms are involved, that is, in toxic chemicals, that we may be opening an area that will be good for lawyers who are involved in tort situations and might really cause ourselves a lot of difficulty and I see you are saying there should be no limits on the amount or type of recovery.

Ms. **Hall**. In judicial proceedings.

Mr. **Rowland**. Maybe I misinterpreted that since I am not an attorney, you are not talking about the amount that a person who may be injured could recover?

Ms. **Hall**. No, in Tier II, which is the court cases area, there should be no limits on recovery.

I feel fundamentally that putting some sort of statutory limitation on recovery will result in the problems we have, for instance, in the nuclear area where essentially government is subsidizing, underwriting, the costs of doing business, and I don't feel that that mistake should carry over to this area.

Mr. **Rowland**. Then am I to interpret this statement as meaning then that the amount of money that a person could recover is unlimited?

Ms. **Hall**. Commensurate with his injury and whatever he is able to prove.

Mr. **Rowland**. You don't have any concern that we might be opening up an area that could cost a lot of money to industry for example, and the costs being passed along and we would be providing a better livelihood for attorneys in an expanding area?

Ms. **Hall**. I have two comments.

One is I feel right now that industry is not paying the full costs of its activities because they are causing a lot of injuries that are not being compensated. As I discussed briefly in my testimony I think that is economically inefficient and that they should be paying the full cost of their activities.

Second, yes, as a lawyer, not as a tort lawyer but as a lawyer, I am sometimes alarmed by the amounts of recoveries that I see but the problem is not limited to toxic tort cases. The problem is a general problem now with tort cases. I am somewhat relieved to read that on review a lot of these very, very high awards are being overturned. I don't think there should be an artificial ceiling placed on recovery because that artificial ceiling as we have seen in other areas is bad policy.

I think the problem has to be addressed broadly in the tort area and not specifically with regard to environmental problems.

Mr. **Rowland**. You think then that having industry to pay the full amount or a great deal is a way to get industry to clean up their act so to speak?

Ms. **Hall**. I think it is one of the ways of getting industry to clean up their act, that is correct. I think it is one of the economic incentives that can complement Federal and state regulatory programs.

Mr. **Rowland**. You are saying that to do this would do two things, compensate the victim and get industry to clean up their act?

Ms. **Hall**. I am saying that it may have the second effect. I think the primary purpose is compensation of the victim but there are studies that seem to indicate that it may also have a salutory effect on the social behavior of the entities that caused the toxic chemicals to enter the environment in the first place.

Mr. **Rowland**. You feel that the industry costs for this should be passed along as a cost of doing business to the citizenry as a whole?

Ms. **Hall**. That is correct.

Mr. **Rowland**. Thank you, Mr. Chairman.

Mr. **Levitas**. Thank you.

Mr. Molinari.

Mr. **Molinari**. I have a further question that I would like to ask if you would yield, Mr. Chairman.

We heard from some victims last week, specifically an individual who allegedly sustained a very severe injury from exposure to toxic chemicals. The amount of expenses that he has incurred so far run over $100 thousand—I don't recall the exact amount.

The impression I got was that he had no money, so he is probably receiving public assistance in order to survive. I am not sure of that; that is speculation on my part.

Assuming that is the case, when we look at a so-called victim of that circumstance and we try to estimate who should pick up the tab, we are faced with these considerations, among others: Should it be the taxpayers? which would naturally be the case if he was on public assistance; or should it be borne by the industry which has created the chemicals that caused the problem, or any other intermediaries who contributed to causation?

Just an observation, really, rather than a question. I think it answers itself; but it is an interesting philosophical question and one which Dr. Rowland raised very well.

Thank you.

Mr. **Levitas**. Do you want to comment on that?

Ms. **Hall**. I agree that the costs should be borne by the people who caused the problem and not by the public at large.

Mr. **Levitas**. Yesterday one of the witnesses on that excellent panel of attorneys indicated that he left a lucrative practice of law in the field of aviation and structural scaffolding to move into toxic tort areas as an almost social conscience decision. I won't express any judgment on that. But based on what you know at the bar and what is occurring, take the case that Mr. Molinari just talked about, a person has a couple hundred thousand dollars' worth of medical expenses or other expenses. That translates into some rather substantial recovery plus pain and suffering and loss of future income and things like that.

Ms. **Hall**. Yes.

Mr. **Levitas**. Operating on contingent fees if nothing else—isn't this a pretty lucrative area of the law?

Ms. **Hall**. Being a public interest lawyer, I cannot say anything about that.

Mr. **Levitas**. I thought you would be more objective, that is why I asked you the question.

Ms. **Hall**. I think for the people who rise to the top of it and there will obviously be people who rise to the top of this area of specialization, it will be lucrative. For the ordinary lawyers who represent the ordinary citizens in the ordinary cases I don't think it will be necessarily all that lucrative, no.

Mr. **Levitas**. Well, I thank you for your testimony. I was particularly interested in your observations on the evidentiary question which I think places a new light or avenue that we certainly ought to be exploring and because of your expertise in this area you have helped us greatly.

Thank you so much.

Ms. **Hall**. Thank you.

Mr. **Levitas**. The next witnesses I will ask to come together as a panel, are Mr. Leslie Cheek III, vice president-Federal affairs for Crum & Forster Insurance Cos.; and Ms. Sheila Birnbaum, of counsel, Skadden, Arps, Slate, Meagher & Flom.

May I ask that you raise your right hand.

[Witnesses sworn.]

Mr. **Levitas**. Thank you very much.

TESTIMONY OF LESLIE CHEEK III, VICE PRESIDENT-FEDERAL AFFAIRS, CRUM & FORSTER INSURANCE COS.; AND SHEILA L. BIRNBAUM, OF COUNSEL, SKADDEN, ARPS, SLATE, MEAGHER & FLOM, REPRESENTING AMERICAN INSURANCE ASSOCIATION SOCIATION

Mr. **Levitas**. We might start with you, Mr. Cheek, and we will hear from Ms. Birnbaum and then the subcommittee can put its questions to both of you.

Mr. **Cheek**. Thank you, Mr. Chairman.

If I may, I would like to ask that my prepared statement be put in the record.

Mr. **Levitas**. Yes. Without objection the prepared statements of all of the witnesses today will be made part of the record and my request again, if you would try to limit your opening remarks to approximately 10 minutes so we can get into the questions.

As I said earlier, the role of the insurance field in this area as well as many other areas is very significant in terms of how we determine what social policy to pursue through legislation. So I am most interested in your comments.

Mr. **Cheek**. Thank you, Mr. Chairman.

The Crum & Forster Insurance Cos. are the Nation's 14th ranked group of property-casualty, insurers with 1982 premiums of approximately $1.7 billion. Our companies are among the largest writers of the principal nonfault system in the United States, workers' compensation, and also of coverage for businesses subject to tort and environmental cleanup liability.

We also write a substantial amount of insurance for vehicle owners in States that have hybrid fault and nonfault automobile insurance compensation systems. We think that the lessons we have learned from working with these different kinds of systems might be useful to the subcommittee in evaluating which way to go on this victim compensation issue.

Our principal interests in any compensation or liability system are the predictability and affordability of its costs. Without a reasonable certainty as to how many persons will make claims against the system and how much on the average each claim will cost, we cannot determine the appropriate premium to charge our customers. And if our customers can't afford what we are trying to sell them, we are out of business.

The Congress, too, we think, should be sensitive to both the predictability and the affordability of the costs of any system it creates to deal with what so far, in our view, is a very poorly defined and perhaps even nonexistent problem.

The Congress' last venture into a specialized compensation mechanism, the Black Lung Benefits Act, as you well know, produced a system that was initially estimated to cost $40 million a year and is now running along at a cost of about $2 billion a year.

The premise underlying the study group's proposals for new mechanisms to deal with hazardous substance-related claims is that there are or will be in the future large numbers of persons with demonstrable injuries or diseases resulting from exposure to hazardous substances who are not now being or will not in the future be compensated under current law.

We have to tell you that we are unable to find now in our loss and expense experience or elsewhere, any evidence that current legal remedies are failing to provide redress to persons eligible to make claims for injuries resulting from such exposures.

We will concede that in a few States what we regard as anachronistic statutes of limitations still pose procedural barriers to claims in long latency disease cases in the occupational and nonoccupational context. We are in the process of trying to overcome those barriers. We think that the statute of limitations should run from the time the disease manifests itself rather than from the date on which a given person was exposed to a hazardous substance.

In addition, we cannot and we don't know of anybody else who can determine how many persons who have a demonstrable injury resulting from exposure to hazardous substances are not availing themselves of remedies currently available. That is the great unknown. The anecdotal evidence we are able to come across, for example, the experience under California's Hazardous Substance Compensation Act, suggests that the number of people in this category is quite small. It leads us to suggest that more evidence that there are people out there with injuries who are not, one, pursuing the legal remedies that are available; and two, not recovering under those legal mechanisms, until there is firmer evidence on that point we would suggest that Congress stay its hand on any kind of compensation system.

We also think, looking further ahead, that Congress ought to be concerned about the insurability of those who might be required to fund any system the Congress ultimately creates. As no tax mechanism can, insurers distribute costs among their policyholders primarily on the basis of their loss history, thereby assuring that those who cause harm ultimately pay for it and providing a marketplace system for loss prevention incentives.

Unfortunately, virtually all the victim compensation the legislation proposed by Congress to date today is uninsurable. Whether these bills create a private cause of action, a nonfault compensation mechanism, or, worst of all, in our view, both, these bills to one extent or another would so weaken the evidentiary and burden of proof requirements in the common law as to cast upon defendants and their insurers what we view as the impossible task of identifying which of the millions of Americans with long latency diseases and other health problems did not contract them through exposure to hazardous substances and therefore are ineligible for compensation.

In much the same fashion as the 1976 Swine Flu Immunization Program did, these bills would so broaden the potential class of eligible claimants as to render our previous insured loss and expense experience essentially meaningless in trying to predict the costs that these bills would impose on industry.

Many of the pending proposals would dramatically weaken the common laws barriers to admissibility of irrelevant or highly prejudicial evidence on questions of disease causation. Some would not only permit the introduction of such evidence but also would allow that introduction to create a rebuttable presumption in favor of the claimant or plaintiff on the crucial disease causation issue.

Questions of fairness aside for the moment, consider the implications of these changes in the context of the manner in which most hazardous substance-related claims are likely to arise; that is, through surveillance that would typically result in discovery of an incremental increase in the historically expected morbidity or mortality from a given disease in a given locality.

If, upon investigation, a particular hazardous substance in the water supply were implicated in those excess illnesses or deaths, how would it be possible under this new system to determine which of the expected and unexpected or excess illnesses or deaths resulted from the ingestion of the substance?

For example, if the historical mortality from liver cancer of 100 persons per year increased by only 2 percent in a given locality and a hazardous substance in the local water supply were implicated in those excess deaths, only 2 of the 102 estates involved would be theoretically eligible to make a claim but in actual practice with reduced evidentiary rules, weakened standards of proof and a presumption of causations, it is going to be impossible to deny the claims from all 102 of those estates since all the decedents involved did ingest the substance.

The resulting overinclusivity of such a remedy, in our view, would be tantamount to a national health insurance system for all sufferers of long latency disease, and its costs would be beyond calculation.

If the costs of such an overinclusive system were imposed, through a tax, solely on those enterprises producing or handling hazardous substances, those enterprises would be required to internalize many times the costs of the harm they actually cause. This would not only be patently unfair but would so unhinge the relationship between responsible conduct and the imposition of liability as to make any industry's investment in loss prevention and safety research a waste of money.

The only plausible rationale, in our view, for a change in the current law is the observation by the study group that the difficulty and expense of bringing lawsuits make the tort system an inadequate remedy for persons who believe that their relatively minor injuries and/or damages resulted from exposure to hazardous substances. If it is the goal of the advocates of change to deal with the problem of potentially large numbers of persons with relatively minor losses, the tort system is the worst possible starting point for a victim compensation mechanism.

Yet virtually all the pending bills seek either to convert a fault-based system into a nearly nonfault compensation mechanism through corruption of its evidentiary and burden of proof requirements, or to piggyback the traditional tort remedy on top of a nonfault compensation program.

We have to tell you, from our experience with State no-fault auto insurance laws which sought to mate a nonfault approach to compensation with fault-based remedies, that the two systems are inherently incompatible. It is simply not possible to create an affordable system that will compensate all injured persons without regard to fault and give them the very generous damages that the tort system occasionally awards to some plaintiffs.

There simply has to be a choice. Either everyone receives limited benefits without regard to fault, or the few who prove another party at fault receive unlimited damages.

Unhappily, the Study Group's compromise approach would be nearly the most expensive of the conceivable options. Essentially, the study group recommended that claimaints be permitted to recover, under unbelievably lax tier I evidentiary and procedural rules, compensation for economic loss for which they have already been or easily can be reimbursed from private sources, and to turn the entire proceeds from these adminsitrative awards over to attorneys to finance tier II lawsuits for pain and suffering and other intangible damages.

If you don't mind, let me tell you exactly how this would happen. The study group recommended that only public sources of collateral benefits—Medicare, Social Security and the like—be deducted from tier I awards. Approximately 90 percent of all Americans have some form of health insurance, and the overwhelming majority of working Americans have employer-provided sick leave and wage continuation benefits. But under the tier I proposal of the study group, none of these benefits would be considered in calculating administrative awards.

Thus, if a typical American were to allege under a tier I proceeding that his disease was the result of a nonoccupational hazardous substance exposure and the defendant or a fund were unable to overcome a rebuttable presumption to this effect, the administrative agency would be required to award the claimant all his $10.000 in medical expenses and $5,000 in lost wages without consideration of the fact that his private health insurer would cover all the medical expenses and his employer would cover or already had covered most, if not all, of his wage loss.

Having received his administrative award of $15,000 and having turned his unpaid medical bills over to his health insurer, the tier I claimant could afford at no cost to himself to turn the entire award over to his attorney to finance the expert testimony and other expenses necessary to maintain a tier II lawsuit against a defendant who has already paid, either through taxes or subrogation, or both, the costs of the administrative award.

Since the study group proposed no limitation on a tier II plaintiff's ability to plead damages already reimbursed in tier I, recommending only that he be required to reimburse the tier I system for anything he recovers in tier II, and since the study group also recommended no variation in the traditional collateral source rule in the tier II proceeding, the plaintiff's lawyer in this case could plead the $15,000 already reimbursed by the tier I system as special damages or economic loss in the tier II lawsuit.

Now in most jurisdictions general damages—noneconomic loss, pain and suffering and the like—are calculated as a multiple of the special damages or economic loss involved in the case. So, looking at our hypothetical $15,000 in special damages, and assuming pain and suffering damages of five times those special damages, our well-financed plaintiff could win a tort judgment of $90,000, $15,000 in special damages, and $75,000 in general damages. Out of this judgment, the gratuitous $15,000 advanced by the tier I system would be reimbursed, the lawyer would take his one-third, or $30,000, contingent fee, and the plaintiff would pocket $45,000 tax-free and clear.

Now, if the tier I system were to require the deduction of all collateral sources of benefits paid or payable from administrative awards, and the statute were to prohibit the pleading in a tier II proceeding of benefits received from other collateral sources, the tier II picture would be quite different. In our hypothetical, the tier I claimant would be awarded only that portion of his medical expenses and wage loss not covered by his health insurer or his employer and would be able to plead none of these benefits as damages in a tier II proceeding, a fact that in our hypothetical would end the lawsuit before it began.

I mention this only to suggest that if the Congress believes what the study group assumed, that there are large numbers of persons suffering hazardous substance-related injuries too insignificant to make pursuit of a tort remedy economically feasible—and wishes to create an administrative alternative for these persons, we suggest that it consider building into that remedy two safeguards against its abuse: (1) the mandatory deduction of all paid or payable collateral benefits; and (2) a prohibition against introduction in a subsequent lawsuit of any damages reimbursed or reimbursable by any other source, including the administrative system.

In this fashion, the victim compensation bill could be limited to its intended purpose and prevented from becoming a back-door route to a national health insurance program or the greatest engine for tort litigation since the advent of the contingent fee.

A similar result could be achieved by requiring claimants to make an irrevocable election between the two remedies, so long as all collateral sources paid or payable are deducted from administrative awards.

We thank you for your consideration of our views.

[The statement of Mr. Cheek follows:]

STATEMENT OF LESLIE CHEEK III, VICE PRESIDENT-FEDERAL AFFAIRS, CRUM & FORSTER INSURANCE COS.

Mr. Chairman and members of the subcommittee, The Crum & Forster Insurance Companies are the Nation's fourteenth-ranked group of property-casualty insurers, with 1982 premiums of approximately $1.7 billion. We appreciate the opportunity to appear before you to discuss proposals to create new mechanisms for the compensation of persons alleging injuries or diseases resulting from exposure to hazardous substances.

As insurers, it is our chosen lot to collect and pool the funds that those subject to a variety of liability regimes will need to meet their legal obligations and that we need to fulfill our contractual obligation to defend our policyholders against third-party claims.

Our companies are among the largest writers of coverage for the principal non-fault system in the United States, workers' compensation, and of coverage for businesses subject to tort and environmental cleanup liability, Comprehensive General Liability (CGL) and Environmental Impairment Liability (EIL). We also write a substantial amount of insurance for vehicle owners in States with hybrid fault and no-fault systems of auto accident compensation. The lessons we have learned from our experience in servicing these liability and compensation schemes may be useful to the Subcommittee.

Our principal interest in any compensation or liability system is the predictability of its costs. Without reasonable certainty as to how many persons will make claims against the system, and how much on the average each claim will cost, we cannot determine the appropriate premium for the coverage we provide.

The Congress, too, must be sensitive to the predictability of the costs of any system it creates to deal with what we believe is a poorly defined and perhaps non-existent problem. Congress' last venture into a specialized compensation mechanism, the Black Lung Benefits Act of 1969, was initially estimated to cost less than $40 million per year. Costs under the Act are currently $2 billion per year.

The premise underlying the Superfund section 301(e) Study Group's proposals for new mechanisms to deal with hazardous substance-related claims is that there are (or will be) large numbers of persons with demonstrable injuries or diseases resulting from exposure to hazardous substances who are not being (or will not be) compensated under current law.

We are unable to find, in our loss and expense experience or elsewhere, any evidence that current legal remedies are failing to provide adequate redress to persons eligible to make claims for injuries resulting from exposure to hazardous substances. (In a few States, anachronistic statutes of limitation still pose procedural barriers to claims in long-latency disease cases in both occupational (workers' compensation) and non-occupational contexts, but the number of such States is dwindling rapidly.)

Indeed, our experience suggests that the success rate of claimants under both workers' compensation and the tort system in hazardous substance-related cases is improving steadily, as innovative counsel develop, and sympathetic courts adopt, new theories of liability. Case names that send shudders through underwriters' frames—*Borel, Sindell, Bichler* and *Beshada,* to list only four—are ample evidence of this trend.

But neither we, nor anyone else that we know of, can determine how many persons with demonstrable injury resulting from exposure to hazardous substances are not availing themselves of current legal remedies. Anecdotal evidence (e.g., the paucity of claims under California's 1981 Hazardous Substance Compensation Account) suggests that the number is extremely small, but the proponents of legislative change (e.g., the Superfund section 301(e) Study Group, the Environmental Law Institute) have assumed that the number is (or will be) large.

Insurers are prohibited by law and the demands of competition from basing their pricing decisions on speculation. While the past has not always been a reliable clue to the future, we have thus far survived looking only backward. We think the Congress would be wise to take a leaf from our book on hazardous substance compensation legislation, and postpone action until there is hard and consistent evidence that it is needed.

We also think Congress should be concerned about the insurability of those who may be required, in whole or in part, to fund any system it ultimately creates. Insurers apportion commercial insurance premiums among policyholders primarily on the basis of their loss history, thereby assuring that those who cause harm ultimately pay for it, and providing a marketplace incentive for loss prevention. An uninsurable system funded by a tax would be blind to variations in corporate conduct and would remove any incentive to invest in safety.

Moreover, an uninsurable compensation mechanism would be like a new car without an engine: it would look great, but it wouldn't get you anywhere.

Unfortunately, virtually all of the hazardous substance compensation proposals advanced in the Congress to date are uninsurable. Whether they create a private right of action, a non-fault compensation mechanism, or (worst of all) both, these bills, to one extent or another, would so weaken the evidentiary and burden of proof requirements of the common law as to cast upon defendants and their insurers the impossible task of identifying which of the millions of Americans with long-latency diseases and other health problems did not contract them through exposure to hazardous substances and therefore are ineligible for compensation.

In exactly the same fashion as the 1976 Swine Flu Immunization Program did, these bills would so broaden the potential class of eligible claimants as to render previous insured loss and expense experience useless to insurers trying to predict the costs the bills would impose on industry.

Beyond unpredictable costs, we see in many of the pending proposals a serious threat to efforts to prevent harm from occurring. To the extent that they would (1) compensate those who do not deserve to be compensated as well as those who do; (2) assess costs against chemical feedstock manufacturers and hazardous substance handlers with no regard for their conduct as corporate citizens; (3) abandon legal principles long regarded as fair and evenhanded; and (4) impose no controls either on the number of those eligible to recover or on the amount of individual recoveries, these proposals would entirely eliminate the incentives that most States' tort law provides to business to invest in safety and to prevent harm from occurring. Let me address each of these points.

First, overinclusivity. Many of the pending proposals would dramatically weaken the common law's barriers to the admissibility of irrelevant or prejudicial evidence on questions of disease causation. Some not only would permit the introduction of such evidence, but also would allow that introduction to create a rebuttable presumption in favor of the claimant or plaintiff on the crucial disease causation issue.

Questions of fairness aside for the moment, consider the implications of these changes in the context of the manner in which most hazardous substance-related claims are likely to arise—as a result of surveillance by the Centers for Disease Control (CDC) and/or the Agency for Toxic Substance and Disease Registry. Such surveillance would typically result in the discovery of an incremental increase in the historically expected morbidity or mortality rate from a given disease in a given locality. If, upon investigation, a particular hazardous substance in the water supply were implicated in the excess illnesses or deaths, how would it be possible under the new rules to determine which of the expected and excess illnesses or deaths resulted from ingestion of the substance?

Moreover, the smaller the incremental increase in expected morbidity or mortality, the larger would be the class of persons whose illnesses and deaths were plainly not caused by ingestion of the substance, but who, by virtue of the relaxed causation requirements, would be eligible for compensation. For example, if historical mortality from liver cancer of 100 persons per year increased by only two percent in a given locality, and a hazardous substance in the local water supply were implicated in the excess deaths, only two of the 102 estates involved would be theoretically eligible to make claims, but, in actual practice, it would be impossible to deny claims from all 102 estates, since all the decedents ingested the substance.

Under ordinary common law rules, the burden would fall on the members of the class to demonstrate, by a preponderance of the relevant evidence, which of the deaths, if any, resulted from disease caused by ingestion of the hazardous substance. Most of the class, and perhaps all, would be unable to sustain this burden. If none could, then perhaps injustice would result to the estates of the two decedents who might, in fact, have contracted cancer as a result of ingesting the substance.

Under some of the pending proposals, however, it would be the defendant's burden to show which of the 102 decedents he did not cause to contract cancer, and he would be required to rebut a presumption of causation arising in part from the mandatory admission of possibly irrelevant and perhaps prejudical evidence. If he could not overcome this presumption, then all 102 members of the class would recover, despite the fact that both they and the defendant know that 100 of them deserve nothing from the defendant. Is this not a greater injustice?

If you say it isn't, and that massive overinclusivity of the remedy is the price society must pay to make sure that the deserving two are compensated, then we submit to you that such a remedy would be tantamount to a national health insurance system for all sufferers of long-latency diseases, and that its costs would be beyond calculation.

We also submit to you that such a remedy would rob the chemical industry not only of its money and its capacity to compete in international commerce (and therefore to provide jobs), but also of any incentive to invest in safety research and technology, for "victim compensation" would bear no relationship whatsoever to harm actually caused by defendants.

If you say that such a system would be an injustice to defendants, but that deserving claimants should be compensated nonetheless, does that not suggest the absurdity of trying to convert the tort law from a mechanism for the adjudication of individual conduct into a mass compensation system?

Second, assessment of costs. The concept of cost internalization is based on the theory that the conduct of an enterprise can be favorably influenced by requiring it to absorb the costs of the harm it causes. The corollary of this concept is that the conduct of an enterprise can be unfavorably influenced by requiring it to absorb either more or less than the costs of harm it causes.

If the costs of an overinclusive "victim compensation" system were imposed solely on those enterprises producing or handling hazardous substances, those enterprises would be required to internalize many times the costs of the harm they actually cause. This not only would be patently unfair, but would so unhinge the relationship between responsible conduct and the imposition of liability as to make investment in loss prevention and safety research a waste of money. Is this really the price our society wants to pay for "victim compensation"?

Third, the abandonment of traditional legal principles. The premise of the Superfund Safety Group's characterization of the common law as "inadequate" in toxic tort cases was the assumption that difficulties in establishing proof of causation by the traditional preponderance of the evidence would prevent deserving claimants from recovering for their hazardous substance-related harm. We see no evidence to support this assumption in current toxic tort litigation.

But if one concedes, for the sake of argument, that there is some basis for this assumption, what should be done about it?

The Study Group correctly identified the problem of proving causation in toxic tort cases as a matter of science, rather than of law, but sidestepped the awkward, yet patently obvious, conclusion that dealing with the incapacity of most current scientific techniques to establish causal links between exposure and disease will take enormous amounts of both time and money. The Study Group instead proposed to paper over the evidentiary inadequacy of current scientific data by rigging the legal framework into which it would be introduced so as to give it presumptive legal weight in favor of claimants and to thrust upon defendants the burden of overcoming a presumption of causation.

Most of the pending bills adopt variations of this approach, and they would both encourage the debauchery of science by making even evidence which only "tends" to show causation admissible on that time issue, and debase the law by reversing the fundamental principle of our system of civil justice, that a defendant is presumed innocent until proved liable.

From an insurer's perspective, the more radically a statutory cause of action or compensation mechanism for alleged victims of "toxic torts" departs from traditional common law rules and procedures, the more difficult it will be for insurers to enter or remain in the market for environmental liability insurance.

Environmental tort liability is insurable, and insurance against such liability is currently affordable, primarily because the tort system does not compensate everyone who is injured merely because he is injured. A plaintiff must establish the defendant's liability by enough evidence to convince a judge or jury that it is more likely than not that the defendant's conduct was the proximate cause of the plaintiff's harm.

Except in the area of product liability, tort law is a seamless fabric, in that the same basic procedural and evidentiary requirements pertain in any claim, whether for automobile accidents or for hazardous substance injuries. A tug at the thread called burden of proof, undertaken to deal with a narrow range of potentially difficult claims, would undo the entire fabric, as enterprising lawyers beseech sympathetic judges to apply the precedent in other tort claims for the sake of restoring the tort law's traditional procudural uniformity.

Similarly, it is unrealistic to believe that liberal evidentiary rules in "tier one" of the Study Group's proposed system would not find their way into "tier two" tort claims, with totally unpredictable results.

No matter what legal mechanism is used to resolve toxic tort claims, the shortcomings of science will prevent the system from answering most causation questions with the certainty the law currently demands. And these shortcomings will never be fully overcome, not only because there is no one "cause" for most long-latency diseases, but also because of the sheer volume of chemicals in our environment and the moral infeasibility of treating humans as laboratory animals. But the prospect of imperfect scientific solutions to proof of causation problems should not become the basis for abandoning legal principles that our society has long regarded as fair and evenhanded. It seems to us that a higher priority should be given to bettering our understanding of human reactions to the chemicals in our environment.

We and others have repeatedly proposed, as an alternative to ill-advised tinkering with our legal system, the establishment and funding of a mechanism whose responsibility it would be to identify and document cause-and-effect relationships between certain durations and intensities of exposure to specific substances and the development of particular diseases. We are encouraged by the recent creation of the Agency for Toxic Substances and Disease Registry.

Fourth, and finally, controls on aggregate claim frequency or individual recoveries. The only plausible rationale for change in current law is the observation by the Study Group that the difficulty and expense of bringing lawsuits make the tort system an "inadequate" remedy for persons who believe their relatively minor injuries and/or damages resulted from exposure to hazardous substances.

The observation is correct, of course, but it is applicable to the victims of all kinds of injuries, not just toxic torts. The tort lawsuit is not, and was never intended to be, the vehicle for the resolution of all claims. Even among those who make use of it, only about half are awarded damages against defendants. Most tort claims are settled, with or without payment, outside the courtroom.

If it is the goal of the advocates of change to deal with the problems of potentially large numbers of persons with relatively minor losses, the tort system is the worst possible starting point for a compensation mechanism. Yet virtually all of the pending victim compensation proposals seek either to convert the fault-based system of tort law into a nearly non-fault compensation mechanism through corruption of its evidentiary and burden of proof requirements, or to piggyback the traditional tort remedy on top of a non-fault compensation program.

The plain goal of these proposals is to provide all hazardous substance victims with the best of both non-fault and fault-based remedies: eased evidentiary burdens coupled with generous awards. As laudable as this objective may be in theory, it cannot realistically be achieved if the system's costs are to be controllable, predictable, and, ultimately insurable.

We know, from experience with State auto insurance laws that sought to mate non-fault compensation programs with fault-based tort remedies, that the two systems are inherently incompatible. It is not possible, at one and the same time, to compensate all injured persons without regard to fault and to give them the generous damages that the tort system occassionally awards to some plaintiffs. There simply has to be a choice between the two extremes: either everyone receives limited benefits without regard to fault, or the few who prove another party at fault receive unlimited damages.

Unfortunately, the composition of the Study Group guaranteed not only that this necessary choice would not be made, but also that its "compromise" approach would be among the most expensive of the conceivable options. Essentially, the Study

Group's proposal would permit claimants with long-latency diseases to recover, under unbelievably lax "Tier I" evidentiary and procedural rules, damages for which they have already been reimbursed from private sources, and to turn the entire preceeds of their administrative awards over to their attorneys to finance "Tier II" lawsuits for pain and suffering and other intangible damages. Let me illustrate how this would happen.

The Study Group proposed that only public sources of collateral benefits—Medicare, Social Security disablility, State statutory disability income programs, etc.—be deducted from "Tier I" awards, and that "Tier II" tort plaintiffs be faced with the possibility of paying defendants' legal costs only if their tort damage awards were less than 125 percent of their administrative recoveries.

Approximately 90 percent of Americans have private health insurance protection, and the overwhelming majority of working Americans have employer-provided sick leave and wage continuation benefits. Under the Study Group's "Tier I" proposal, none of these benefits would be considered in calculating administrative awards.

Thus, if a typical American worker were to allege, under a Tier I proceeding, that his disease was the result of a nonoccupational hazardous substance exposure, and the defendant (or a fund) were unable to overcome a rebuttable presumption to this effect, the administrative agency would be required to award the claimant all of his $10,000 in medical expenses and $5,000 in lost wages, (assuming these were within the maximum permitted under the system), without consideration of the fact that his private health insurer will cover all of the medical bills and his employer will cover most, if not all, of his wage loss.

Having received his administrative award of $15,000, and having turned his unpaid medical bills over to his health insurer, the "Tier I" claimant can afford, at no loss to himself, to advance the entire award to his lawyer to finance (at no cost to the lawyer) the expert testimony and other expenses necessary to maintain a "Tier II" lawsuit for pain and suffering against a defendant who has already paid, through taxes or subrogation (or both), the costs of the administrative award.

Since the Study Group proposed no limitation on a "Tier II" plaintiff's ability to plead damages already reimbursed in "Tier I," (recommending only that a plaintiff be required to reimburse the "Tier I" system out of any tort proceeds), and no variation from the traditional "collateral source rule" in "Tier II" proceedings, the plaintiff's lawyer may plead $15,000 in "special" damages, or economic loss.

In most jurisdictions, "general" damages—pain and suffering and other non-economic losses—are calculated as a multiple of (anywhere from four to eight or nine times) "special" damages. Assuming general damages of five times special damages, our well-financed plaintiff could win a tort judgment of $90,000—$15,000 in special damages and $75,000 in general damages.

Out of this judgment, the gratuitous $15,000 advanced by the "Tier I" mechanism for expert witness and other costs of the lawsuit would be repaid, the lawyer would take his $30,000 contingent fee, and the plaintiff would pocket $45,000, tax-free and clear.

Now, if the "Tier I" system were to require the deduction of all collateral sources of benefits, paid or payable, from administrative awards, and the statute were to prohibit the pleading, in a "Tier II" proceeding, of any damages reimbursed by these sources or in a "Tier I" proceeding, the "Tier II" picture would be quite different.

In our hypothetical, the Tier I claimant would be awarded only that portion of his medical expenses and wage loss not covered by his health insurer or his employer, and would be able to plead none of these benefits as damages in a "Tier II" proceeding, a fact that would end the lawsuit before it began.

If the Congress believes what the Study Group assumed—that there are large numbers of persons suffering hazardous substance-related injuries too insignificant to make pursuit of a tort remedy economically feasible—and wishes to create an administrative remedy for these persons as an alternative to the tort system, we suggest that it consider building into that remedy two safeguards against its abuse: (1) a mandatory deduction of all paid or payable collateral benefits from administrative awards; and (2) a prohibition against the introduction in a subsequent lawsuit of any damages reimbursed or reimbursable by any other source, including the administrative system.

In this fashion, a "victim compensation" bill could be limited to its intended purpose and prevented from becoming either a backdoor national health insurance program or the greatest engine for tort litigation since the advent of the contingent fee.

A similar result could be achieved by requiring claimants to make an irrevocable election between the two remedies, so long as all collateral resources, paid or payable, are deducted from administrative awards.

We thank you for your consideration of our views, and would be pleased to answer your questions.

Mr. **Levitas**. Thank you, Mr. Cheek.

Ms. Birnbaum.

Ms. **Birnbaum**. Thank you, Mr. Chairman.

Mr. Chairman and members of the subcommittee, I appreciate the opportunity to speak with you this morning. I am here today representing the American Insurance Association, which is composed of more than 160 publicly owned insurance groups which presently provide the majority of general commercial liability insurance in the United States.

It is likely that the members of the American Insurance Association will be called upon to insure the defendants and manufacturers that will be caused to pay under any scheme of liability that is going to be adopted.

Mr. Cheek, I think, has explained some of the elements that the American Insurance Association believes must be considered in any compensation scheme. I think I have some expertise that might be helpful to the committee based on some of the questions the members have been asking and perhaps in my remarks I can leave my prepared statement and try to focus on some of the issues that I think are of great concern to this committee.

I have taught tort law at New York University Law School for nearly 10 years and I have participated in many of the types of toxic tort cases that are presently being litigated in the country. For example, I have participated in lead cases, and in cases involving DDT, DBCP, formaldehyde and other kinds of chemicals and drugs.

In my capacity as an academic and as a lawyer I have watched this area of the law develop from its infancy in 1973 with the Fifth circuit decision on the *Boree Asbestos* landmark case that set the parameters for determining a liability in toxic tort case.

Speaking in both capacities I think I can answer some of the questions that have been raised previously. Perhaps I can put this area in some sort of context for you from the defense point of view.

I think you have heard from Mr. Baron, the plaintiff's point of view; and from the questions—I wasn't present to hear the testimony—I think much of what he said would be reflected in my own testimony. That is the tort system, in so far as toxic torts are concerned is not broken and should not be tampered with.

I think one of the things that we all should do as lawyers and as Congressmen is give that system, which has worked so well in so many areas, an opportunity to resolve the problems in the toxic tort area. I don't believe that there are many victims out there—assuming they are injured—who are not able to get compensation through the tort system.

Let me give you some examples.

In New York there are the famous *Love Canal* cases. These are presently pending in Buffalo, NY with over 1,300 lawsuits being handled by very vew law firms who have built up enormous expertise in this area. Anyone in the Buffalo area who live around Love Canal dumpsite has available a legal remedy; many are already using the tort system to pursue that remedy.

Now we will have to see how those cases progress. At the moment they are in discovery. But if there is proof of causation—and that proof is being developed by plaintiffs' attorneys—and the people have been injured, there is presently in place a system that can provide adequate compensation. I believe we should use that system.

Another example of .how the system is operating is the *Agent Orange* cases. There is presently pending a class action in the eastern district of New York of potentially 2.5 million servicemen. That case is progressing and the court, under the common law system, will have an opportunity to resolve many of the issues of class action participation, and causation liability that the plaintiffs are presently advancing.

Mr. MOLINARI. Could I interrupt you please?

Ms. BIRNBAUM. Certainly.

Mr. MOLINARI. You made the statement that you see no reason to disturb the existing system because those who have been injured have been receiving recoveries. Then you go on to recite instances where there have been multiple plaintiffs who have instituted litigation which is in the pending stage.

Ms. BIRNBAUM. I am sorry; if I said that, I misspoke. What I intended to say is that the tort system is responding not only in these lawsuits that are progressing but also in other lawsuits which I can recite to you that have been settled or that have gone to verdict.

Mr. MOLINARI. Can you recite—I think it would be very helpful—could you recite cases reflecting extensive litigation? I am talking about large numbers of cases.

Ms. BIRNBAUM. Yes, sir.

Mr. MOLINARI. Litigated and recoveries? We would be happy to receive that.

Ms. BIRNBAUM. What I would like to do if I may is submit a written statement after this hearing where I can cite you to reported cases and to cases that have been settled, which are a matter of public record. I can give you some examples now, but I can certainly provide a much broader range of cases in a written admission. For example, there was recently a case in Idaho brought by children who had been living around lead smelters. That case was settled and payment of several million dollars was made to the children who had brought the claims.

A case brought by more than 1,100 plaintiffs was recently settled in Alabama. That case involved allegations of pollution from a DDT plant, was settled for several million dollars.

There have been many lead smelting cases that have been settled and substantial amounts of money have been provided. I can supply for the record other examples of these kinds of settlements that have occurred, some with great fanfare and some without great publicity. There was recently a case that went to trial and the verdict was over $50 million. The claim arose out of a chemical spill in which railroad workers were exposed to dioxin during the clean up efforts.

I might question the amount and reliability of these verdicts, but I think what these cases indicate is that there is a tort system presently in place, which is responding and which will continue to respond to claims. Until we find that the tort system is not working or not responding adequately to these needs of injured persons, I think we should not be tinkering with an entirely new system which will pose enormous administrative costs.

[The following was received from Ms. Birnbaum:]

COMPILATION OF SOME RECENT VERDICTS OR SETTLEMENTS INVOLVING EXPOSURE TO TOXIC SUBSTANCES

1. DIOXIN

Lowe v. *Norfolk & Western.*—Forty-seven railroad workers, alleging exposure to Dioxin while cleaning up a chemical spill, were awarded nearly $58 million by an Illinois state court jury. Three defendants had settled for $7 million the day trial was to begin. An appeal is presently pending in the Illinois Fourth District Court—Appellate Division.

2. LEAD

Bunker Hill.—On April 4, 1983, an Idaho state court approved a settlement awarding more than $23 million to 37 children. The children claimed that their health had been damaged due to lead emissions from the Bunker Hill smelter in Kellogg, Idaho (*Prindivill* v. *Bunker Hill Co. and Gulf Resources, Inc.*).

A similar suit in federal district court in Boise was settled in 1981 (*Yoss* v. *Bunker Hill Co.*). The settlement awarded nine children annuities and cash worth a total of about $7 million.

3. BENZENE

Lemaire v. *Texaco, Inc.*—A $4.3 million long term settlement was awarded in the Eastern District of Texas on January 8, 1982 to two shipyard workers and the widows of two former shipyard workers. The workers had contracted leukemia after cleaning barges and vessels of a chemical residue containing benzene.

4. PARAQUAT

Fereoee v. *Chevron Chemical Co.*—The family of a gardner who died of pulmonary fibrosis following exposure to the herbicide paraquat was awarded damages of $137,500 in the District Court of the District of Columbia on November 4, 1982. Appeal is presently pending in the District of Columbia Court of Appeals.

5. DDT

Olin Corp.—Parties in three suits (*Cloud* v. *Olin Corp., Freeman* v. *Olin Corp.* and *Erskine* v. *Olin Corp.*) reached settlement in the Northern District of Alabama as announced on December 27, 1982. The plaintiffs, residents of Triana, Alabama, claimed a higher than normal incidence of cancer and high blood pressure due to high concentrations of DDT near a former Olin manufacturing plant. The settlement was for $24 million, $19 million of which was earmarked for personal injury claims.

6. DBCP

Consolidated Chemical Exposure Cases.—Six chemical workers and three wives were awarded $7.9 million in compensatory damages by a California state court jury on March 14, 1983. The award held Dow Chemical Company liable for sterility of the workers due to exposure to the pesticide DBCP. A number of similar suits were settled out of court, as were claims against the University of California and one of its scientists.

7. FORMALDEHYDE

Alley v. *Gubser Development Co.*—On August 31, 1983, a couple alleging complication of a liver condition, respiratory problems, headaches and nausea due to inhalation of formaldehyde fumes in their mobile home, was awarded $50,000 in compensatory and $510,000 in punitive damages by a jury in Denver federal district court.

Boelens v. *Redman Homes.*—A Texas federal district court jury returned a verdict of $291,402 against a manufacturer and a retailer of a mobile home on April 27, 1983. The plaintiffs, a woman and her two daughters, claimed respiratory ailments and toxic hepatitis had resulted from inhalation of a formaldehyde present in their mobile home.

Lee v. *Service Insulation Co.*—In April, 1982, a settlement of $150,000 was reached in Oregon state court for a husband and wife who had sustained injuries upon inhalation of formaldehyde released from residential insulation.

Eckert v. *Guerdon Industries, Inc.*—A $600,000 verdict was returned in the District of South Carolina on January 12, 1982 for a woman who sustained lung damage through inhalation of formaldehyde in her mobile home.

8. CHEMICAL EXPLOSIONS

NL Industries.—NL Industries has agreed to pay between $500,000 and $1.2 million to each of four persons who had filed suits in the Southern District of Mississippi. The suits were filed after a June 2, 1982 chemical explosion killing three and injuring about 60 (*Price* v. *NL Industries, Spivey* v. *NL Industries, West* v. *NL Industries* and *Robinson* v. *NL Industries*).

Lara v. *Navaho Refining Co.*—On August 20, 1981 a landfill worker obtained a jury verdict of $335,632 for injuries caused by inhalation of methyl chloride gas produced by the explosion of chemical storage barrels.

Harrington v. *B.F. Goodrich.*—A settlement was recently reached for $1.25 million after a jury returned a $2 million verdict in favor of a rubber plant worker who allegedly contracted bladder cancer as a result of exposure to chemicals used in the manufacture of synthetic rubber.

Ms. BIRNBAUM. In addition, the tort system makes distinctions between good conduct and bad conduct, and I think this factor is very important. Ms. Hall made the comment that we really want to encourage and provide incentives to manufacturers to do the right thing. Well, I don't believe we do that by taxing them in a way that everyone pays according to the numbers of products they produce; rather than by holding liable only those who have acted poorly. In fact, it is the common law system that imposes punitive damages on those who have acted in gross and reckless disregard of human safety. In this way the tort system acts as to deter bad conduct.

We should be providing an incentive to companies to act promptly and responsibly, other than devising a means of holding everybody absolutely liable. Those who have done everything properly in depositing the chemical into a dumpsite will be held just as liable under an absolute liability system as those who have acted recklessly and criminally and may now even be out of business.

There is something about such a system that is unjust. I am not suggesting that there shouldn't be compensation for people who have been exposed and injured by toxic substances. What I am suggesting to you is that there is now a system in place to provide compensation and we should give that system an opportunity to develop; if it doesn't respond then perhaps maybe 5 years from now we will have to come back to the drawing board and consider whether we can do something.

[The statement of Ms. Birnbaum follows:]

STATEMENT OF SHEILA L. BIRNBAUM

Mr. Chairman and members of the subcommittee, I appreciate the opportunity to speak with you today as counsel representing the American Insurance Association. The American Insurance Association is composed of more than 160 publicly owned insurance companies providing property and casualty coverages—indeed, presently providing the majority of general commercial liability insurance in the United States. It is likely that the members of the American Insurance Association will be asked to provide insurance in the future for companies subject to liability to those who claim injury as a result of exposure to hazardous substances. The members of the American Insurance Association, and other insurers, have responded in the past to the emerging environmental liabilities; new coverages have been developed and competitively marketed. As Congress proceeds to investigate approaches to the compensation of victims of hazardous substance exposure, the American Insurance Association has an interest in seeing that the system as it develops is reasonable, equitable and insurable.

The general concerns of the American Insurance Association in this area have been made known to you through their position paper, dated July 12, 1983, which I understand you have already received. That position paper addresses comments both to the pending legislation generally, and specifically to certain recommendations made by the majority of the CERCLA section 301(e) Study Group.

I wish to focus my remarks today on two legal principles which form the centerpiece of the Study Group majority's recommendations: first, its recommended presumption that a disease or injury resulted from exposure to hazardous chemicals when no proof of that fact is available; and second, its proposal to use a formulation of strict liability which leaves no room at all for the traditional notion that a party at fault pays for the damage it causes.

THE PRESUMPTION OF CAUSATION

The Study Group majority recommended that in its proposed administrative proceeding, a claimant be afforded a presumption of causation upon a showing that his disease was of "the kind" which is "known" to be caused by a particular chemical to which he was exposed. The fund would be entitled to the same presumption in any subrogated proceedings to recover against a private company.

As a practical matter, this presumption makes private industry the guarantors of the health of large segments of the population at large. Many of the diseases which are alleged to be caused by hazardous substances—for example, cancer—are thought to have many possible causes. Claimants who can show that they were exposed to any particular carcinogen would be able to shift the burden the disease. Although the particular cancer might have been caused by other chemicals, smoking, diet, overexposure to sunlight, repeated irritation, or combinations of these and other factors, it would be all but impossible to prove that the particular chemical at issue did not cause this claimant's cancer. After all, the claimant, and not the defendant, is in possession of the facts concerning the onset of disease and the symptoms involved, personal diet and habits (such as smoking) that might be other causes of the disease, and all background information concerning the claimant's history and exposure to this or other hazardous substance. Yet despite claimant's possession of the relevant facts, the Study Group majority's proposed presumption would defy traditional principles of proof and place an impossible burden of disproof on the defendant.

As a result of this scheme, the defendant would be required to pay, not only for any cases of cancer caused by the release of the particular carcinogen, but also for all other cases of cancer that may arise from any other cause in the environment or even genetically. It has been estimated that approximately 2 percent (and no more than 5 percent of cancer deaths are caused by air, water and food pollution in all its forms; [1] 1 percent or less may result specifically from exposure to pollutants found in hazardous waste sites.[2] Thus, 95 percent or more of the damage payments that would be required to be made to those who have cancer and reside near a dump site, based on this presumption of causation, would be for cases not caused by any chemical released by the defendant.

Further, although the Study Group majority itself only recommended the use of this presumption of causation in proceedings for administrative awards and resulting subrogated proceedings by the fund to obtain indemnity from private companies involved, the likelihood is that this approach would find its way into the common law in time. Thus, in addition to medical expenses and lost earnings, pain and suffering awards would also be based on the presumption, despite the fact that a very large percentage of such awards should never be made because the injury or disease was really due to other causes.

This result is inequitable; it is also unwise. To fund what amounts to a social health scheme by loading the entire costs onto the chemical or oil industries—especially in the face of foreign competitors who are not burdened by such staggering costs—could prove disastrous to vital American industries.

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[1] See R. Doll and R. Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," 66 J. Nat'l Cancer Inst. 1192, 1245–50, 1256 (1981); Technology Assessment Board, 97th Cong., "Assessment of Technologies for Determining Cancer Risks from the Environment," 91 (June 1981).

[2] Comments of Jim J. Tozzi at a conference on May 7, 1983 concerning Office of Management and Budget-sponsored Inter-Agency Study of Toxic Torts.

"STRICT LIABILITY"

The Study Group majority recommended that all consideration of fault be eliminated in assessing liability against defendants. This is a radical departure from traditional legal principles. Even under the principles of "strict liability" as applied today in many courts, and as embodied in the Restatement (Second of Torts) §§ 519-52, some evaluation of the conduct of the defendants remains an integral part of the decision of any court as to whether or not it should impose liability "without fault." The recommendations of the Study Group majority however, would not permit the courts or administrative tribunals to consider the good or bad conduct of the defendant and, therefore, would not encourage companies to act carefully in handling hazardous waste.

The traditional reasons for imposing liability on defendants who are in some way "at fault" are two-fold: (1) it comports without notions of fairness, and (2) it provides an incentive for the defendant and others to behave properly in order to avoid liability. A failure to consider conduct would remove incentives for industry to behave well. Under the Study Group majority's recommendations, the cost penalties would be assessed regardless, and so there would be no point (from industry's standpoint) in spending time or money "up front" to be careful or to act wisely. Such a result would be a national tragedy, since surely it is wiser to encourage industry to prevent harm in the first place.

This result is exacerbated by the Study Group majority's other recommendations. For example, the fact that a defendant can be jointly and severally liable without regard to fault, means that a very prudent manufacturer who merely had the misfortune to place its waste in the same disposal site as another company which acted recklessly, or even criminally, could nonetheless be held liable for the entire judgment. Given the operation of the Study Group majority's proposed presumption of causation, discussed above, a prudent manufacturer might be held liable even though the entire harm was caused by the missing, negligent manufacturer. If the negligent enterprise cannot be found, or is judgment proof, the prudent manufacturer will have no recourse for contribution or indemnity. Thus the care taken by the "good" company will be of no avail whatsoever.

Further, the Study Group majority suggested that the administrative recovery mechanism be funded initially by a tax on certain industries. Once again, such a tax would fall on the heads of "good" and "bad" actors alike, imposing huge financial burdens on private industry without providing any incentive for proper, careful behavior.

If Congress feels that there should be compensation for all injured persons who contract cancer or other diseases that might be caused by hazardous wastes, it should consider an effective method for doing so. The costs of these benefits ought not be levied in such a way as to leave no incentive at all for those who act responsibly. We have all benefited by obtaining the products, the high standard of living, and the relatively longer and healthier lives that modern industry and society have afforded. Although those who have negligently or recklessly disposed or waste products should certainly pay any victims who have been injured by their acts, the huge costs of a broad compensation scheme should not be placed on a small segment of American industry without regard to fault.

CONCLUSION

Our traditional legal principles have been based on concepts of justice and fairness. Any wholesale, radical abandonment of these principles runs the risk of seriously affecting the goals of justice and accident prevention that have been built into our tort law. The Study Group majority's recommendations are not only poorly thought out in the context of "toxic torts", as we have seen above, but they also run the distinct risk of weakening the overall fabric of tort law in other, unintended areas. I respectfully suggest that considerably more thought and care must be expanded by Congress, academicians and others before any solution to the toxic tort problem is adopted. Irresponsible "solutions" to our perceived problems may turn out to be worse than the problems they seek to cure.

AMERICAN INSURANCE ASSOCIATION:

PRELIMINARY PAPER ON PROPOSED HAZARDOUS SUBSTANCE VICTIM COMPENSATION LEGISLATION

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I. INTRODUCTION

The American Insurance Association is a trade association composed of more than 160 publicly owned insurance companies providing property and casualty coverages. The Association's members provide the majority of general commercial liability insurance in the United States. It is likely that the members of the American Insurance Association will be asked to provide insurance for those subject to liability for hazardous substance compensation. A market for such insurance can develop only if the compensation liabilities to be insured are both reasonably predictable and affordable. The members of the American Insurance Association, and other insurers, have responded in the past to the emerging environmental liabilities; new coverages have been developed and competitively marketed. Thus, the American Insurance Association has an interest in seeing that the liability system as it develops is reasonable, equitable and insurable.

This Paper expresses the position of the American Insurance Association in response to state and federal legislative proposals concerning "hazardous substance" liability claims. Many of these bills are based in part on recommendations made by a majority of the

"Superfund Section 301(e) Study Group" (the "Study Group majority") in a Report dated September 1982 (the "Report") entitled Injuries and Damages from Hazardous Wastes -- Analysis and Improvements of Legal Remedies, submitted to the United States Congress pursuant to Section 301(e) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 ("CERCLA"), P.L. 96-510.

II. EXECUTIVE SUMMARY

The law of torts, as implemented through our nation's courts, is the primary medium for resolving disputes as to compensation for claimants alleging injury from exposure to hazardous substances. The tort liability system as it has evolved over the centuries -- and as it continues to evolve today -- is designed to serve three functions: justice, compensation, and incentives for proper conduct. Ideally, a plaintiff will receive compensation in the courts only if it is just to require the defendant to pay, and if requiring such payments overall will tend to provide proper incentives for prudent behavior and to minimize the incidence and seriousness of such damage in the future.

Under traditional principles, a defendant whose fault or abnormally dangerous activity is determined to have caused an injury must compensate an injured claimant; such a determination is consistent with our notions of justice and fair play, and also serves as an incentive to that defendant and others, to do better in the future. Moreover, an injured plaintiff may not obtain compensation from parties randomly selected out of the general population who have not caused the injuries at issue and who have not engaged in any improper or abnormally dangerous activity; compensation is not provided in such a case because it would not be consistent with our notion of justice, would not penalize the parties actually at fault or encourage them and others to be more careful in the future.

The Study Group majority, and the sponsors of various bills subsequently introduced, have advocated radical departures from the present common law tort system to ease plaintiffs' burden of obtaining damages for personal injury or property loss allegedly resulting from exposure to certain hazardous substances. These proposals include devices such as:

1. A presumption that hazardous substances caused a plaintiff's injuries or disease where plaintiff cannot show that this is the case.

2. Absolute liability, without any regard to the presence or absence of fault on the part of the defendant.

3. The imposition of liability on a particular defendant far in excess of the harm that defendant caused, through the use of presumptions that a particular defendant's substance caused harm when no proof of that fact is available, and the use of joint and several liability.

4. The use of administrative remedies partially or completely funded by a tax on certain industries, which falls on good and bad actors equally and removes any incentives for proper conduct.

5. The imposition of retroactive liability.

6. The use of an administrative "compensation" scheme as an add-on to potentially unlimited tort liability, thus providing both widespread recoveries under relaxed standards of proof, and no limitation on the amount of recoveries that are available.

The American Insurance Association opposes such radical proposals because they detract from the combined ideals of justice, compensation and incentives for proper conduct that are the basis of the present tort system, and because it has not been demonstrated that such legislation is necessary or will improve the present system.

The proposals made thus far have the potential of providing awards for every case of cancer, tumor, genetic damage, and other diseases of the type claimed to result from "exposure" to hazardous substances. The cost of the proposed legislation would run into tens of billions of dollars per year for American industries -- a cost far in excess of any harm that has been demonstrated to be caused by hazardous substances, as opposed to the many other factors that affect our everyday lives and health.

Enactment of the specific proposals recommended by the Study Group majority and others, in addition to providing costly and unfair presumptions of causation not warranted by scientific or other evidence, would remove all incentives for industry to use due care in handling hazardous substances, since everyone would be penalized (through absolute, joint and several liability and through taxes) without any regard to fault or lack of fault. Thus the goal of preventing future harm would be abandoned in favor of an overbroad system of after-the-fact compensation. Moreover, the use of an add-on system of administrative recovery in addition to a system of unlimited courtroom recovery would constitute perhaps the most expensive and cumbersome compensation system that could possibly be devised.

The American Insurance Association believes that the proposals, which combine staggering costs, unlimited and unpredictable liability, and the abandonment of the traditional requirements of fault and causation, would be uninsurable, unaffordable, unjust and unwise.

III. THE STUDY GROUP MAJORITY'S RECOMMENDATIONS

Because most of the proposed legislation suffers from the same generic problems that are inherent in the Study Group majority's Report, a brief summary of the Report is included here.

The Study Group was comprised of twelve lawyers, three each appointed by the Presidents of the American Bar Association, American Law Institute, American Trial Lawyers Association, and the National Association of (State) Attorneys General. No medical, scientific, epidemiological, economics, industrial or underwriting experts were included in the Study Group.

The Study Group majority recommended that a two-tier recovery system be established. Tier One would provide an administrative remedy administered by the states but governed by federal law. (Report at 206).* Tier Two -- which would also be available even if a claimant first received a Tier One administrative award --

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* Citations in this form refer to the pagination of Volume One of the Report as printed by the Government Printing Office, Serial No. 97-12. Volume Two contains lettered appendices to the Report.

would consist of the judicial tort-claim process, modified to make recovery of damages (including damages for pain and suffering, and punitive damages) far easier for plaintiffs.* Vehement dissents from these proposals were recorded in the individual comments of certain Study Group members, especially Chief Judge (ret.) Charles D. Breitel and George Clemon Freeman, Jr.**

Tier One

Tier One administrative remedies as recommended by the Study Group majority would permit recovery of full medical costs (without limit) and 2/3 of lost wages (up to a fixed monthly limit). Death benefits would also be paid to family members who had relied on the decedent for support. (Report at 206, 234).

Tier One administrative recoveries would be based in many cases upon a presumption of causation:

> "If the claimant in an administrative compensation proceeding proves:

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* Through the use of joint and several liability, broader strict liability principles, liberal joinder rules and a more liberal statute of limitations. (Report at 255-60).

** Comments by Charles D. Breitel, Study Group Member (Report at 295); Comments of George Clemon Freeman, Jr. (Report at 305)

> 1) that the source involved in the claim was engaged at the time of exposure in the production, transportation or disposal of hazardous wastes; and
>
> 2) that the claimant was exposed to such hazardous waste by proximity, contact, ingestion, inhalation, or by some other form of exposure; and
>
> 3) that the claimant suffered death or the kind of injury or disease which is known to result from such exposure

then the following rebuttable presumptions should arise:

> (a) that the exposure to the hazardous waste generated, transported or disposed of, by or in the source, contributed to the death, injury or disease of the claimant; and
>
> (b) that the source by or in which such hazardous wastes were generated, transported or disposed of was responsible for the claimant's exposure and for the resulting death, injury or disease." (Report at 213-14 (indentation added)).

A claimant would not be required to demonstrate any fault on anyone's part in order to recover. (Report at 196, 209).

The Study Group majority made no study of the expected or potential costs of its proposed program. It did recommend that the fund out of which these administrative recoveries would be paid be provided initially through a tax on industry. (Report at 245). Further funding would be provided by subrogated agency claims against the owner, operator, generator, transporter, or other "alleged responsible party" after the agency had paid a claimant. (Report at 246). Such subrogated claims by the agency would be made on the same basis as a claimant's original claim -- i.e., including the presumption of causation and lack of any required proof of fault; subrogated claims would not require proof that an owner, operator, generator, or transporter, etc., knew or should have known of the hazard. (Report at 246-47). Thus, a defendant's liability would be nearly absolute.

Tier Two

A Tier One administrative award would not bar a claimant from pursuing further damages in court under Tier Two. The agency granting a Tier One award would be reimbursed from any court award. The Study Group majority recommended that the courts be empowered to assess costs against a plaintiff who had previously obtained an administrative award, if the plaintiff failed to receive an award in court at least 25% larger than that granted by the agency. Such cost assessment, however, would not be mandatory. (Report at 197-98).

The Study Group majority recommended that the judicial process adopt a very liberal statute of limitations (based not only on discovery of the injury or disease but also on discovery of the cause of the injury or disease), liberal joinder rules, and joint and several liability for each defendant who has "contributed" to the injury, above some de minimis level. (Report at 255-58).

IV. DEFECTS IN THE PROPOSALS

A. Proponents' Failure to Demonstrate a Need for Radical Change

The proposals of the Study Group majority and others have been put forward despite the complete absence of evidence that large groups of plaintiffs exist with injuries caused by hazardous substances who will not be compensated by the present common law tort system. In the absence of any such showing, the American Insurance Association opposes legislation that would so radically alter traditional common law remedies and would render uninsurable those who would be subject to the new liabilities.

The lack of any reliable scientific basis underlying these calls for legislative reform is admitted in the Report:

> "The discussion of legal issues relating to injuries from hazardous wastes must proceed in a setting of factual and scientific uncertainty because, at this time, in mid-1982, it is impossible to determine the number of persons who

> have been injured or who could be injured by exposure to such sites." (Report at 21, footnote omitted).

Study Group Member George C. Freeman, Jr. in a letter dated March 2, 1982 (included in Volume Two of the Report) noted:

> "We have very little reliable information about how much truly toxic wastes are out there and how many persons may or may not have been exposed to them in circumstances that have caused, or are likely to cause, death, serious bodily injury or disease.
>
> * * *
>
> If relatively few people have in fact been injured by (or even exposed to) toxics, the fact that few cases for damages have been successfully brought should not be regarded as evidence that the current law is inadequate or unfair." (Id.)

Premature public outcry is not unprecedented in the field of hazardous substance legislation. In 1980 the Environmental Protection Agency released a study purporting to show chromosome defects among residents near the Love Canal. Based in large part on this study, more than 700 people were evacuated from the area by the federal government; a public outcry was launched and CERCLA was passed later the same year. Subsequent reports, however, reveal that this study "was so poorly designed that it should never have been undertaken."* A subsequent study released on May 17, 1983, by the Centers for Disease Control found no increase in chromosomal abnormalities among Love Canal residents (including 17 such residents who had participated in the original study) when compared to a comparable control group.** This newest study also noted that other "[e]xtensive studies at the Love Canal dump site in Niagara Falls, New York, giving particular attention to reproductive abnormalities and to cancer, have thus far defined no firm evidence of increased disease incidence or mortality . . . ."***

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* Boffey, "Study of Love Canal Finds No Direct Link to Genetic Disease", New York Times, May 18, 1983, at A1, col. 6. No comparable control group was used in the original study.

** Centers for Disease Control, Brookhaven National Laboratory & Oak Ridge National Laboratory, A Study of Cytogenic Patterns in Persons Living Near the Love Canal (hereinafter "CDC Study"); see "Study of Love Canal Finds No Direct Link to Genetic Disease", New York Times, May 18, 1983, at A1, col. 6. See also Dionne, "U.S. Finds Love Canal Neighborhood Is Habitable," New York Times, July 15, 1982, at A1, col. 1.

*** CDC Study at 1. Similarly, there is no evidence that the tort system has failed in the oft-cited Environmental Law Institute study entitled Six Case Studies of Compensation for Toxic Substances Pollution: Alabama, California, Michigan, Missouri, New Jersey, and Texas (Senate Comm. on Environ. and

(Footnote continued)

Given this background, legislatures should be cautious and not rush headlong into enacting overbroad "reforms." For example, in September 1981 the State of California established a Hazardous Substance Compensation Account (Cal. Health & Safety Code §§ 25300-95) to provide compensation "for loss caused by a release, in California, of a hazardous substance" if the release was from an unknown source or an unknown liable party, or if the liable party could not be found, or was judgment proof. As of March 1983, not a single direct claim had been asserted against the fund.* Despite publicity concerning the lack of claims made against the fund,** at

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(Footnote *** continued from previous page)
Public Works, 96th Cong., 2d Sess., Serial No. 96-13, Comm. Print, June 1980) (hereinafter "Six Case Studies"). Of the six cases studied -- which six were chosen specifically because they were believed to demonstrate "substantiality of harm" and not on any random basis -- only two included any evidence of serious personal injuries due to chemical exposures, and certain injured persons received compensation in each of those two instances. This two-month study heavily reflected the personal views of the authors, but failed to afford those views an evidentiary support.

* See Hazardous Waste Management Council, Compensation: An Analysis of Hazardous Waste Facility Insurance Requirements and Alternatives 28 (January 1983).

** "California Cleanup Fund Has Money Begging," New York Times, Monday, March 21, 1983, at A12, col.6.

the present time less than a dozen claims have been filed.

In view of these facts, North Carolina has recently adopted a more cautious approach. The 1982 Annual Report and Special Reports of the North Carolina Governor's Waste Management Board, while recommending certain limited changes in the application of strict liability principles under the tort law system, rejected an administrative remedy or any presumption of causation. (_Id._ at 53-56). With respect to administrative remedies:

> "The Board concluded that it is premature at this time to assume that the volume or complexity of hazardous waste liability cases will be so great that the general court system cannot handle them adequately." (_Id._ at 56).

B. Proponents' Failure to Consider the Staggering Costs of Their Overinclusive Proposals

It would be disastrous to structure a radical new "solution" in such a way as to eradicate the traditional tort law requirements that a plaintiff show a defendant was at fault and caused the harm complained of.* Such a "solution" would, in effect, require the chemical and oil industries to pay the costs of cancers, tumors, genetic damage, joint pains, headaches and other diseases or symptoms every time a claim of "exposure" to toxic waste was lodged, even if (as would almost always be the case) the claimant's disease resulted from a combination of other causes such as normal environmental or genetic factors unrelated to exposure to toxic waste. (See further discussion of the specific legal principles below).

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* Moreover, any "solution" that abandons these elementary principles might have serious ramifications for tort law in other areas, which no one intended to "reform."

For example, consider the present cancer statistics - just one disease that _might_ result from exposure to hazardous substances. In 1983, an estimated 440,000 Americans will die of cancer, and an estimated 855,000 people will be diagnosed as having cancer.* Statistics show that more than 20% of the total deaths among Americans resulted from some form of cancer -- the second highest cause of death, after heart disease.** Yet, it has been estimated that approximately 2% (and no more than 5%) of cancer deaths are caused by air, water and food pollution in all its forms;* 1% or less may result specifically from exposure to pollutants found in hazardous waste sites.** However, the Study Group's proposal, echoed in many of the pending legislative proposals, would permit recovery by anyone who can claim non-occupational "exposure" to hazardous waste dumps and whose ailment cannot be demonstrated _not_ to have resulted from such exposure -- an impossible burden for a defendant to meet, as more fully discussed below. Such a skewed system would certainly involve many times the number of recoveries than would be warranted by the small percentage of diseases arguably "caused" by hazardous substances.

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* E. Silverberg & J. Lubera, _Cancer Statistics, 1983_ 10 (reprinted from _Ca-A Journal for Clinicians_, Vol. 33, No. 1, 1983 (American Cancer Society).

** Figures for 1978 (the last available year). _Cancer Statistics, 1983_, _supra_, at 11.

In light of these facts, the cost of the Study Group majority's overinclusive "solution" would be staggering. Just as with other types of compensation programs begun in the 1960s and early 1970s the costs can be expected to balloon to truly massive proportions. See "Toxic-Liability Bills Plant a Time Bomb of Entitlement Costs," The Wall Street Journal, Friday, June 3, 1983, p. 20, col. 3; Office of Management and Budget, Compensation for Toxic Substances Exposure (May 19, 1983). The experience under the federal Black Lung Benefits Act provides an apt warning:

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* _See_ R. Doll & R. Peto, _The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today_, 66 J. Nat'l Cancer Inst. 1192, 1245-50, 1256 (1981); Technology Assessment Board, 97th Cong., _Assessment of Technologies for Determining Cancer Risks from the Environment_ 91 (June 1981).

** Comments of Jim J. Tozzi at a conference on May 7, 1983 concerning Office of Management and Budget-sponsored Inter-Agency Study of Toxic Torts.

> "The Black Lung Act provides an example of what can happen when a compensation system is not well thought out. A General Accounting Office report issued in January 1982 found that 84 percent of claims were paid without adequate medical evidence.
>
> The result: In 1969, Congress estimated that the total program would cost between $40 million and $385 million. By 1972, costs to the federal government alone amounted to $33 million per month. In 1979, such costs amounted to $1.5 billion and reached $2 billion in 1982. The total cost of the program, including benefits paid directly by industry, is unknown."*

As a result, in 1981 Congress eliminated many of the presumptions of causation in the Black Lung Act because they had "led to an excessive number of claims and vastly greater expenditures than had been anticipated." (Report at 228). Black Lung Act costs are expected to level off or slightly decrease over the next few years.* Nonetheless, one critical presumption remains -- a rebuttable presumption that certain identified diseases were work-related if the employee could show 10 years of employment. Consequently, costs will not decline very quickly, and in fact continued to rise in 1982.**

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* J.B. Browning (Chemical Manufacturers Association), Public Compensation: A Chemical Industry Perspective 9 (April 5, 1983); see Problems Relating to the Insolvency of the Black Lung Disability Trust Fund: Hearings Before the Subcomm. on Oversight of the Comm. on Ways and Means of the House of Representatives, 97th Cong., 1st Sess. 2-15, 159-88 (1981).

The Black Lung Act is very limited in scope; costs for a sweeping hazardous substance program would be enormous, given the broad presumptions, unlimited awards and huge administrative costs inherent in the pending proposals.

To obtain a rough concept of this cost, assume that the number of cases that would be filed for all the types of diseases which plaintiffs may claim could result from exposure to hazardous substances -- cancer, respiratory ailments, tumors, genetic defects, coronary disease, etc. -- might be so low as one-fourth of the diagnosed annual number of cases of cancer alone. Rounding downwards, such an assumption yields an incidence of approximately 200,000 claims filed per year.*

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* Office of Management and Budget, Compensation for Toxic Substances Exposure, Chart 2 (May 19, 1983).

** Id. Further, the use of a presumption that a disease is work-related in the context of the Black Lung Act or workers' compensation statutes is inherently a limited one; the list of employees on the roster is finite and easily ascertainable. Such a presumption relating to hazardous substance exposure is open-ended, uncontrollable and potentially limitless in scope.

At this conservatively estimated rate of claims, and given the same average recovery in Tier One that is estimated for current workers' compensation cancer cases of $130,000 per award,** the Study Group's program would result in an annual cost of $26 billion in Tier One claims payments alone.*** Assuming allocated loss adjustment expenses (i.e., defense legal fees, etc.) of approximately 10% added to the total cost of these cases,* the annual bill in Tier One alone might be on the order of approximately $28 billion.

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* In view of the claims of the Environmental Protection Agency ("EPA") and legislative proponents, this number of cases is entirely within the bounds of possibility. EPA has claimed there are over 80,000 sites in the nation with contaminated surface impoundments, 90% of which "are believed to pose at least a potential threat of ground water contamination." Technology Assessment Board, 98th Cong., Technologies and Management Strategies for Hazardous Waste Control 5 (March 1983). Under the Study Group majority's approach, residents around these sites showing "exposure" may be able to recover, even where such "exposure" did not actually cause their disease or injury.

** Source: National Council on Compensation Insurance. See Technical Appendix A.

*** I.e., 200,000 claims times $130,000 per award. To put this annual cost in perspective, it represents more than twice the total workers' compensation loss and loss expense incurred by private carriers and state funds in 1981 (almost $12 billion), and would (Footnote continued)

Additional awards to plaintiffs in Tier Two would add massive costs such as pain and suffering awards, attorneys' fees and litigation expenses. To the extent this estimate is conservative in its assumptions, it follows that the actual cost of the program will run even higher.

To require that this extraordinary cost -- far in excess of any proven level of injuries caused -- be shouldered by a few American industries would seem very

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(Footnote *** continued from previous page) add 7.0% to the inflation rate of chemical and petroleum product prices (given 1982 sales of $371 billion), along with a similar effect on the prices of all other goods and services that incorporate these products, if an industry tax were passed through to consumers.

* The National Council on Compensation Insurance figures showed allocated loss adjustment expenses of 10%, but it is reasonable to expect that for cases proceeding on through Tier Two litigation, such expenses would be three to four times higher. The Study Group majority's proposal provides no abatement in these transaction costs, since plenary court lawsuits are not barred; indeed, the Study Group's proposal merely adds the administrative costs to the present system.

unwise, especially in the face of foreign competition which would not be burdened by similar costs. See Letter dated March 10, 1982 by Study Group Member George C. Freeman, Jr. (included in Volume 2 of the Report). In addition, as the cost of complying with the law rises, the incentive to engage in illicit, secret dumping or to employ other illegal means of waste disposal rises.* Further, to assume that such an extraordinarily costly scheme is insurable, or worse yet to dismiss the issue with the Study Group majority's assertion that:

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* Concern has already been expressed that organized crime may be involved in the garbage and waste disposal industry. See, e.g., "Corruption Cited in Waste Business," New York Times, Sunday, May 1, 1983, at A52, col. 4; "Attorney Denies Dumping, Mob Connection," UPI Regional News story, Friday, April 29, 1983; "Poison Dump Stirs Alarm in Poconos," New York Times, Sunday, April 24, 1983, at A20, col. 1. As the New York Times reported last year in connection with the conviction of Russell W. Mahler:

> "New York can handle only 60 percent of the 1.4 million tons of industrial waste that companies in the state produce annually, and the pressure to get rid of some of it by illegal means is tremendous, according to state officials. 'If you pay the price, you can dispose of hazardous waste, but the price can be prohibitive,' said Jeremiah B. McKenna, counsel to the State Senate's Select Committee on Crime."

Chavez, "Toxic Waste Entrepreneur," New York Times, Thursday, May 27, 1982, at D1, col. 1.

> "insurability is not the sole or even the dominant factor that ought to determine whom the law protects and how,"*

is to court disaster. If no insurance were written to cover these risks, or if industry is unable to afford the premiums, there would be no effective means of spreading the enormous cost of the proposed system while at the same time offering the incentives to careful conduct that are traditionally provided by variable commercial insurance rates. Without insurance, it is unclear that our vital industries would even be able to meet this level of risk without jeopardizing their very existence. Unfortunately, this is precisely the perilous course which the Study Group majority, and proposed state and federal legislation modelled on its recommendations, would have us adopt.

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* Report at 185. See Letter dated May 17, 1982 from George C. Freeman, Jr. (included in Volume Two of the Report), which questioned the availability of insurance to cover the Study Group's proposals.

C. Proponents' Failure to Consider the Ramifications of the Specifics of Their Proposals

1. The Presumption of Causation

The heart of both the Study Group's Report and many of the legislative proposals introduced is a presumption that diseases contracted by an "exposed" person were caused by that exposure. But in fact, such diseases have a multiplicity of possible causes, of which hazardous substance exposure may be one of the least significant when compared with diet and certain habits (such as smoking).* The Study Group majority's attempt to legislate science, without regard to the actual facts that are available, is echoed in the vast majority of the bills that have been submitted to state and federal legislatures.**

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* *See* R. Doll & R. Peto, *The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today*, 66 J. Nat'l Cancer Inst. (1981); Technology Assessment Board, 97th Cong., *Assessment of Technologies for Determining Cancer Risks from the Environment* (June 1981).

** *See*, *e.g.*, Massachusetts H. 5179, § 7(a); S.917, § 5(c)(3); S.946, § 3(b); H.R. 2330, § 102(a); *cf*. Minn. Stat. § 115B.07. Indeed, Senator Mitchell's proposal in S.946, after providing a "rebuttable presumption", goes on to state:

> "Evidence as to the physical weakness or personal habits of the individual on whom

(Footnote continued)

It may be impossible for a defendant to prove lack of causation from an exposure, given the long latency period for many toxic waste related diseases. Plaintiff, and not defendant, is in possession of all the facts concerning the onset of disease and the symptoms involved, personal diet and habits that might be the cause of the disease, and all background information concerning plaintiff's history and exposure to this or other hazardous substances. None of this information is public, unlike much of the information about hazardous substances in dump sites collected by the federal government under CERCLA. See, e.g., 42 U.S.C. § 9603(c).

Given ordinary human sympathies, a burden to disprove causation would be much harder for a defendant to sustain than it would be for a claimant to prove causation affirmatively. Thus, industry members would become virtual guarantors for the costs of all diseases and

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(Footnote ** continued from previous page)
the claim is based shall not be used to rebut the presumption created by this paragraph."

Thus, for example, a person who contracted lung cancer after an exposure to hazardous substances would presumably recover under this formulation on the basis of the exposure, no matter how heavy a smoker he is. Smoking, however, might be many times the more significant contributory factor.

genetic defects occurring in an indeterminate population that lives, works, and vacations in, and perhaps even passes through, the vicinity of every hazardous waste site, without any real regard for whether the harms claimed were actually caused by the hazardous substances, or whether they resulted from or were primarily contributed to by other causes.

Insurance industry representatives and members of the Study Group itself also expressed concern that, despite a lack of any recommendation so providing, the presumption of causation available to claimants in a Tier One administrative proceeding would likely be imported into Tier Two by the courts,* resulting in full recoveries (including pain and suffering damages) to a large number of plaintiffs without proof of causation. This concern is heightened by examples such as the Minnesota Environmental Response and Liability Act (the "Minnesota Act"), passed in May 1983, which provides in Minn. Stat. § 115B.07 for restrictions on directed verdicts -- a provision that amounts in practice to a presumption.* This de facto presumption is by its terms made applicable to actions brought under the Minnesota Act "or any other law to recover damages for death, personal injury, or disease arising out of the release of a hazardous substance . . . ." Id. (emphasis added).

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* Report at 175, 264-65; Comments of Study Group Member Charles D. Breitel (Report at 300); Comments of Study Group Member George Clemon Freeman, Jr. (Report at 318).

2. Liability Without Regard to the Defendant's Fault or Lack of Fault

The Study Group majority recommended that all consideration of fault be eliminated entirely in both Tier One and Tier Two proceedings.** Even the attenuated fault concepts embodied in the "strict liability" provisions of the Restatement (Second) of Torts § 519-52 would be disregarded by the Study Group majority.

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* Minn. Stat. § 115B.07 prohibits a directed verdict for the defendant on the issue of causation if plaintiff "produces evidence sufficient to enable a reasonable person to find that" (a) the defendant is responsible for the release of the hazardous substance; (b) the plaintiff was exposed; (c) "the release could reasonably have resulted" in the amount and duration of plaintiff's exposure; and (d) "the death, injury, or disease suffered by the plaintiff is caused or significantly contributed to by exposure to the hazardous substance in an amount and duration experienced by the plaintiff." The statute continues:

> "Evidence to a reasonable medical certainty that exposure to the hazardous substance caused or significantly contributed to the death, injury or disease is not required for the question of causation to be submitted to the trier of fact." Id. (Emphasis added).

** Report at 196, 209, 260, 265-66.

The Second Restatement applies a strict liability standard to abnormally dangerous activities. At the same time, it:

> "cautions against defining a type of activity as 'abnormally dangerous' in and of itself, however, and advocates considering the activity in light of surrounding circumstances on the facts of each case. This, in essence, shifts consideration from the nature of the activity to the nature and extent of the risk."

Clark-Aiken Co. v. Cromwell-Wright Co., 323 N.E.2d 876, 887 (Mass. 1975) (stated with respect to the Tentative Draft of the Second Restatement). Section 520 of the Second Restatement provides:

> "In determining whether an activity is abnormally dangerous [and thus gives rise to strict liability], the following factors are to be considered:
>
> (a) Whether the activity involves a high degree of risk of some harm to the person, land or chattels of others;
>
> (b) Whether the gravity of the harm which may result from it is likely to be great;
>
> (c) Whether the risk cannot be eliminated by the exercise of reasonable care;
>
> (d) Whether the activity is not a matter of common usage;

(e) Whether the activity is inappropriate to the place where it is carried on; and

(f) The value of the activity to the community."*

Thus, the Second Restatement requires a court to consider whether an activity is necessary to society and is being carried on in an appropriate location, among other things, before finding it to be "abnormally dangerous" and imposing "strict liability" for injuries that result from such an activity.

The Study Group majority, however, would impose strict liability without first considering all the circumstances. The Study Group majority stated:

> "Formulations of strict liability that balance the hazard of the activity against its utility and its appropriateness to the locale import considerations of negligence that entail significant barriers to the recovery of damages for injuries from exposure to hazardous wastes. To remove these barriers, the application of a theory of strict liability is needed that focuses on the nature of the hazardous waste activity itself, and on the magnitude of the risk." (Report at 260).

The trend in tort law today is not toward absolute liability such as was recommended by the Study Group majority, but rather toward more limited "strict liability" concepts based on the Second Restatement.* Today the overwhelming majority of states that have considered Section 520 of the Second Restatement have utilized its factors in determining cases.**

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* Under the case law, disposal of hazardous waste has not uniformly been found to be abnormally or inherently dangerous. See Ewell v. Petro Processors of Louisiana, 364 So.2d 604 (La. Ct. App. 1978).

This acceptance of the Second Restatement's factors is based upon sound considerations. These same factors are, in part, the criteria used by underwriters to determine insurance premium rates for a particular insured. Absolute liability, without any regard to proper conduct, on the other hand, leaves no room for consideration of fault, and provides no incentive for companies to behave well in handling their hazardous substances. An absolute liability system would penalize necessary industries conducted in a reasonable manner just as heavily as marginally useful, irresponsibly conducted activities; careful conduct would become irrelevant. The balanced approach of the Second Restatement should not now be abandoned in favor of an ill-considered stampede to an absolute liability standard.

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* *See*, *e.g.*, *Dye v. Burdick*, 553 S.W.2d 833, 839-40 (Ark. 1977).

** Report at 120-24; *see*, *e.g.*, *Cropper v. Rego Distribution Center, Inc.*, 542 F. Supp. 1142, 1150 (D. Del. 1982); *City of Northglenn v. Chevron U.S.A., Inc.*, 519 F. Supp. 515 (D. Colo. 1981); *Indiana Harbor Belt Railroad Co. v. American Cyanamid Co.*, 517 F. Supp. 314, 319 (N.D. Ill. 1981); *Harper v. Regency Development Co.*, 399 So. 2d 248 (Ala. 1981); *Cities Service Co. v. State*, 312 So. 2d 799 (Fla. Dist. Ct. App. 1975); *National Steel Service Center, Inc. v. Gibbons*, 319 N.W.2d 269 (Iowa 1982); *John T. Arnold Associates, Inc. v. City of Wichita*, 5 Kan. App. 2d 301, 615 P.2d 814 (1980); *Yommer v. McKenzie*, 255 Md. 220, 257 A.2d 138 (1969); *Locke v. Mach*, 115 Mich. App. 191, 320 N.W.2d 70 (1982); *Ferguson v. Northern States Power Co.*, 307 Minn. 26, 239 N.W.2d 190 (1976); *Otero v. Burgess*, 84 N.M. 575, 505 P.2d 1251, 1255 (Ct. App., 1973); *Doundoulakis v. Town of Hempstead*, 42 N.Y.2d 440, 368 N.E.2d 24, 398 N.Y.S.2d 401 (1977); *Wirth v. Mayrath Industries, Inc.*, 278 N.W.2d 789 (N.D. 1979); *McLane v. Northwest Natural Gas Co.*, 255 Or. 324, 467 P.2d 635 (1970); *Fleege v. Cimpl*, 305 N.W.2d 409 (S.D. 1981); *Pacific Northwest Bell Tel. Co. v. Port of Seattle*, 80 Wash. 2d 59, 491 P.2d 1037 (1971); *Peneschi v. National Steel Corp.*, 295 S.E.2d 1 (W.Va. 1982). *But see* *New Jersey v. Ventron Corp.*, 182 N.J. Super. 210, 440 A.2d 455 (App. Div. 1981).

3. Imposition of Excessive Liability on Non-Responsible Defendants

The use of absolute liability without regard to fault is particularly far-reaching in light of the Study Group majority's recommendation that defendants be jointly and severally liable in a court proceeding. (Report at 257-58). Thus, a very prudent manufacturer who merely had the misfortune to place his waste in the same site as another, who acted intentionally or even criminally, could be held liable for the entire judgment. If the negligent enterprise cannot be found, or is judgment proof, the prudent manufacturer will be forced to bear the entire liability without recourse despite careful and proper behavior.

This situation is exacerbated further by the presumption proposed by the Study Group majority. Under that presumption, for example, a claimant might simply prove his exposure to "Substance X" and that a particular manufacturer deposited "Substance X" in the dump site the claimant asserts is the source of the contamination affecting him. In a subrogated liability proceeding against the manufacturer, liability would be found unless the manufacturer meets a burden of showing that somebody else's "Substance X" was the source of the contamination. If there was a lack of historical evidence, as may often be the case, such a burden could not be maintained. Thus, a perfectly prudent, careful and non-responsible party who dealt in "Substance X" might ultimately bear a liability for harm it never caused, but which was instead caused by the irresponsible activities of another person. The unpredictability of this liability, coupled with its potentially huge amount, would make it extremely difficult to insure.

4. The Initial Funding Tax and Further Removal of Incentives for Careful Handling of Hazardous Substances

The proposal that a compensation program should be initially funded through taxes on industry* would place an impossibly high burden on domestic industries already faced with stiff foreign competition unburdened by such expenses. See Comments of Study Group Member George Clemon Freeman, Jr. (Report at 311-14). Funding by such a generic tax burden prevents an enterprise from reducing its cost by prudent behavior.

> "The Study Group claimed that the costs of injuries should be carried by the industry or enterprise that creates them 'because efforts to reduce the cost will result in greater care, more safety and protection of the public' . . . [see Report at 248]. Though this generalization is often true, it is an inappropriate justification of the proposed tax.
>
> The Study Group's rationale is wrong on at least two counts. First, a tax on industry output provides little or no incentive for any single enterprise to reduce its liability. A firm does not reduce its tax burden by taking due care to avoid hazardous waste damages; therefore, such a funding mechanism simply does not act as a deterrent. Secondly, the fund is only applied to exposures that have either happened in the past or for which there is no responsible party; obviously, it is impossible to deter this type of exposure and, consequently, the tax provides no appropriate incentives. . . ."**

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* Report at 245.

(Footnote(s) ** will appear on following pages)

Such a tax would eliminate the use of commercial insurance, thus further removing any incentive to handle hazardous substances properly, and eliminating a valuable social mechanism of industrial self-regulation.

Insurance is a method of assessing and spreading risk, which has proven to be efficient in determining proportionate potential liability. Each insurer, in competition with all other insurers, puts itself at risk based upon its determination of an insured's potential liability. Using underwriting criteria that include not only statistical data but also loss control and loss prevention expertise, an insurer makes a determination as to which facilities or activities it will insure. In so doing, insureds that may treat or deal with the same quantity and type of toxic substances may be charged different premiums for their coverages because of judgments made by underwriters that reflect tangible and intangible qualities ranging from record keeping, to attitude toward loss control, to the level of personnel dealing with loss control and prevention.

** National Economic Research Associates, Inc., The Role of Economics in Tort Law Reform, (Draft Memorandum by F. Dunbar), at 10-11 (November 8, 1982).

Myriad factors, together with the personal experience of the underwriters, will determine the effectiveness of the underwriting process. This process is totally unlike a tax system, which treats the facilities disposing of the same or similar quantity or type of toxic substance in an identical manner, as though they were identical. The insurance mechanism, operating in conjunction with a sound tort system, provides carefully honed financial incentives to proper behavior and injury prevention, which can never be duplicated by a tax-based system.

5. Retroactivity

> "[I]f regulation under RCRA is effective, there is hope that there will be few cases of injury from hazardous waste in years to come. The recommendation for the compensation remedy is based on the possibility and concern that past hazardous waste disposal practices may have created a backlog of injuries and exposures that the legal system will need to clean up." (Report at 195).

With these words, the Study Group majority focused its attention on the issue of retroactive liability, relying heavily upon the speculative "possibility" that past practices "may have created" an as yet unidentifiable "backlog" of cases for the legal system to "clean up."

The pitfalls for the insurance industry of creating new, massive, retroactive liability for past practices are well known. The imposition on insurers of new obligations beyond those contemplated by the parties to the insurance contracts could be devastating to the entire insurance industry. Insurers have collected their premiums and set aside reserves based upon the law as the parties understood it at the time of contract. It is neither just nor sound practice to expand these obligations by legislative fiat. Further, as to future underwriting, the idea that such a massive liability might be created at any future date would certainly chill the most courageous underwriter. This type of sudden liability expansion is more likely to deter the growth of insurance markets than it is to provide a financially sound system of injury compensation; it would therefore defeat its own objectives.

The Study Group majority itself was aware of the problem that an overtly retroactive liability scheme would present; it attempted to circumvent retroactivity to some extent. While its recommended scheme would allow a number of retroactive claims by claimants* against the

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* Claims could be filed within three years of discovery, whether discovery occurred before or after passage of the implementing legislation. (Report at 209). Further, since discovery of injury might take place many years after the initial exposure to

(Footnote continued)

administrative agency (Report at 209-10), it would permit subrogated claims against industry members only for injuries or damages caused by exposure to "wastes disposed of, transported or spilled after the adoption of this proposal. . . ." (Report at 246). However, this is not an effective remedy to the problem of retroactive liability. Industry would end up footing the bill retroactively anyway because, under the Study Group majority's recommendations, unsubrogated claims would be paid through a tax on industry. In all fairness, if society decides to pay such retroactive costs, society in general should foot the bill, rather than attempting to load it unfairly on a small segment of its members.

Further, as we have seen, "toxic torts" may often involve diseases that take as long as 20 or 30 years to develop; claimants may have been exposed to a combination of substances deposited by a number of different companies both before and after implementing legislation is passed. It might be virtually impossible to sort out injury resulting from substances deposited both

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(Footnote * continued from previous page)
toxic waste, many claims -- and certainly the most catastrophic ones -- would in effect be "retroactive."

before and after the cut-off date, or from substances deposited by different companies. Subrogation suits would thus become an expensive nightmare of claims and cross-claims.* Alternatively, legislative proposals such as the Minnesota Act have permitted suits retroactively unless there is proof that "the substance was placed . . . in or on the facility wholly before" a cutoff date -- in Minnesota the date is set at January 1, 1980.** But for activities that continued after the cutoff date, the Minnesota Act provides extensive retroactive liability as a practical matter, and also fails to solve the problem of cross-claims, thus creating the worst possible "solution."

6. Add-On Remedies

> "[F]ree access to the plenary tort action after the claimant had already had a compensation award on Tier One would be prohibitively expensive from the insurance point of view. A system which called for a binding election between the tort remedy and the compensation remedy

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* The continued or increased use of joint and several liability of defendants in Tier Two lawsuits would also presumably result in a tangle of cross-claims for contribution in the courts.

** Minn. Stat. § 115B.06(1)(b). The Minnesota Act also provides an additional defense if the substance was deposited prior to January 1, 1973 and defendant's activity was not an "abnormally dangerous" one. Minn. Stat. § 115B.06.

> would be more readily insurable." (View Expressed to the Study Group, Report at 174).

Administrative remedies, in order to be practical, have traditionally involved a trade-off: relaxed standards of proof and easier awards must be balanced by a limitation on the amount recovered. Despite its claims that Tier One is modelled on workers' compensation (Report at 195), the traditional quid pro quo associated with such a compensation system has not been adopted by the Study Group majority's recommendation. Instead, the Study Group majority:

> "adopted the worst of both worlds: an administrative compensation system with liberal evidentiary standards and generous benefits, as well as continued availability of traditional tort law." Office of Management and Budget, Compensation for Toxic Substances Exposure 13 (May 19, 1983).

By providing an administrative recovery with reduced evidentiary burdens for plaintiffs, while doing nothing to provide either a bar or a binding election against further tort claims in court, the Report has gone far beyond the workers' compensation model it claims to follow. Since plaintiffs will typically be seeking large "pain and suffering" awards, which they would not receive in Tier One, the incentive to "go for it" in court would be high. A plaintiff could use his Tier One administrative recovery to fund a subsequent lawsuit in court, thus (in effect) causing the likely defendants to pay the cost of suits against themselves.

The Study Group majority's sole attempt to limit the use of both Tiers by plaintiffs was to permit (but not require) a judge to impose court and expert witness costs on the plaintiff if the plaintiff fails to recover at least 25% more in court than he did in the Tier One administrative proceeding.*

> "Such an assessment, to be made at the discretion of the judge in the plenary court proceeding, is to take into account whether the plaintiff's action was brought in good faith, with a view of avoiding frivolous and duplicatory proceedings." (Report at 202).

In reality, it is doubtful that such costs would be assessed with any frequency. Plaintiffs will usually be individuals of limited means suing defendants who are large corporations; as a practical matter, it would be a rare case in which a judge would find plaintiff's pleading sufficiently frivolous to warrant the imposition of a cost sanction.

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* A Joint Statement of dissenting Study Group members proposed that once an administrative award was given, claimant should be required to submit to a hearing in court to determine if a lawsuit is warranted, before being allowed to proceed with the lawsuit itself. (Report at 286). Even that system, however, involving yet another hearing, would proliferate legal proceedings and transaction costs. In this regard, consider the view expressed by Study Group Member Jeffrey O'Connell (Report at 354-55) that the three-proceeding structure proposed by the Study Group (i.e., administrative claim, subrogated claim, claimant's lawsuit) was excessive. Mr. O'Connell nonetheless joined in the Joint Statement.

With much to gain and virtually nothing to lose, it is unlikely that many plaintiffs would be deterred from going forward in both Tiers. Thus the Study Group majority's proposal would simply increase the administrative costs of the system overall by adding the Tier One proceedings and administrative costs to the present proceedings and costs of court litigation continued in Tier Two. Such an expensive, cumbersome system lacks any logical support.

V. CONCLUSION

The recommendations of the Study Group majority, and the legislative proposals based on those recommendations, are both unfair and unwise. It makes little sense to rush into such a radical "solution," involving so many pitfalls and draw-backs, as well as staggering costs well in excess of the harm caused. Instead, concerted effort should be devoted to the prevention of such problems in the future, by rigorous enforcement of existing laws, such as the Clean Air Act, Clear Water Act, and RCRA, and by retention of the incentives for proper conduct inherent in the tort system.

In addition, funding should be increased for professionally-conducted studies to determine in a rigorous way what causal links exist between exposure to hazardous substances and the harms claimed to result from such exposure. Once science has established what causal connections exist, there will be little need for Congress or state legislatures artificially to ordain such connections by legislative fiat, or to require compensatory payments from those who are not at fault and not the responsible parties. The American Insurance Association suggests that dispassionate scientific inquiry, coupled with traditional tort law principles assigning liability to those at fault who caused the harm, is the best and most rational means of achieving a just, economic, insurable and workable compensation system for injuries caused by hazardous waste.

TECHNICAL APPENDIX A

Estimation of Average Claim Costs for Workers' Compensation Cancer Cases

The National Council on Compensation Insurance (NCCI) has estimated the current average cost of a worker's compensation cancer claim to be $130,000 based on data for claims which have already been filed, as explained below.

Worker Benefits

A review of reported workers' compensation cancer cases showed that the typical claimant was a fifty year old male who lived for three years after he became disabled and incurred medical expenses approximately equal to his wage loss benefits during that period. The costs of these claims were calculated using current wage rates so that no adjustment for inflation was made.

Survivor Benefits

Existing rules allow for the worker's widow to continue receiving benefits after his death for a duration of up to ten years unless she dies or remarries prior to that time, whereupon benefits cease. Widow's benefits were limited according to these rules using standard mortality tables and the results of an NCCI remarriage study which provided distributions, by age, of the number of women expected to die or remarry in each subsequent year.

Mr. **Levitas**. Ms. Birnbaum, first let me say without objection we would like to receive from you the listing of the types of cases or settlements that you are aware of. That would help us.

Ms. **Birnbaum**. Yes, sir.

Mr. **Levitas**. We heard the other day from at least one witness, who said we ought not to create a system of victims' compensation because there isn't enough scientific evidence to prove the case that people are injured by hazardous waste sites. You make the argument we ought not to establish a toxic tort system, statutory victims' compensation system because the tort system itself, the common law system, has not yet had a chance to prove itself to work.

My question to you would be—because you are approaching it from a different angle than Dr. Karrh did the other day—is that where we see a system, or a circumstance that has evolved in a relatively short period of time in terms of public awareness, and in this case the hazardous waste sites and the relationship to personal illness and ailments resulting therefrom, why should we wait 5 years to create a system that will address the issue, because one of the necessary hypotheses of the common law approach is that it has to be based upon precedent and unless the first case decided establishes a precedent from that time forward then you have literally hundreds or thousands of individuals who are not getting treated the way that they would at the end of the line.

Yesterday I mentioned the case of *Buick* v. *McPherson*. How many people went uncompensated before 1933 or whenever it was because they couldn't prove privity? That may be fine, but if we have the opportunity to let those people go uncompensated, shouldn't we avail ourselves of that?

At that point, I am going to give you plenty of time to think about your answer because those are the second bells of a live quorum which is taking place on the floor and we are also going to be asked to leave this room because they need it for another meeting at a later time this afternoon.

So what I am going to suggest—I said there would be a luncheon break—we will recess now until 20 minutes until 2 and reconvene in room 2253 on the second floor of this building and we will start off with your answer to that last question.

Ms. **Birnbaum**. Thank you for the opportunity to reflect on it.

Mr. **Levitas**. Thank you.

[Whereupon, at 12:35 p.m., the subcommittee was recessed, to reconvene at 1:40 p.m., in room 2253, on the same day.]

Mr. **Levitas**. The subcommittee will please come to order.

While Mr. Cheek has not yet returned, I think, Ms. Birnbaum, you have the floor in any event.

Ms. **Birnbaum**. Would you like to restate the question, Mr. Congressman?

Mr. **Levitas**. No.

Ms. **Birnbaum**. Well, I think the gist of the question was, if we wait for the common law system, maybe some people will not be compensated while the common law system is developing. The response to that is that the common law system is already in place. It is not in a developing stage with repect to doctrines and standards of liability. They are in existence now. Over the last 10 years, especially as a result of the asbestos litigation, a whole body of law has been developed which is being applied in all types of toxic substance cases.

I think that, when I was talking about waiting and seeing, I meant that we should wait and see whether the common law will keep expanding to meet new situations, which in the past it has done. The precedents are in place. The concept of strict liability, whether through product liability law or through the restatement of torts principles applicable to abnormally dangerous activities, is in place. I believe there is no need to wait for precedents. There are some questions in the relatively new context of hazardous waste litigation that might remain unanswered in a particular lawsuit but the courts are dealing with these, and that has always been the process of the tort system and common law development.

I think it would be something of a mistake not to allow the courts in the first instance to try to handle these problems as well.

Mr. Levitas. Let me ask you this. Based on your experience as an attorney in toxic tort cases, you made reference in answer to Mr. Molinari's question of the situations in which claimants or plaintiffs have recovered. You are going to furnish us additional information.

Let me turn that around. Tell us about the cases in which the claimants have failed or been denied compensation, and why.

Ms. Birnbaum. I think we can look at the asbestos cases, which is a category of toxic torts in which there has been much litigation. In many of the cases where the plaintiff does not recover, it is because of the insufficiency of the evidence as the issue of causation. In other words, the plaintiff has failed to establish to the satisfaction of the jury either that he has the illness he is alleging he has, or that he has been exposed to the particular toxic substance alleged to have caused his injury.

So in most instances, after a jury trial in the asbestos cases, which I think we can use as some sample of what has been happening. The plaintiff who does not recover has failed to establish causation: that is, failure to establish that in fact there is an injury that is associated with any degree of reasonable medical certainty with the particular toxic substance involved, or that the plaintiff does not have the injury that is allegedly associated, on a statistical basis, with the toxic substance. And it is in those two areas—general and individual causation that I think, if we reviewed the cases, we would find that most of the plaintiffs have failed.

Mr. Levitas. We have heard a lot of testimony over the last several weeks by persons who are advocating changes in the tort system that suggest a shifting burden of proof, or a shifting requirement to go forward with evidence, or a presumption under certain circumstances. This morning we heard Ms. Hall suggest three criteria for threshold of shifting, of either going forward or burden of proof, or as a requirement to raise the presumption.

I think the first was a showing of exposure that is reasonably related to the chemical or toxic substances involved. A scientific link between the chemical and the injury or ailment that the person complains of. And finally, the injury itself. Those were her three criteria.

Would you address yourself to the question of shifting burdens, presumptions, thresholds, and so forth?

Ms. **Birnbaum**. Thank you for the opportunity to do that. I think that if a plaintiff proves those three elements, the plaintiff doesn't need the burden to shift. The plaintiff has met the burden of proof at least as it is spelled out in most courts today. So there is no need to shift the burden. In fact, as a practical matter, the burden shifts to a defendant in a courtroom in any event if the plaintiff is able to establish the elements you describe.

Now, I think what you might have been suggesting is that the burden of all of those elements should be lessened so that the plaintiff could meet the burden and then a presumption would arise. That is, the plaintiff would have met his or her burden by establishing exposure over a sufficiently long period of time to have gotten the illness; a scientific link, that is, an expert is going to be able to testify with some degree of medical certainty that this injury may be caused by exposure to this substance and that the person is injured.

That is not very different from what is happening in most courtrooms today. That is what the plaintiff has to in effect prove by a fair preponderance of the evidence. In my opinion in most of the cases I have seen, the plaintiff has, in cases have established causation or in cases where causation can be statistically shown, or there is a statistical association, the plaintiff has not had a great burden at least after the initial cases were brought, in establishing that a product or exposure to a substance has caused the plaintiff's injury.

I think what we are hearing a lot about now are the newer cases where in the first instance the proofs are uncertain. Even in these cases, however, juries do what their job is—that is they sort out the facts and try to come down with a reasoned determination after listening to the experts. We are dealing in areas where there is not a great deal of established scientific fact. This raises the whole issue, I think, of whether there should be presumptions in the first place as to causation. It seems to me that the tort system, where one group of experts attempt to spell out the plaintiff's theory, and another group spell out the defense theory, and then the jury or trier of fact attempts to sort all this evidence out, makes more sense than a presumption.

She is suggesting really is what in reality in most instances we have in the courtroom today. The plaintiff has the burden. Once the plaintiff meets that burden, then the plaintiff goes to the jury. If the defendant does not rebut it, even though the jury may not be told this is a presumption in real life, that is what occurs.

Mr. **Levitas**. What about the other point Ms. Hall made about the fact that a great deal of the evidence in cases of this sort are of a statistical or epidemiological nature? What is your view about dealing with the questions of admissibility of that type of evidence?

Ms. **Birnbaum**. I don't have the statistics on it, but what we are seeing more and more is that courts are permitting that type of evidence in, I think as you pointed out, through expert opinion. The expert gives an opinion based on all that the expert has read or experienced. Part of that opinion could be based on epidemiological studies, animal tests, pathological tests. All of these are things that the expert, when he testifies, depends upon in supporting his opinion.

Now, the problem in some cases is that some of this evidence is so speculative that it may be stricken, or not be given a great deal of weight. But then again, perhaps it shouldn't be given alot of weight because of its highly speculative nature.

In Federal courts and many State courts, medical articles and other scientifice articles that the expert relies on are admitted into evidence for the truth of what they say. In the asbestos cases, for example, all of this data gets put in evidence through the plaintiff's experts. In the future courts will have more and more of that kind of evidence put before them, and are going to deal with that because this is the way cases get proved.

Mr. LEVITAS. I believe that is the point I was getting to, using the expert.

Mr. Cheek.

Mr. CHEEK. I apologize, Mr. Chairman, for being late. It is very hard to get lunch outside of the building.

Mr. LEVITAS. Do you want to hear my sad story?

Mr. CHEEK. The two questions you raise are very closely interrelated. Many of the bills that have been introduced so far would allow virtually any kind of statistical evidence to be put into the record and would give that evidence the status of a presumption in favor of the claimant.

The problem really lies in giving what Professor Birnbaum calls highly speculative evidence the status of a presumption. I think it would be useful to get on the record some discussion of the problems that statistical evidence presents.

First of all, in the context of long latency diseases, it is just not medically possible, except in a handful of cancers, to assign a single cause to any of those diseases. And proof of causation in such cases, therefore, becomes necessarily a statistical exercise as to which of the multitude of possible causes is the most probable among the causes.

That brings to mind the old adage that "statistics don't lie, but liars use statistics." As you have seen in other contexts, I am sure, statistics can be extremely misleading. But in most other contexts, absent a presumption, as Sheila points out, the misleading effects of statistics can be offset by either expert testimony on the other side or empirical proof. But that is really not possible in these hazardous substances injury claims. If you load up the statistical proof with presumptive weight, it becomes almost impossible to create any kind of rebuttal to that.

Abuses are possible in each of the three major categories of tests commonly used to establish associations between certain substances and the onset of certain diseases—epidemiological studies, animal bioassays, and bacteria and mammalian cells in tissue culture.

First, epidemiology. Although every human carcinogen we know of has been identified by means of epidemiology, it is very expensive. Epidemiology's expense and relative statistical insensitivity are complicated by cancer's long latency, which requires the studied population to review decades of personal habits and occupational and medicinal exposures, and to estimate the dose levels of suspected causative agents.

Epidemiology is further complicated by multiple exposures and possible interaction between suspected agents like smoking and asbestos, for example.

Finally, epidemiology is helpless to deal with the countless new chemicals which are put on the market each year, to which very few humans have been exposed. But the fact remains that epidemiology is the only tool that we have that can directly identify human risk factors and the only tool that does not have fundamental problems of interpretation.

Turning now to the second major tool, animal bioassays, they are subject first to the criticism that they are really just surrogates for human experience and therefore can't tell us with any certainty that a given substance even can cause human cancers, much less that it in fact does cause human cancer.

More importantly, animal bioassays are subject to dosage abuse so severe that it is possible, for example, to establish that such common substances as sugar, pepper, eggs and vitamin D can cause cancer in animals. In fact, there are now instances in the scientific literature where epidemiology has been unable to confirm a cancer risk suggested by rodent tests. Examples include saccharin, hair dyes and DDT.

Given the potential for abuse in such tests we would think that if you were looking at the use of such evidence in a victim compensation measure, you would want to be very sure to prescribe the protocols that would have to be met in any animal test submitted in support of a claim that a hazardous substance caused the claimant's disease.

Third, and finally, bacteria and mammalian cells in tissue culture. These, too, are proxies for human experience. They are used to examine capacity of the test substance to induce mutations or other genetic damage. While the results of the tests do correlate highly with those in animal bioassays, the Okey-Ames test, as it is called, misses certain chemicals, like dioxin, for example, that are proven carcinogens in animal tests and humans. Thus they have inherent limitations that should be identified and safeguarded against in any victim compensation legislation.

If you don't have controls on the kind of evidence a claimant is able to bring into a system, the Congress would be inviting what I call "junk science" in support of claims for compensation. We have already seen, for example, in the first so-called study of chromosome damage at Love Canal the dangers of junk science. If you don't have, for example, a control group in an epidemiological study, it is essentially worthless.

Finally, there are ways, I think, to assure that the best science is available to both toxic substance claimants and defendants. There is underway, of course, the new Toxic Substances and Disease Registry, but we think that the Congress might want to consider going further than that, by combining the capacities of that new organization and all the other existing Federal agencies that are looking at aspects of disease causation: the National Science Foundation, National Academy of Sciences, NIOSH, etc.

If all of that expertise could be brought together in a single entity, a great many of the problems that are leading to pressures for presumptions could be solved by making sure that there was a source of reliable and good scientific evidence to back up or rebut claims of hazardous substance disease causation.

I am sorry for the length of that answer but I thought it was something that should be covered thoroughly.

Mr. **Levitas**. I want to get back to your answer in a minute. I think I will save it for my next go round. But I do want to get back to that because you come perilously close to another concern that I think we have. But I will save that.

You, in your testimony, Mr. Cheek, put out a proposition that at the time struck me as being fascinating. I had never heard it put that way before. Not being an expert in this field, I can't say that it is novel, but it struck me. You were making the point that in the use of statistical evidence to ascertain the causal effect between a toxic substance or hazardous waste site and individual ailments, that one of the methods that is used and that certainly has persuasion to the layman such as myself is that in a particular community where you have an unusual incidence of a particular disease, and that particular disease has scientific linkage with particular toxics, that that is utilized as a basis for the cause and effect requirement.

Then you went on to say that whatever the statistics, the numbers were, but the numbers you used was what, liver cancers as I recall, a hundred——

Mr. **Cheek**. Normally expected.

Mr. **Levitas**. Would be the normal. There is two percentages which you used, but let us say 5 percent incidence, which places it in the abnormal category. Then you make the point, which I thought was very interesting, you say you thereby create a situation of over-inclusivity, because not only are the additional five people or two people the ones who presumably——

Mr. **Cheek**. Could have.

Mr. **Levitas** [continuing]. Have had this ailment because of the toxic substance, but how do you know which of the two—of the 105 or 102 it is. Thereby everybody in that community would be a potential claimant. When I first heard that it sort of struck me that, gee, that is very, very interesting. And it creates a problem, because the other hundred would have developed the cancer under any circumstances, or without this hazardous waste site, or the toxic chemical relationship.

And now they would qualify theoretically at least as potential claimants. On the other hand, as I began to think about it, it occurred to me that there are some at least possible mitigating pieces of evidence that work against that. You take the situation in Congressman Molinari's district, where they ran a number of tests in a particular community. And they used the Staten Island area really as a control for this, what is it, Brookfield?

Mr. **Molinari**. Brookfield.

Mr. **Levitas**. The Brookfield community is close to the dump where hazardous wastes were illegally disposed. In the Brookfield community they found an unusual number of these particular types of ailments, far in excess of what you would find elsewhere in the Staten Island area. I remember in the decision that EPA made on, I forget the numbers, but it was the herbicide that they sprayed out in Oregon, if I recall correctly. And there were a number of abortions, miscarriages, that were not tremendous in terms of the number over what was occurring, but it was abnormal as compared to the rest of the population where this particular substance was not sprayed.

Now, having laid out the dilemma, surely a layman such as myself would conclude based on Mr. Molinari's statistics or the statistics in an Oregon or the Mirex statistics in Mississippi and some other places, that there is a cause and effect relationship. And since we are able to place the people in Brookfield close to the toxic site, and those further away not close to the toxic sites, doesn't that provide a cutoff?

Mr. CHEEK. It does in many instances. The only place you would have a problem is where you have something that is common to the whole area. Clearly, if you are talking about people in proximity to a dump site, yes, that would seem to be almost a self-sealing off kind of population that would admittedly be eligible to bring either a tort claim or some kind of administrative claim.

It would seem to me that where there is so clear a relationship between the exposure and the health effects that those people are suffering, a normal tort action would not only lie, but would almost certainly succeed given, what is it, a 50 percent higher incidence among the people around the dump than elsewhere on Staten Island? That is an extraordinary——

Mr. MOLINARI. One hundred percent.

Mr. CHEEK. That almost by itself exceeds the normal tort threshold of 51 percent in effect of proof. It would seem to me that there is a clear instance where, if the people in that community brought a class action, they would be certified as a class and presumably have very little difficulty in establishing that there is tort liability on the part of the defendants involved in placing that or those hazardous substances in that dump.

There I think is a clear instance where you would not need either the benefit of a presumption, or what I would call junk science, to substantiate the claim.

Mr. MOLINARI. Suppose there were only three people—would the Chairman yield?

Mr. LEVITAS. Yes.

Mr. MOLINARI. Suppose there were three people living in the area, all three suffering from the same symptoms. You don't have 1,000, 1,500.

Mr. CHEEK. There, too, I would think, if there were a common association in their view as to the cause of that illness, and no similar illness elsewhere in the community, there, too, there would be no need for any presumption that the disease was related to the exposure.

Mr. LEVITAS. Well, I happen to agree with that conclusion, that judgment. On the other hand, it still poses the problem to me intellectually that there would obviously be some people who would be certified as being appropriately part of that plaintiffs' class who developed the particular ailment, but developed it quite apart from their proximity to the toxic waste site. I don't think there is anything you can do about it. It is either a question of compensating some people who were not in fact injured by that toxic waste site, or not compensating those who were.

If you are going to err, you have to err, it seems to me, on the side of compensating victims, and, you know, the old theory of some of the guilty go free. Or innocents get convicted, whatever.

Anyway, Mr. Molinari, I will come back to that other line after you have had an opportunity.

Ms. **BIRNBAUM**. Could I speak to that?

Mr. **LEVITAS**. Yes. I thought you would.

Ms. **BIRNBAUM**. In part to that.

Mr. **LEVITAS**. Yes.

Ms. **BIRNBAUM**. I think the answer to that is that the over-inclusive nature also has an impact on the costs. If, say, just using those statistics, we are talking about 100 people that are going to be able to recover, maybe only two of them have been injured by exposure to a hazardous substance; the other 98 may have developed disease from extremely unrelated sources.

We have 400,000 cancer deaths in this country a year, 880,000 people were diagnosed with cancer last year. Statistically there are all kinds of numbers that people throw out of what may or may not be caused by toxic substances in the environment or the work place. The latest studies, the Dahl study which I make reference to in my paper, seems to indicate that less than 2 percent of cancers, 2 to 5 at most, come from chemicals and pollutants in all areas of the environment: air, water, food.

So what we are talking about here is compensating anybody who not only might live near a hazardous waste site, but even those people who just passthrough the area or work there for several hours. I mean it is very hard to draw the kinds of lines in an administrative proceeding that you are talking about. And the costs I think would really be prohibitive under that kind of system. But that is only a partial answer I think.

Mr. **LEVITAS**. Thank you.

Mr. Molinari.

Mr. **MOLINARI**. I don't know where to start. I am trying to recall the source. But there was a source—it might have been the Chemical Manufactures Association paper—which indicated that people who work in chemical companies, with all of the protective equipment, would be exposed to a risk greater than that of the public on the outside because of some opportunities for exposure when there shouldn't be exposure.

Yet it was interesting to note in their final statistical analysis that the chemical workers have a lower incidence of cancer than the general populace. Of course they want you to believe if you want to avoid cancers you should work in a chemical company.

Ms. **BIRNBAUM**. Chemicals are good for you.

Mr. **MOLINARI**. There is no doubt that they have done a lot of good. I can understand, Mr. Cheek, your statements indicating the difficulties with the predictability of loss and how that poses a problem in determining rates that you would charge to one of your insureds. But take a person in my area who would die, let's say, from lung cancer, which is one disease for which we do have a very high rate.

If the person lived in the Brookfield area, and was to sue the party responsible for the chemicals, the chances are that he would be hit with the comment that his cancer could be caused by smoking or some other problems, for example, coal burning power plants. But if we go to the Tobacco Institute we would hear, as I heard here as recently as a week ago, a vigorous denial of the assertion that smoking will cause lung cancer.

Then we go to the remaining source, and that is the smokestack emissions coming from coal burning sources. And I listen to the coal industry and they say it is a lot of ballyhoo. They don't to this day admit, for example, that there is any causal relationship between coal burning power plants and acid rain, though the evidence is starting to come in to the contrary.

Brookfield is not an isolated case. I listened to your assessment as to the legal position of the residents of Brookfield. I couldn't help but think that if we wanted to, on this subcommittee, put on a parade of witnesses victims who have had the same health problems as the people near Brookfield, we could have hearings every day of the week, go forever, and never catch up. I am sure we would hear the same story time and time and time again. As a matter of fact, we have had witnesses before us that told us the same thing.

Now we have also heard from lawyers whose views somewhat sustain your position. Mr. Baron, in particular, was one who said that given a set of facts lawyers generally can prove a case.

But the overall picture, looking at the masses, is one in which we are not necessarily looking at those who have been in severe straits, debilitated. What I am hearing from the trial lawyers about toxic tort litigation is that given the serious case, they will take their chances. But I don't think the average case is that serious. I put quotes around "that serious" because I don't think anybody knows what really "serious" is because of latency periods in particular. But we do know that lawyers have a way of evaluating a case when it comes into their office in order to determine whether they are going to take it on on a contingent fee basis and invest the amount of time and resources that they have to, in order to, hopefully, come out ahead at the end.

But the average case is not a cancer victim. Maybe the case will involve special damages that might amount to several hundreds of dollars. The victim is still faced with the difficulty that we have heard time and time again of getting medical experts. In fact, we heard the rates they were charging were $350 an hour for their time. So how can we say that the system is in place for these people? When I think in terms of Brookfield we may be talking about 1,000 people. But again I don't want to isolate on Brookfield because I don't view Brookfield as a unique case.

I think that there are thousands of Brookfields throughout the country. In fact, there are over 1,000 sites under investigation in New York State alone. So I really see out there the possibility of many hundreds and hundreds of thousand of people who have injuries that are caused by exposure to nearby dump sites and toxic chemicals at the sites. But they don't have sufficient amounts to warrant a lawyer's time.

In my community, as I have said to the chairman, we have maybe 600 or 700 lawyers. If any one individual of those Brookfield residents went to a lawyer, I suspect he wouldn't get 15 minutes of the attorneys time. The lawyer would dismiss them and say to his partner, "Thank God I got rid of that one. That is another loser." You know, lawyers can go broke taking cases like that.

Now, if toxic tort litigation is such a lucrative practice, and if the system is in place and the technology emerging as is claimed, why is it we only have Mr. Baron's firm, which is eight lawyers and, I think he mentioned, only one other firm in the country, which has two lawyers, handling toxic tort litigation. I have difficulty reconciling the assertion with the reality.

Ms. **Birnbaum**. Let me just say, my own experience is that there are many, many, many lawyers practicing in this area of the country. Mr. Baron may be one of the most outstanding ones and one of the better known ones. But if you look around at what has happened in the asbestos litigation, all of those lawyers are trained, competent people. Not all.

Mr. **Molinari**. I would like to stay away from asbestos.

Ms. **Birnbaum**. But that is where you find the cadre of lawyers that are going into this whole new area. That is how Mr. Baron, among others, started out learning the toxic tort area.

Mr. **Molinari**. Sure, but it is a lot easier because you are dealing with cases where you have a known source—asbestos.

Ms. **Birnbaum**. Yes, but you have——

Mr. **Molinari**. You have an isolated source that you are dealing with. Ninety percent of your problem is solved before you start. If you are living near a dump, you may have hundreds or thousands of chemical compounds. You don't know what you are facing when you don't even know what the compound is. How much does it cost to prepare a plaintiff's case in a toxic tort litigation case? What would be a minimum sum required?

Ms. **Birnbaum**. I think it is impossible to give you an estimate without knowing the dump site: what has been put into the dump site: what are the relationships statistically and in the medical literature between those particular substances and what is being alleged. And what I think happens, and I would be surprised if it wasn't happening in your area as well, is that if there are people who are injured, a lawyer doesn't handle just one case. What happens is a lawyer begins to handle hundreds of cases involving people in that neighborhood, sometimes consolidating the actions and bring them together.

So it isn't a question of handling one case, either through joinder or through class actions, what happens is lawyers go out there and sign up. "victims." And if there are real injuries it seems to me there is no shortage of lawyers in this country to handle the cases.

Mr. **Molinari**. Let us go to the next step then and I will tell you what happened.

Ms. **Birnbaum**. OK. I would be curious to hear. It sounds like you may need a lawyer.

Mr. **Molinari**. Some Staten Island residents living near Brookfield went to the attorney who handled the Love Canal case. From the information I have received, he came in and apparently made some type of evaluation. He reportedly wanted a certain flat fee from each one, I suppose to cover the initial work that they would have to do. There was a great reluctance on the part of a rather substantial percentage of the people to put up that money.

These people have had so much difficulty for 19 months trying to get any of the health agencies to tell them what is there and what remedial action can be taken, that they are just totally puzzled. In fact, what some of them have done, frankly, is to sell their homes and get out of there.

So that when I hear that the present system is adequate and then try to equate that with what I know is happening back home, it doesn't square with the experiences that I have seen.

Mr. CHEEK. Mr. Molinari.

Mr. MOLINARI. Yes?

Mr. CHEEK. I think Fred Baron referred yesterday to several lawyers that he knows in New York who would be more than happy to hear about the Brookfield case. As I recollect, he was willing to supply you with their names. I think he even mentioned a couple during his testimony.

Mr. MOLINARI. I made note of that, as a matter of fact. However, as a Congressman, I would not as a matter of practice recommend an attorney. If somebody calls my office, I would likely refer them to the Bar Association.

Mr. CHEEK. I think New York City is certainly one of the centers in which toxic tort law is developing at its most rapid pace.

Mr. MOLINARI. We talked the other day about a 6-year-old child, not from Brookfield but from another area of Staten Island, who is dying from a condition which it is thought might be the result of exposure to toxic chemicals. One of our attorney witnesses said he would take the case of the 6-year-old child. But he was speaking off the cuff, and probably assuming everything else was in place.

I also think he was influenced by the young age of the victim, as opposed to an older individual whose life expectancy would have some impact upon the measure of damages. But I still see this very serious question of trying to prove causal relationship. I don't truly believe that the state of the art is such today that, with the tests that we have, you can indeed prove that.

I took a population health survey. The city did one, also. We came up with amazingly similar results which told us there was a problem in the area. That was enough for us to be satisfied that further action had to be taken by the health department. Whether there was enough evidence to enable these people to go into a court of law, to seek redress and collect damages, I wouldn't want to speculate.

But I would suspect they had a lot further to go.

Ms. BIRNBAUM. I think that is probably true. But isn't it a concern of where you draw lines?

Mr. MOLINARI. Yes.

Ms. BIRNBAUM. Let us say there is someone in your area near that dump site, and that person has headaches. Now, is that person living a half a mile from the dump site, 1 mile from the dump site, 2 miles from the dump site? What about the person who has headaches and lives 3 miles from the dump site? Once you start on this route, the question of whether the headache is related to the dump site becomes extremely difficult to resolve.

The question of where to draw lines is a very hard one. I think even around Times Beach, what occurred at first was the Government was willing to buy the houses of residents that were *x* feet or miles from the dump site. And very soon after that, there was a question of should they buy the houses that were the next *x* miles, and then the next *x* miles until the Government might have to pay for all the houses in Missouri.

That is an enormous problem with the way all these bills have been put forth, even assuming there is causal——

Mr. **Molinari**. Have you read much about Times Beach?

Ms. **Birnbaum**. Just from what I have read.

Mr. **Molinari**. Would you hesitate to live there?

Ms. **Birnbaum**. I live in New York City. That is polluted enough. I am not sure if someone asked me that question where I would live. I don't think I would move to anywhere in Missouri, but that is lifestyle. But I don't think I would move to Times Beach, either.

Mr. **Molinari**. Would you move to Staten Island?

Ms. **Birnbaum**. I sometimes visit.

Mr. **Molinari**. You don't stay too long. How about you, Mr. Cheek, would you live there?

Mr. **Cheek**. Would I live in Times Beach? I don't think I would want to, sir.

Mr. **Molinari**. I appreciate that candor. The chemical manufacturers' representative said he would have no hesitancy to do that. I asked, "How long would you stay?" He said he would have no problems staying there 4 or 5 years. I think it was the following day that NIOSH came out with the report indicating that indeed there might be some causal relationship between dioxin exposure and cancer.

Mr. **Cheek**. Mr. Molinari, could I ask you a question about the people around that dump?

Mr. **Molinari**. Yes.

Mr. **Cheek**. Is there concern that they are not being made whole for the medical expenses and lost wages that they are now incurring?

Mr. **Molinari**. No, not really. Their concern is a health concern.

Ms. **Birnbaum**. Of the future?

Mr. **Molinari**. Present and future. Those people have health problems now, themselves and their families. There are some interesting phenomena. We had a witness from the area last week testify that when the family goes away for a vacation for a limited period of time, a lot of the symptoms disappear. Hours after they come back to the home the symptoms reappear.

Mr. **Cheek**. My sense of what your concern is is not that they are not being compensated for the out-of-pocket expenses associated with those health effects, but rather the perception that they don't have the front money to, in effect, bring a lawsuit for other than economic loss associated with those health effects.

Mr. **Molinari**. Let me lay it out for you. My primary concern is for the unknown health effect. I do know that they have health problems. How serious they are, I don't know. I get asked for example, "Congressman, should I move?" How do you answer the question? What a terrible question to be asked. What a terrible position to be in, trying to answer it.

If you say yes, you will probably instill a sense of panic in the neighborhood. If you say no, then you have your own conscience to answer to if they stay there and they do have a problem.

I know of another area where toxic dumping has been alleged to have occurred. It has not been publicly divulged. I have been attempting to prove that that is the case before I release any information so I don't send panic through the neighborhood. We've been a year working on that and have not really made any progress. Organized crime is involved, compounding the difficulty in getting information, and I rest very uneasy on that.

But now let's go from there. If, in fact, the people there have legitimate health problems caused by the existence of toxic chemical substances, then, yes, indeed, they should be compensated. I assume the amount of their recovery should be like any other negligence case, depending upon the nature of their injuries, permanency of their injuries, their special damages.

As I said before, Brookfield is not an isolated case; I am totally convinced that there are thousands of Brookfields in this country. And I have, myself, done some more limited inquiries into sites throughout the country. I appeared on C-Span here in Washington for an hour. For the first half hour we talked about this problem. Then the second half hour I took phone calls, which came in from all over the country, from people alleging the same kinds of things and asking me for help.

As is true for any of my colleagues, I have enough problems trying to do my job here and answer to the people of my own district. People out in Starrett City called me and asked me to go out there and help them. They are terrified. I would love to do it, but I don't have the time and I don't know how much help I can be. My point is this: we do have a toxic waste victim problem in this country. I think the primary concern for all of us is to try to assess how serious it is so we can try to assist those in need of some kind of help. Then, if there is a guilty party, just like in any other area of the law, that guilty party should compensate the innocent person that has been injured as a result of the guilty party's practices.

Ms. BIRNBAUM. You just used the word that is the heart of what I think I have been saying. If there is a guilty party. If there is a party who was at fault. Many of the suggestions are to hold companies liable who may not have been guilty parties at all, who may have just had the circumstance of dumping properly in the same dumpsite as——

Mr. MOLINARI. The joint and several liability aspect. I understand. That is a very valid point. You mentioned that point before and I made a note of it because I think it is very legitimate. I have other questions, but I think I am going to have to cut them short because one of our other witnesses has a time limitation. But I appreciate your help.

This may be difficult for you to answer, but how about punitive damages? Do you believe there is a place for punitive damages in this area? One of the witnesses used the term "outrageous conduct," stating he felt that punitive damages might appropriately be improved in such a situation.

Ms. BIRNBAUM. Speaking for myself, and I can speak only for my own personal feeling about punitive damages in this context, I think there is a place for punitive damages where there has been outrageous conduct that is in disregard of human safety. When companies try to cover up information, knowledge that they have, to the detriment of people's safety, then the common law response is to hold them liable for punitive damages. I think the problem may be that in some instances we are using punitive damages in the products liability area too expansively, taking in other than outrageous conduct.

But the deterrent effect of punitive damages is a very valuable one in common law, and there is a place for it, so long as there are correct guidelines and standards.

Mr. **Molinari**. Is that insurable?

Ms. **Birnbaum**. It depends on the State you are in.

Mr. **Cheek**. New York, for example, I think prohibits insurance of punitive damages as being contrary to public policy.

Mr. **Molinari**. Public policy.

Mr. **Cheek**. We, and I think most other companies, routinely exclude the coverage of civil fines and punitive damages from normal general liability contracts.

Mr. **Molinari**. I find the testimony intriguing and I thank you both. I am sorry we have to cut it short.

Mr. **Levitas**. There is one question that I want to ask you, Mr. Cheek, because I don't want to let you leave without shedding some light on the record for this.

I have wondered ever since this whole question began to arise several years ago, how in the world do insurance companies arrive at their premium costs in an area that there is really no statistical data on in terms of historic liability. Normally you set your rates on what you have had to pay off in the past. Now, this is a new field. You don't have this historical basis. Do you do it on the types of chemicals produced, the methods of production, the matter of disposal?

You know, I am very curious in finding out how the insurance industry decides how much to charge for its premiums to people who manufacture toxic chemicals or who dispose of or transport toxic chemicals. How do you do it?

Mr. **Cheek**. We are required by State law to look back at our previous loss history. In product liability, for example, we take statistics countrywide over a period of 5 years. The loss and expense experience in that period and the trend in it is the basis for setting the future rates.

The law is changing very quickly in this field and in many States we are finding that past experience is not really a useful guide when new decisions come down that overnight entirely change the whole basis of liability.

The *Beshada* case in New Jersey last year is an example. A productmaker in New Jersey is now in effect an insurer in perpetuity for any harm caused by his products because, the court said, a manufacturer may be held liable for failing to warn of a defect in his product even if at the time of his manufacture of same he did not and could not have foreseen the harm that it might cause.

But the short answer to your question is that we have to look backwards at actual loss and expense experience. We cannot speculate on the relative toxicity of a chemical or its reactivity with other chemicals, or its carcinogenicity. We have to look back at the aggregate of experience for that particular chemical maker and for chemical makers as a whole in setting product liability rates.

Mr. LEVITAS. Could you provide us, if you have it now, if not, provide it for the record, the premium cost increases that have occurred over the last 10-year period?

Mr. CHEEK. We can easily provide that.

Mr. LEVITAS. Would you do that? I would like to have it for the record.

Mr. CHEEK. In the product liability area?

Mr. LEVITAS. Whatever category. When you insure the ABC Chemical Co., how do you fix its premium when they are disposing of hazardous wastes?

Mr. CHEEK. In most of the big chemical companies we are as an insurance industry in what we call an excess or reinsurance posture. Most of the major chemical companies are to one extent or another self-insurers of their exposure. Often they are insured per occurrence as in the tens of millions of dollars which means it has to be a real disaster before their insurance is called upon to respond.

So that much of the data we would give you leaves out a very large universe, probably the major portion of the chemical manufacturing universe.

Mr. LEVITAS. Because they are self-insured.

Mr. CHEEK. Yes.

Mr. LEVITAS. We will be speaking to chemical companies before the hearings are over and I would like to ask them the same question.

Mr. CHEEK. We will give you what we have.

Mr. LEVITAS. There have been other cases where new technologies have come into play that have created insurance—new avenues of insurance coverage. The automobile, for example, the airplane.

Mr. CHEEK. Jumbo jets.

Mr. LEVITAS. That is right. All of those things have created new dimensions. I have a feeling that this is an area where it is going to happen a lot quicker than it has in these other areas. I see in the last few years and certainly in the next 4 or 5 years, I see just a real upward curve in lawsuits and claims.

Mr. CHEEK. So do we.

Ms. BIRNBAUM. And premiums.

Mr. LEVITAS. You may wish to submit something further for the record on how the insurance industry is looking to the future in trying to ascertain how they insure these risks as we are moving into a new area. I think that would be very, very helpful to us.

Mr. CHEEK. I would be delighted to do that.

[The information received from Mr. Cheek follows:]

Crum & Forster Insurance Cos.,
Washington, DC, October 29, 1984.

Mr. Robert S. Prolman,
Professional Staff Member, Subcommittee on Investigation and Oversight, Committee on Public Works and Transportation, House of Representatives, Rayburn House Office Building, Washington, DC.

Dear Bob: Thank you for sending me the transcript pages from my appearance before Congressman Levitas' subcommittee on July 27, 1983. I appreciate your reminding me of the information that I agreed to provide for the Subcommittee's record.

The progress of a hazardous substance from production through use, storage and treatment, to final disposal as a waste involves several lines of insurance, a fact that did not clearly emerge from my exchange with Mr. Levitas. I think it would help clarify the record if I described various coverages involved in our exchange.

First, when a hazardous substance is a "product," the coverage involved is product liability, which is usually sold as part of the manufacturer's Comprehensive General Liability (CGL) policy, which, with certain exceptions discussed below, provides insurance protection against any liability arising out of the operation of the business. The product liability component of the policy will cover the manufacturer's liability arising out of harm caused by the products he produces, whether that harm results from defects in the products, breach of express product warranties, defective product design, or failure to adequately warn of possible product dangers.

For example, if a manufacturer warrants that its product will remove grease and it fails to do so, his product liability coverage will compensate the product user for the resulting damages. Similarly, if the product is caustic, and the manufacturer fails to provide a product warning to this effect, his product liability coverage will provide coverage for the damages suffered by persons burned by the product.

As noted in my testimony, most of the chemical industry is self-insured for its product (and most other) liability, so that there is very little commercial insurance data that would indicate, in any meaningful fashion, the frequency and average amount paid in product liability claims against chemical manufacturers.

Indeed, the best data we can provide is the enclosed table from the Insurance Services Office, the industry's principal statistical and rate advisory organization, setting forth rate level increases for all product liability country-wide, for both basic and increased limits products liability coverage. It should be noted that the rate level changes in the table are those indicated by industry-wide loss and expense experience; they are not necessarily a reflection of actual price increases in the insurance marketplace. For the past several years, because of intense competition among insurance carriers, product liability insurance rates have generally lagged far behind the trend in loss and expense experience.

The second line of coverage involved in our exchange is the "sudden and accidental" pollution coverage commonly found in CGL policies. This coverage is designed to respond to situations in which the manufacturer's products or operations result in sudden and unintended pollution. For example, if a chemical tank on the manufacturer's premises ruptures, and floods a neighbor property, the "sudden and accidental" pollution coverage in the CGL policy will pay for any ensuing bodily injury or property damage.

Unlike loss and expense data involving product liability, losses and expenses associated with "sudden and accidental" pollution are not separately coded by insurers reporting their CGL experience to the Insurance Services Office. Thus, although the industry knows that claim frequency and average claim severity in pollution incidents are increasing, it cannot isolate precise pollution loss and expense experience from its other CGL data.

Because of the growing importance of pollution-related losses, the Insurance Services Office Executive Committee decided last month that, effective no earlier than January 1, 1986, all pollution coverage (except for that relating to the product liability or completed operations peril) would henceforth be excluded entirely from the CGL policy. "Sudden and accidental" and "non-sudden or gradual" pollution coverage would henceforth be provided under a separate pollution liability insurance form, whose loss and expense experience would be separately reported.

However, because the changeover will not be mandatory until January 1, 1987, there is likely to be little meaningful pollution liability insurance data available before the end of the current decade. And the credibility of such data as does become available will be affected by demand for the coverage and the frequency and severity of claims under it.

The third major coverage involved in our discussion is the so-called Environmental Impairment Liability (EIL) contract, which is designed to cover "non-sudden or gradual" pollution and thereby dovetail with the "sudden and accidental" coverage now provided under the standard CGL form. The EIL market is quite small, with a premium volume of perhaps $40 million last year, as contrasted with the $7.4 billion in general liability premiums during the same period.

The small size of the EIL market results not only from the relative novelty of the coverage (the first policy was written in 1973), but also because of the relative instability of the EIL reinsurance market and the high degree of uncertainty about the need for EIL coverage created by court decisions holding that transparently non-sudden damages are covered under the "sudden and accidental" language of the CGL policy. Some courts have gone so far as to hold that "sudden" is simply another word for "accidental," and that, as long as the insured's polluting act was not intentional, coverage is provided even for pollution incidents of many years' duration. These holdings have discouraged the growth of the non-sudden pollution liability insurance market.

As would be expected in such a small and fragmented market, EIL insurance loss and expense data is sparse and insufficient in either duration or dollar volume to be credible for rate making purposes. Indeed, as Mr. Levitas surmised during our exchange, rate-making in EIL, and in the pollution component of the CGL, is more a matter of judgment than of reliance on (occasionally non-existent) loss and expense experience.

Mr. Levitas was intuitively correct in identifying some of the factors that underwriters consider in granting and pricing pollution coverage. These factors include, as Mr. Levitas suggested, "the types of chemicals producted, the methods of production, (and) the manner of disposal." However, these considerations, and others, are applied with respect to a manufacturer's entire product line, rather than to individual chemicals, because all of his liability, from whatever product line, is covered by the contract. Only in rare instances (e.g., asbestos, swine flu vaccine) have specific products been explicitly excluded from a given manufacturer's product or environmental liability coverage.

It has been suggested on a number of occasions that insurers could isolate, and separately rate, individual products or wastes produced by insured manufacturers, on the theory that more dangerous products and wastes should carry a higher liability insurance premium than less hazardous substances. However, because of the paucity of experience with individual chemical products or wastes and the critical role that management attitude plays in loss prevention, it's simply infeasible to separately price the exposure for all, say, 20,000 chemicals produced by a single manufacturer. The expense would bear no relationship to the manufacturer's exposure. Moreover, volume is usually a more reliable indicator of potential loss exposure than either toxicity, reactivity, or even carcinogenicity. There is clearly more opportunity for leaks and spillage in handling hundreds of tank cars of a low toxicity product than in delivering a few drums of a highly toxic material.

Most chemical manufacturers' insurance coverage provides enormous aggregate limits (usually in the hundreds of millions of dollars), but does not come into play until the liability arising out of a particular occurrence has exceeded the manufacturer's self-insured retention, which will typically be on the order of tens of millions of dollars. The rates for the coverage provided under these circumstances largely reflect actual loss and expense experience or, more recently, reinsurers' perception of the exposure involved.

Although there is little data on the rates charged for EIL coverage for firms who cannot afford to self-insure their gradual pollution liability, we can tell you, on the basis of our three years (1981–1983) experience in the EIL business, that rates increased drastically (an average of 30 percent) in each year of the program, not because large numbers of claims were paid, but rather because a relatively small number of possible claims were reserved for very large amounts, requiring our reinsurers to sharply increase their prices to cover their potential exposure under the policies we had written. It is fair to say, given the instability of the legal regime governing pollution liability, that our European reinsurance underwriters grossly underestimated their loss exposure. After three years of steadily worsening experience, they simply withdrew from the market, and we were forced to follow suit this year.

Our experience in the EIL market was not unique. A year ago there were perhaps a dozen active EIL markets in the United States. There are now perhaps three. The principal reason for the shrinkage of the market is the almost total withdrawal of foreign reinsurers from the American pollution liability market. Court decisions like that in the Jackson Township case, where $15.6 million was awarded to 97 families in which there was no evidence of any clinical illness, and the incredibly extreme liability standards being proposed in both State legislatures and the Federal Congress, combined with very poor loss experience, have driven even the world's most adventurous underwriters out of the market.

It is my judgment, one I believe is shared by most of my colleagues in the industry, that the pollution liability insurance market will remain unstable for several years to come. Indeed, there may be a serious availability and affordability problem in pollution liability insurance as early as next year, when the final phase of the Environmental Protection Agency's Resource Conservation and Recovery Act financial responsibility regulations become effective for all hazardous waste storage, treatment and disposal facility owners.

The source of future instability will obviously not be loss and expense experience, since so little coverage is currently being written, but will rather be the unsettled state of the law governing liability for and coverage of harm caused by exposure to hazardous substances. Mr. Levitas was, as usual, perceptive in his comment that there will be "just a real upward curve in lawsuits and claims" in the environmental area.

Judging from the insurance industry's reaction to the Minnesota Superfund liability system (see enclosed article from the Wall Street Journal of October 23, 1984), and the violent conflicts in the House last summer over the "Federal cause of action" in H.R. 5640, it is likely that major insurers will wait until the law of pollution liability is more settled than it currently is before making a commitment to the pollution liability market.

Generally speaking, the industry has been able to insure tort liability in the United States for the past two hundred years because the common law evolved very slowly. As Congressman Levitas noted during our exchange, change in the "toxic tort" are "is going to happen a lot quicker than it has in these other areas." The very rapidity of that change cuts against the essential element in the insurability of any risk—predictibility.

I hope the foregoing is sufficient to answer the questions left unanswered during my appearance before the Subcommittee. I would be pleased to discuss these issues in more detail if you feel it necessary. I also look forward to visiting with you about the Subcommittee's report on its "victim compensation" hearings at your convenience.

Best wishes.

Sincerely yours,

LESLIE CHEEK III,
Vice President-Federal Affairs.

Enclosures.

Insurance Services Office, countrywide price activities, product liability rate level revisions, 1975–84

[All States combined total limits]

Year:	Percent
1975	+117.3
1976	+35.7
1977	+3.1
1978	+0.1
1979	−1.6
1980	−0.7
1981	−5.9
1982	+6.4
1983	−3.9
1984 (9 mo.)	+8.2

[From the Wall Street Journal, Oct. 23, 1984]

MINNESOTA'S POLLUTION STATUTE GENERATES BOYCOTT BY INSURERS

(By Eugene Carlson)

Minnesota is a paradox. Few states are so blessed in terms of educated workers, squeaky clean environment, honest government and productive industry. Yet few states in recent years have been so thoroughly bashed by their own business community.

Like North Woods wolves on the prowl, Minnesota business executives have ganged up on their state government, howling about high taxes and onerous regulation that they say have bled the state's business climate. The Legislature, cornered by the business lobby, has redressed a number of these complaints.

Now business smells blood again. The target this time is Minnesota's year-old Superfund law that deals with the problems of toxic waste. The focus of anger is a section of the law that lays out a new, strict standard for liability in the case of personal injury, if that injury can be traced to the delayed effects of toxic material. The standard is so tough that the insurance industry has taken an unusual step: It has stopped writing peronal-liability coverage in Minnesota for damages stemming from "non-sudden" pollution.

Example: A train crashes, chlorine leaks from a tank car, and people are injured by the fumes. That's a "sudden" occurrence in the eyes of insurers. Claims are covered by the railway's normal liability coverage. But cancer that a family says resulted from toxic chemicals leaching into their residential water supply over 20 years is a "non-sudden" occurrence. Coverage against claims from this sort of long-term pollution has dried up in Minnesota.

The Minnesota environmental-insurance boycott is being closely watched by other states, among them Massachusetts, Michigan, New Jersey and North Carolina. These states are studying Superfund-type laws of their own. Minnesota, with the first and only such state law to date is the model.

So far, Minnesota's Superfund experience hasn't been a model for anyone. Ask Gov. Rudy Perpich, who signed the Superfund bill into law in May 1983, after his predecessor had vetoed it a year earlier. Last week the governor was in London with a state economic development delegation, selling Minnesota as a swell place for British business to invest. But Gov. Perpich also had the awkward assignment of visiting Lloyd's of London. There, he listened to insurance underwirters explain the "Minnesota exclusion" that is now standard in the reinsurance policies that insurance companies buy from Lloyd's to cover their own risk in writing environmental-impairment liability (EIL) insurance.

When Lloyd's decided late last year that Minnestoa was a bum EIL risk, the rest of the industry fell in line.

The insurance industry says it has no choice but to give Minnesota the cold shoulder. Insurance executvies point to two parts of the liability section that they say are heavily biased toward plaintiffs. In the hands of a clever trial lawyer, the settlements could be enormous, the industry fears.

Under the "causation" section of the law, a defendant in a toxic tort case, say a company that handles hazardous waste, must prove that it didn't cause a worker's injury. Under common law, the burden of proof is traditionally the plaintiff's.

"Our conclusion was that if you can't keep causation away from the jury, the juring being sympathetic human beings, will find for the plaintiffs in virtually every case," says Leslie Cheek, vice president for government affairs of Crum & Forster, a property-casualty insurance holding company.

A second provision makes some toxic waste-generating companies liable for problems stretching back to 1960. And the liability is "joint and several," meaning that one defendant could get hit with all the damages if the others can't pay.

Critics also point out that Minnesota's victims aren't likely to be just the gritty smokestack industries normally associated with toxic waste. Most high-tech concerns cast off environmentally hazardous material as a manufacturing by product. This makes them potential Superfund targets. Attacking high tech in Minnesota is a jab in the gut.

Fans of Superfund, however, suggest industry fears may be overblown. EIL insurance itself is new and it is still a tiny market: about $40 million in premiums written nationwide last year, compared to $300 million in workers compensation premiums written in Minnesota alone, according to a state insurance survey.

"There's no history of claims data," says a state insurance official. "The industry's dealing with a new line and they don't know how to price it. It makes them overly cautious." Says Ralph Marlatt, president of the Insurance Federation of Minnesota: "We want to write this coverage. There's money to be made here. But all the parties have to sit down and come to some agreement."

That may just happen. Gov. Perpich says he'll gather the key players, including the Lloyd's underwriters, around a table in the next few weeks and discuss the feasibility of modifying the Superfund in the legislative session that begins Jan. 1.

And the two authors of the Superfund bill, while loyal to the principle, sound ready to compromise. Rep. Dee Long says that if a survey now under way shows the causation section to be a stumbling block, "then it behooves us to take a look at it. * * * I don't think we ever ought to pass something that's cast in concrete."

Superfund co-author Sen. Gene Merriam goes futher. "I think the only way to modify it is to get rid of the causation section if indeed that's what's causing the problem," he says. "It's a nice notion, but it may be ahead of its time."

Mr. CHEEK. If I could comment on one thing, historically the common law has tended to evolve, that is, the movement has been fairly slow but there has been a gradual adaptation to new kinds of liability and new concepts of liability. But there is some possibility that, as you suggest, that evolution is on the brink of becoming a revolution.

Mr. LEVITAS. I think so.

Mr. CHEEK. We have some concern about the concept that, as between an innocent injured party and an innocent product manufacturer, it is easier for the product manufacturer to be the insurer of that harm than otherwise. We see the law moving in that direction.

If that movement accelerates, then we may be the ones coming before you and recommending that the common law be arrested and that a nonfault kind of system be established.

Mr. LEVITAS. I predict that will happen. I was in the State legislature when we passed the no-fault insurance law and I remember who was there advocating it and I remember who was there opposing it.

Mr. CHEEK. That is right. We don't see in the data—we have to look backward—the evidence that that is yet translating itself into larger numbers of claims. There are certainly larger numbers of claims, but their costs have not yet become so uncontrollable that we cannot predict it and keep up with them.

But that day may come. I hope it doesn't frankly. I would be happy to provide something for the record.

Mr. LEVITAS. I think that would be very helpful and it might be useful even for your own industry to focus on that.

Mr. CHEEK. It certainly would, Mr. Chairman.

Mr. LEVITAS. I want to thank both of you very much for your testimony. It has been quite helpful and has opened up new thoughts for all of us on this subject and we appreciate your being here.

Mr. CHEEK. Thank you for having us.

Ms. BIRNBAUM. Thank you, Mr. Chairman.

Mr. LEVITAS. The next witness is Dr. John Beljan, provost and senior vice president, Wright State University, Dayton, OH. He is here representing the American Medical Association, and is going to be accompanied by Mr. Harry N. Peterson, director of division of legislative affairs, American Medical Association.

Gentlemen, if you would raise your right hand, please.

[Witnesses sworn.]

Mr. LEVITAS. We appreciate your being here for a number of reasons. First of all we are quite interested in the medical profession's view on the subject of toxic chemicals and specifically on the subject of one toxic chemical, the substance dioxin. I know, Dr. Beljan, that you were the chairman of a committee or study group, a task force on agent orange in which the compound dioxin is present, and therefore have a great deal of experience in this area.

In addition to that--this was just from my own perspective—when I read in the newspaper the resolution adopted at the meeting of the AMA in Chicago and then some subsequent action taken shortly after the original press report, I frankly was confused to begin with. I was bewildered to begin with and confused at the end and thought it would be very important to clarify for the record in these hearings—I know there has been other testimony, but in these hearings—just what occurred so we can put that one aside and get on to the substance of other matters in this regard.

So thank you so much, Dr. Beljan. We look forward to your testimony.

TESTIMONY OF JOHN R. BELJAN, M.D., PROVOST AND SENIOR VICE PRESIDENT, WRIGHT STATE UNIVERSITY, DAYTON, OH, REPRESENTING THE AMERICAN MEDICAL ASSOCIATION, ACCOMPANIED BY HARRY N. PETERSON, DIRECTOR OF DIVISION OF LEGISLATIVE AFFAIRS, AMERICAN MEDICAL ASSOCIATION

Dr. **Beljan**. Thank you, sir.

As you say, I am John R. Beljan, M.D., and I am senior vice president of Wright State University in Dayton, OH. I was the former dean of its school of medicine and I would like to say that I teach medical science and not law and with that stipulation, please, I would like to have that entered on the record.

I am currently a member of the Council on Scientific Affairs of the American Medical Association. I served as chairman of the Association's Advisory Panel on Toxic Substances that prepared the AMA's 1981 report on "Agent Orange" and dioxin contamination. Accompanying me is Harry N. Peterson, director of the Association's Division of Legislative Activities.

Mr. Chairman, we are pleased to have your invitation to appear here today to discuss the AMA's view concerning the human health effects of exposure to dioxin.

In 1981, the AMA House of Delegates adopted a report entitled "The Health Effects of 'Agent Orange' and Polychlorinated Dioxin Contaminants." Copies of this report were submitted to the subcommittee. I believe you have them in your possession, Mr. Chairman.

This report was developed by the Association's Council on Scientific Affairs and was based on the investigation of its Select Panel on Toxic Substances of which I was chairman. And with your permission I would like to take a few minutes after this testimony to describe how that was assembled.

This report was initiated because of concern about the reported adverse effects to humans from exposure to agent orange during the Vietnam conflict. Medical complaints noted by veterans included weight loss, liver damage, recurrent skin rashes, deformed offspring, stillbirths, cancer, sterility, personality changes, and other illnesses. The report, however, was not limited to studies involving exposure of U.S. forces to agent orange but included a review of all reported exposures during the last four decades.

Dioxin, TCDD, was present in agent orange as a byproduct of its manufacture. If you recall, agent orange is a 50/50 mix of 2,4,5-T, and TCDD, and dioxin is a byproduct contaminant in the manufacture of 2,4,5-T.

The information used in this study was the currently available scientific information published in the world's literature. This study by our panel was a major undertaking given the very substantial amount of literature reporting experience with animal and human populations that has been generated over the last four decades. This report states, "TCDD by itself is extremely toxic; along with botulinus bacillus toxin and aflatoxin, it is one of the most lethal organic chemical compounds known to man."

The report recognizes and describes the toxicity of TCDD, pointing out the varying toxicity among different animal species and the relationship between toxicity and the amount and type of exposure and ingress to the body. This position, formulated in 1981, is still the policy of the AMA.

The report concludes that the suggested adverse health effects on human beings attributed to dioxin exposure, there is conclusive evidence of a cause and effect relationship only with respect to chloracne. Chloracne is not an uncommon form of occupational dermatitis and may be caused by other chlorinated chemical compounds. It usually clears within several months after cessation of exposure, although severe chloracne may be persistent and has been known to last for as long as 15 years following cessation of exposure to the causal agent.

With respect to other human health effects that have been attributed to dioxin, TCDD, the Council on Scientific Affairs and its advisory panel concluded that there was insufficient published data subject to peer review to establish a definite relationship between exposure and adverse effects. The report reviews in detail the published scientific literature that supports these conclusions.

I would note that the report of the Congressional Research Service of the Library of Congress entitled, "Dioxin: Environmental Impacts and Potential Human Health Effects," published June 6, 1983, surveys the body of scientific opinion and its conclusions. This survey refers to and is consonant with the 1981 AMA report.

Mr. Chairman, we feel that the AMA has a continuing obligation to promote the pubic health in matters such as this and to inform the profession and the public of the most current information available. We note that this matter is not static and that new evidence will be reviewed as it becomes available. We do not intend to let this important matter drop.

Action taken by our House of Delegates last June calls for an updating of our report on agent orange and dioxin along with an active program to provide the public with the most accurate information available on dioxin. This review by our panel will begin next month. As a matter of fact it will be 2 weeks from today.

The findings are expected to be presented to the AMA Board of Trustees with subsequent consideration by the House of Delegates. We will inform you of our conclusions.

The AMA at present is also engaged in extending its studies beyond dioxin to the reproductive toxicity of other chemicals. A group of physicians with expertise in toxicology, occupational medicine, pediatrics and obstetrics has been asked to summarize the reproductive effects of chemicals in males and females, including prospects of mutagenicity.

The panel—by the way not necessarily all AMA members—has selected a group of chemicals commonly encountered in industry as an initial project, has developed a standard format for reviewing and describing the effects of chemicals, and is now summarizing the toxic effects of each. This process has been underway for more than 2 years and should be completed in about another year. We believe that this report will add substantially to current information concerning health effects of chemicals.

Another subject of great interest to us is that of the effects of toxic chemicals in waste dumps. Among the concerns are those that relate to the possible effects of the chemicals on drinking water, and effects on the public who are exposed through contact with agents through the skin or through the air. In addition to difficulties of identifying the effects of small doses over long periods of time, there is another concern stemming from the widely varying geologic characteristics of the disposal sites. There is also great variation of course, as you well know, among the sites in terms of the chemicals they contain.

While much scientific study is necessary on the problems hazardous wastesites may pose, it is important that data that is acquired and that is disseminated be sound.

Regarding the question of scientific evidence about human health effects of toxic substances, one significant point must be stressed. Positive findings in experimental animals may be suggestive, but they are not equivalent to positive findings in humans. This is especially true if there are great differences between the experimental animals and humans in terms of usual doses and usual routes of administration, and in terms of metabolism and system responses.

Accurate measures of exposure and of disease, and indeed the specific agents involved, are necessary. And it is almost always necessary to compare results with a group of nonexposed persons with similar characteristics before a definitive finding can be made about the health effects of exposure.

As I have indicated, on the broader issue of health effects of toxic substance exposure in general, the American Medical Association will continue to be active in reviewing the medical literature in this area. However, as I am sure you are well aware, we are talking about many different compounds and various ways in which the public may be exposed to these substances. I think you are aware we use the term "dioxin" but there are 22 isomers of that, 2,3,7,8-TCDD and in that family of agents there are at least 60 or 70 agents chemically related.

We urge the Federal Government and industry to continue to support necessary research in this area so that there will be better information on which to base future decisions related to toxic substance exposure, and they can be rationally made.

Mr. Chairman, we are appreciative for the opportunity to be here. We would like to assure you of the cooperation of and assistance of the association in addressing the investigation problems of this area and we would be pleased to answer any questions the subcommittee may have.

I would like, however, if I may, to just make two brief verbal addenda. One relates to the action of the House of Delegates and, two, how our committees are put together.

Let me first address the latter.

There is a body called the "Council on Scientific Affairs" which is elected by the membership which has within its structure individuals with broadly-based backgrounds in a variety of medical specialties. That council is the coordinating body for a variety of expert panels which are put together in which we draw experts from the United States both from the medical and biomedical community, members and nonmembers, who are experts in the area being addressed to review the world literature in terms of the subject at hand.

That was the committee I chaired and we did review the literature available as of 1981.

We will review the published available public literature available to us not in the New York Times but that which is in the scientific literature 2 weeks hence and in subsequent meetings to that initial meeting.

In regard to the unfortunate publicity regarding the action, the resolution of the last meeting of the House of Delegates, the resolution arose in the Missouri Delegation and was forwarded for review in the House of Delegates by means of the methodology of reference committees.

In those reference committees, although the whereases in the document are considered when they are recommended for adoption by the reference committee, only the resolves appear as official AMA policy.

I have in a previous hearing regretted some of the improvement language that was in the whereases, I was also bold enough to say that I thought Congress on occasion made imprudent comments as well. I can assure you that the AMA did not in effect endorse the language that was embodied in the whereases of that resolution but did in fact adopt as a social policy the two resolves, to wit, the first resolve that the American Medical Association institute an active public information campaign to get accurate information on dioxin before the public to prevent irrational reaction and unjustified public fright and to prevent dissemination of possible erroneous information; and be it further resolved that the AMA Council on Scientific Affairs be requested to update the 1981 report on dioxin.

I would assure you the AMA did not change its policy one bit. We stand by the report and are prepared to revise that report if warranted by critical review of the scientific literature.

Mr. **Levitas**. Thank you very much, Dr. Beljan, for that explanation as well as the testimony you have given us on the subject of dioxin.

I know we have a number of questions and I would like at this time to recognize my colleague from Georgia, Dr. Rowland.

Mr. **Rowland**. Thank you very much, Mr. Chairman, and Dr. Beljan, may I welcome you, my colleague, and Mr. Peterson. It is very nice to have you here.

Dr. **Beljan**. Thank you, sir.

Mr. **Rowland**. And contrary to what I may have heard Congress is a great institution and contrary to what Congressmen may have heard, medicine is a great institution.

Mr. **Molinari**. What about law?

Mr. **Rowland**. I purposely omitted that.

My good friend from New York and I have had a running commentary as doctors and lawyers often have, but it has been fruitful for both of us.

I was particularly interested in the resolution and of course you cleared up the AMA's stand and what is in the "resolves," and what it is not involved in the "whereases."

Dr. **Beljan**. That is right.

Mr. **Rowland**. I was particularly interested in the fact that this resolution was a product of the Missouri Delegation.

Dr. **Beljan**. Yes.

Mr. **Rowland**. And Missouri is where Times Beach is located.

Dr. **Beljan**. Right.

Mr. **Rowland**. We heard from the Mayor of Times Beach here a couple days ago about the illnesses of many, many people in Times Beach. She is convinced these illnesses came about because of the exposure to dioxin there.

Dr. **Beljan**. Yes.

Mr. **Rowland**. And yet the Missouri Delegation to the AMA didn't feel that dioxin was any particular problem. So I was wondering, had physicians from Missouri that may have had input into this resolution have any contact with the people from the Times Beach area? If so, what were their findings?

I found the testimony that the mayor gave somewhat astounding; many of the things that she said were somewhat astounding; and I have asked for some followup from physicians in that area to substantiate or refute many of the things she said. I would just like to have your comments on that if you do have some knowledge in that area.

Dr. **Beljan**. I don't have extensive knowledge and what I would tell you would be perhaps secondhand or hearsay information but I do believe that there were concerns in the Missouri Delegation that too much of the public information that had been released through the media and so forth was anecdotal, suggestive and if I could say, sir, contagious, and that in fact there were patients not from the Times Beach area approaching Missouri physicians with a variety of ailments and concerns about their environment and potential of dioxin and other agents in their immediate environs.

And if you will, there were, I think one of the motivating factors behind that was a sense of almost hysteria that had been generated by perhaps some of the anecdotal information and so forth that related to it. Undoubtedly some of our colleagues in the Missouri Delegation have treated or seen individuals from Times Beach. I am not personally aware, however, of those interactions or what they may have found.

Mr. **Rowland**. She mentioned one instance of a block where there were 15 homes and out of the 15 homes there, 7 of the homes had people with seizure disorders. I doubt if we would find that anywhere in the country out of 15 families.

That was just something that concerned me very much. Is that actually the situation that existed? Can you speak to that?

Dr. **Beljan**. I can't speak to that, sir, I do not know because it has not appeared in the literature that is available to us. I have learned not to rely necessarily on other media for sources of scientific information.

Mr. **Rowland**. From your experience then, in talking with physicians from that area, your experience is that much of what was thought to be symptoms or signs relative to dioxin was in fact not that at all?

Dr. Beljan. I do not want to make attributions to——

Mr. Rowland. I know I am asking you for hearsay.

Dr. Beljan. The concern that I think I saw reflected in the Missouri delegation was concern about how information regarding this agent had been made available to the public and how this was being reflected in the general medical practice of the Missouri physicians. I have not talked directly to physicians who were involved in the care of the Times Beach individuals. So it is a double hearsay thing in terms of their perceptions of what went on.

Mr. Rowland. Let me ask you something else then.

TCDD is an extremely toxic substance related to botulism and aflatoxin.

Dr. Beljan. Yes, sir.

Mr. Rowland. Since it is this toxic how would you explain the apparent lack of severe problems in humans who are exposed to it?

Dr. Beljan. First of all, the toxicity as you well know, Dr. Rowland, varies considerably even among experimental animals and even among families of experimental animals that are similarly related.

For example, one of the most sensitive animals is the guinea pig and yet there is a 500-fold difference between that and the hampster in the same general family.

The agent itself if it is exposed to air is rapidly focalized and under the influence of ultraviolet light will generally disappear within 24 hours. In the ground it binds tightly with soil and raises a whole series of questions, therefore, about bioavailability and ingestion and so forth.

There is a large body of evidence that would tend to indicate that there have been large populations who have been exposed to dioxin and it would appear as if the human being is one of those creatures who may be particularly resistant to it. I would not personally want to ingest it. On the other hand, I am aware of a critical review of the Oregon study, the industrial accidents that have occurred, I have just been provided with a copy of the report, primary report on the ranch hand study by the Air Force.

That is an exquisitely done statistical study.

I am aware of the early report of the Armed Forces Institute of Pathology with the cohort of Vietnam veterans surgical and autopsy material and I am impressed that there is not the clustering or patterning or increased incidences of problems that one would expect to see if the agent in fact were as dangerous as I would think it has been made out to be by some.

No question in the laboratory, it is a terrible agent. But as we pointed out, in the instances in which it has been used and in certain industrial accidents and where a whole town had been bathed in the stuff, and there is no clustering or unusual evidence of abnormality, it would indicate that there has been wide use that man may be particularly resistant, it may be a short exposure or even prolonged exposures of short duration are not particularly dangerous.

It would be useful perhaps, maybe not medically solubrious if we say the patterning and clustering that we see with other agents with direct kinds of relationships. But we have come to that conclusion as a body. The scientific panel on pesticides in Great Britain has come to that same conclusion. That is true of several others who have taken the scientific literature and done a critical analysis of it.

We have not seen some of the reported other studies that have been recently released. We would hope to have access to them. We would like to certainly have our panel and others review that in a critical framework. But from the standpoint of what is available to us in the current literature the AMA policy currently seems to be a reasonable one.

Mr. **Rowland**. Are you familiar with an instance about 2 weeks ago wherein the court ordered Dow Chemical Co. to release some information regarding dioxin.

Dr. **Beljan**. I am only aware of the event having occurred, having read it in the newspaper and hearing it on television.

Mr. **Rowland**. They talked about chloracne and it is well accepted that chloracne is the result of chronic exposure to——

Dr. **Beljan**. Or massive acute exposure as well.

Mr. **Rowland**. But there was also some revelation there that Dow Chemical had determined sometime back that fatalities may result.

Dr. **Beljan**. Again it is conjecture. It would be conjecture on my part. I have not seen the correspondence being referred to or what have you. I do know that words get garbled. For example, I am sure that they were aware of animal toxicity studies and I stipulated it is a terribly toxic agent in the laboratory animal. I think if I were faced with that, again conjecturely on my part, I would be concerned if there are some carryover effects elsewhere. I am not so sure—I have not seen that correspondence, I have not seen what was said, I don't know how it was interpreted.

I certainly believe that the Dow Chemical Co. generally is an ethical pharmaceutical company and conducts its operations in that framework.

Mr. **Rowland**. One other question I have, Mr. Chairman, regards immune systems. We have heard that some chemicals may break down immune systems. Is it possible that some chemicals may in fact activate immune systems?

Dr. **Beljan**. Yes, sir.

Mr. **Rowland**. So that a person might build up immunity rather than going in the other direction?

Dr. **Beljan**. Yes, sir.

Mr. **Rowland**. Do you have any idea about dioxin from that standpoint?

Dr. **Beljan**. We are aware that there are several laboratories conducting studies in the immune area, none of them have been published, none have made conclusive results. We heard of one study being done, testimony a week or two ago at a subcommittee of the Veteran's Affairs. It is a masters or doctoral thesis work currently in process. There is work by others who have shown some changes in the immune mechanism. But, yes, I would submit that this is a possibility with dioxin.

I would like to, again, we will incorporate whatever is available in the world's published literature on this in our review to commence very soon.

Mr. ROWLAND. Mr. Chairman, I have some other questions but I want to defer at this point.

Mr. LEVITAS. Thank you, Dr. Rowland, I appreciate your questions.

I will now recognize Mr. Molinari. We are going to try to get you out of here by 3:35.

Dr. BELJAN. Thank you, sir.

Mr. MOLINARI. Doctor, in your statement you refer to TCDD as being one of the most lethal organic chemical compounds known to man and yet it would seem that there is a lack of scientific literature or scientific evidence so far to be able to determine the impact upon human health.

Dr. BELJAN. Yes.

Mr. MOLINARI. The jury is still out on that, is that fair to say?

Dr. BELJAN. I think it is fair to say that in the laboratory it is a terribly hazardous compound, that the studies that have been available to us to date have not shown a direct carry-over experience with man.

Mr. MOLINARI. Is it true that dioxin is not comparable to other chemical compounds in terms of laboratory testing and that it doesn't exhibit the same relationships as to lethal doses administered to animals?

Dr. BELJAN. The problem in the literature is the problem I alluded to in my remarks, that people use the term "dioxin" and one does not know which of the 22 isomers is under discussion, or whether all of them are or any part of them; or the fact that in the dioxin-related family there are some 70-odd compounds, each of which has different toxic properties.

Mr. MOLINARI. The problem I read about was that in laboratory testing—I don't know which compound was involved, but I assume it was the same one administered to different animals—some animals didn't show the same dose response——

Dr. BELJAN. That is correct.

Mr. MOLINARI [continuing]. That others normally would. So it became more difficult to evaluate——

Dr. BELJAN. Yes.

Mr. MOLINARI. What quantity would be lethal, for example.

Dr. BELJAN. That is correct.

Mr. MOLINARI. Since we don't experiment with humans we don't know what quantity administered directly to an individual would be lethal.

Dr. BELJAN. That is correct.

Mr. MOLINARI. All right.

Dr. BELJAN. As I pointed out, even within families of related animals where one would expect there would be closer relationships in terms of dose effect responses that is not the case, specifically the hamster and the guinea pig is a 5,000-fold difference.

Mr. MOLINARI. Dioxin, correct me if there are some forms that don't fit into the general category, can be absorbed through the skin, can it not?

Dr. BELJAN. Yes.

Mr. **Molinari**. Although there may be a variance in the quantity that becomes lethal or harmful, isn't there some relationship between the toxicity of dioxin and the health hazard you might expect to result from exposure?

Dr. **Beljan**. It is extremely difficult to answer that question generally because one is talking I think in terms of dose response relationships with regard to either acute or chronic dosage, you are talking about the variability of subjects, we do know, for example, that in a variety of other ways that the very young and the very aged maybe because of their immune systems are quite different in their reactions.

We have the problem of route of ingestion, activity of the substance, the bioavailability of it; so it makes it very difficult, sir, for me to try to answer that question.

Mr. **Molinari**. I wonder how you can come up with the conclusion that it is indeed one of the most lethal organic chemical compounds known to man.

Dr. **Beljan**. We give it to animals and it kills animals. I would say that that would classify it as hazardous to animals.

Mr. **Molinari**. I understand that there are others that would be equally lethal or more lethal in doses administered to animals. What I am trying to do is see if there are other compounds, by comparison, which are equally or more toxic. You point out that there are so many varieties and chemical compounds within the generic term "dioxin" that sometimes we do not know what we are talking about.

Dr. **Beljan**. We know it is hazardous to animals; we do not have the same scientific basis with respect to man. That is desperately a missing area in terms of our fund of scientific knowledge, unfortunately. The dilemma, sir, about making assumptions about effects in animals and making translations to man is not restricted to dixoin. It is true with a whole host of almost all compounds; we always search for a per animal model and there is none. We look for other animals. The Ames test talked about earlier in terms of changes in microbes as a screening test for carcinogenicity, and that is imperfect. And cell growths and cell culture, and that is imperfect. Therefore it puts us in a difficult position of trying to look for accidental or inadvertent or other kinds of administration and then drawing data from that on some kind of epidemiological basis.

The fact is, however, there is no question in any of our minds that that agent has been used for 40 years has been used on the west coast extensively, it is still used in certain crop-dusting applications, and is less restricted in the United Kingdom than it is here. And so we—it is hard to reconcile then the fact that there is no clustering or unusual incidents of soft-tissue sarcoma or stillbirths or what have you in the populations of the west coast, particularly the large valley that has been treated for 30 years with this stuff.

Mr. **Molinari**. I am sure the good chairman has a number of questions, and I am going to abbreviate mine.

I would just like to close by asking this one question.

Dr. **Beljan**. Yes, sir.

Mr. **Molinari**. Taking your own statements that TCDD, by itself, is extremely toxic, along with the others you mentioned: that it is one of the most lethal organic chemical compounds known to man; and couple those with the reality that there is a community like Times Beach that is exposed to large quantities of this material and many of the residents have chosen to flee. Would that reaction really be characterized as an irrational reaction?

Dr. BELJAN. I do not think that that is irrational at all. I think that is a normal human reaction to fear and fear of the unknown and fear of a variety of things.

Mr. MOLINARI. What was the irrational reaction you referred to in your earlier testimony?

Dr. BELJAN. Is that in the resolution? It is again a preventive recommendation, it said that the American Medical Association institute an active public information campaign to get accurate information on dioxin before the public to prevent irrational reaction.

Mr. MOLINARI. That is the problem I have, though. We may be visiting some of those areas. If somebody came to the AMA and said we just learned there is a report that came out today that our neighborhood is loaded with whatever form of dioxin, and we all want to move, what shall we do?

What would you counsel them to do? We do get questions like that. You might have heard my comments before.

Dr. BELJAN. Sure, I understand.

Mr. MOLINARI. What would be your reaction to the people asking you that?

Dr. BELJAN. I think that I need to have more facts than are currently available to make that kind of judgment. If you would ask me the question, would I live there—because I heard you ask that previously of witnesses—that is a very difficult question to answer because I for one am comfortable with the Missouri environment, but I would have to respond to a variety of other kinds of criteria.

Mr. MOLINARI. Let me ask you a tougher question. Would you let your family live there?

Dr. BELJAN. That is the point I was coming to. If I had kids that would grow up in the dirt and eat the dirt, probably not. On the other hand, my last teenager is leaving the nest, that is a different lifestyle and different situation, and I do not think my wife and I are going to be eating soil, or in a position to be doing that. That is a totally different kind of situation.

So again it is hard to give you a general reaction to that kind of question. I can understand the public's concern. I can understand their worry about being in an area in which there is heavy dioxin exposure, and yet the documented public literature would indicate that we do not have the evidence that says that. So that does not resolve your problem because your problem is one, also, of public perception and public concern and those are as important to you as legislators as the scientific data, obviously, would be to the medical science. That does not say we are not concerned.

Mr. MOLINARI. I am sure of that.

Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you.

I am going to be very brief because I do want to save time for Dr. Rowland. Let me ask you this and it follows up precisely on what Mr. Molinari was saying. I think the medical profession occupies a very special role in this entire subject area; and it occupies a very special role to the public generally, and to policymakers such as ourselves.

That role, while it is one that certainly should try to avoid unnecessary fear in the public, also, if it has to err, again should err on the side of protecting the public from undue exposure to potential hazards.

For that reason—and I am not trying to place any judgment—but I feel that the resolution that was adopted by the house of delegates of the AMA really does a disservice to the public because it comes from the medical profession, the organized medical profession.

I think it is a perfectly rational thing if a tank car containing TCDD broke down in my neighborhood, I do not have any hesitancy whatever to tell you what I would do, I would get the hell out of there, my wife, my family, everybody, get them out of there, because we make decisions based upon potential risk, and if somebody told me that one of the most lethal organic compounds known to man has just been spilled on the streets of my neighborhood I think I would have to really be nuts not to get out of there.

But here is my question to you——

Dr. BELJAN. Let me ask you the question, how long would you stay out of there?

Mr. LEVITAS. Probably until somebody assured me—if I were going to look for scientific certainty, it would be at that end. It would be when I make the decision to go back.

Dr. BELJAN. I agree. I would be with you. I would be moving out of there too with that kind of spill.

Mr. LEVITAS. I would be more inclined to move out on the basis of reasonable likelihood and more likely to expect scientific certainty to move back in. That is just speaking for myself. But that gets right to my question.

In prudent policy action, whether it be legislative action or medical action, is it not frequently the case that action is taken on something far less than scientific certainty? Based on reasonable likelihood, for example?

Dr. BELJAN. Absolutely, but I would like to differentiate action from what is fact, and those are very important concepts for a physician.

There is responsibility in other professions, too, and maybe that was part of the resolution. It is very difficult for a mother with a youngster to look at someone with a child and have an allegation made of potential problems, or that kind of anecdotal thing. How much balancing was done in the presentation of that information? Damn little, sir. That was a concern of our Missouri colleagues.

I would agree that there are many decisions in the public policy area that have to transcend fact alone but understand that our responsibility in the Council on Scientific Affairs is to deal with facts and then to adjust that fact as appropriate for the public situation.

Mr. LEVITAS. I am confident you will do that in your committee and your committee will do that and this recent report we have referred to that came out last Friday, I think it was, Thursday or Friday, by NIOSH or somebody will contribute to it. But Dr. Rowland for the last week has continually proudly made disclaimers by saying "I am not a lawyer" and then proceeded to pontificate on legal matters, which is his privilege to do.

Well, I am not a physician, but I suggest that based on my experiences with physicians that there are treatments administered every day where the treatment that the physician executes does not have very scientific certainty; that is that the particular treatment will treat or remedy the ailment involved, but it is taking the action based upon "What can I do to maximize the health of this individual?" People who are suffering from some range of diseases, for example, may get treated with broad spectrums of antibiotics and maybe it will hit someplace along the line. But you do something. You just do not sit back and wait until you have an absolute diagnosis of what the disease is before you treat it.

Dr. **Beljan**. That, sir, is the art of medicine, and there is art in science and in both of our professions.

Mr. **Levitas**. Thank you.

Mr. Rowland.

Mr. **Rowland**. I know you have to catch a plane and I would like to talk with you and ask a lot more questions, but I would first like to make this comment. I do have a problem with the word "irrational" in the resolution. If we are referring to the Times Beach situation, the EPA told these people it is a bad situation. They moved out based on that. I do not think their moving out was irrational. I do have a little problem with that word if it was used in that context.

What this hearing on victim compensation law is talking about is the possibility of creating a whole new area of toxic tort law which is relatively new to the trial attorneys, who have said so. I have been concerned about the developing of an area that will be mated more to the benefit of the trial lawyers than it will be to the people injured. You have been listening to some of the testimony here in the hearings?

Dr. **Beljan**. Yes, sir.

Mr. **Rowland**. Would you briefly give us your feelings on that? I know in the area of medical malpractice that the premiums for malpractice have just absolutely gone out of sight and a lot of that has been on the basis of frivolous suits that have not ever gotten to court. I am just concerned that we may be opening up another entire area. Would you briefly respond to that?

Dr. **Beljan**. Well, sir, I do not believe that the AMA has developed a policy related to this, so I do not want to speak for the AMA. There is certainly AMA policy that says if there has been injury there ought to be fair compensation.

I guess I would not like to speculate, but I do share personally a concern about spawning of excessive lawsuits and things of that sort, but there is no firm AMA policy regarding this specific area of new toxic tort legislation, and I think it is one that needs to be addressed and hopefully we can come back with a recommendation regarding that.

Mr. **Rowland**. Thank you both very much for being here. Thank you, Mr. Chairman.

Mr. **Levitas**. Thank you, Dr. Rowland.

Thank you, Dr. Beljan, and Mr. Peterson, you have been extremely helpful to us in what we are attempting to do, and I want to express my thanks to you for your attendance today. You can catch your plane now.

Dr. **Beljan**. Thank you very much.

Mr. **Levitas**. I would like also to express my appreciation to the witnesses who over the last several days have given us the benefit of their thinking. During our hearings in these last few days on victims compensation, we have heard from citizens who believe they have been affected by the presence of hazardous waste in the environment, and we have heard from those in the chemical industry, the legal community, the medical and scientific community. And today we heard from the environmental community and insurance industry, as well as the American Medical Association. Obviously that which we have heard has not exhausted the subject matter, nor have we necessarily heard from all the different groups and affected parties. Further, as we expected, our hearings did not resolve the controversies that surround the area of human health effects, nor did we eliminate the scientific or medical barriers to establishing cause-effect linkages between hazardous waste and human health. The subcommittee intends to review the record of the past several days very carefully as it considers how we should proceed in the question of victim compensation.

Among the things we intend to consider is the possibility of a few additional days of hearings so that we may hear from others in the medical community, such as people from the centers for disease control, Dr. Rowland, which you mentioned on a couple occasions; and from State officials, particularly those that have already taken some steps along the lines that Congress is contemplating which could include California, as well as Minnesota officials.

Further, with respect to making the record of these hearings as complete as possible, the hearing record will remain open for 3 weeks without objection to enable any other interested parties who may wish to submit comments to do so.

I have already announced some of our tentative plans for future hearings in the whole matter of hazardous waste cleanup, Superfund and the like, and we will announce in the next several days the schedule of hearings for the month of August and the weeks following after we return from the congressional recess. But there will be hearings scheduled during the district work period in August. Again, with my appreciation to the witnesses who have testified during this series of these hearings, I find no further business before the subcommittee and, therefore, the subcommittee stands in recess subject to the call of the Chair.

[Whereupon, at 3:35 p.m., the subcommittee adjourned, subject to the call of the Chair.]

HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

TUESDAY, NOVEMBER 8, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 10 a.m., in room 2167, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Mr. Levitas. The subcommittee will please come to order.

This morning, the Subcommittee on Investigations and Oversight of the Committee on Public Works and Transportation continues its inquiry into the matter of hazardous waste contamination of the Nation's water resources, and as part of that inquiry, an examination of the problems that individuals have in obtaining compensation for the health and financial problems they face due to the presence of toxic wastes in their environment.

Earlier this year, in July, the subcommittee held several days of hearings on the subject, at which time we heard from a number of individuals—lawyers, doctors, EPA scientists, environmentalists, representatives of the chemical industry, and some victims as well.

The testimony that we heard clearly pointed out that there are uncertainties about the linkages between toxic wastes and human health problems. It also illustrated that there are limits in our ability to resolve absolutely these questions due to medical and scientific factors.

We also heard differing views on the adequacies and inadequacies of existing remedial or compensatory mechanisms, be they in the courts, workman's compensation, or the industry.

And finally, we heard differing views about the changes that many believe are needed. Some have argued that nothing need be done; others, that changes in either or both Federal and State laws are needed.

Indeed, it seems that the testimony revealed that the victim compensation issue is multifaceted, with perhaps as many considerations to be dealt with as there are toxic chemical wastes.

However, the testimony we have heard to date also clearly shows that there are individuals who live near or adjacent to hazardous waste sites that have serious health problems which they and others believe are associated with the hazardous wastes at those sites.

In a few cases, some of these individuals are getting compensation for their problems through the courts. But, it appears that the vast majority of people who believe they have health problems that are caused by hazardous wastes are not being compensated.

In part, this appears to reflect shortcomings not only in our medical and scientific capabilities, but in our laws as well. Indeed, it would appear that the system itself is in large part an impediment to the compensation of victims of hazardous waste pollution.

Under the Superfund law, we, as a Nation, will compensate for damages to our natural resources, but we will not compensate those who believe their very well-being has been impaired due to exposure to toxic chemical wastes. In short, we will pay for damage to trees but not for damage to people.

Certainly, it would seem that some change is in order. There remain, however, questions about just what should be done, and whether it should be done at the Federal level, or at the State level. I suspect that there will be a need for changes at both levels, and we look forward to hearing from today's witnesses who are particularly well qualified to enlighten us on these matters.

Before I invite the witnesses, I would inquire whether my good friend, the gentleman from Florida, Mr. Shaw, has an opening statement.

Mr. SHAW. Yes, I do, Mr. Chairman. I would first of all want to express my appreciation. I am sitting here in place of the ranking member, Mr. Molinari, who is on assignment for the President at the present time.

I also want to extend a cordial welcome to our witnesses. It is a rare opportunity for us to be able to engage in a frank exchange of views on the record with our counterparts from the State legislatures. I trust their experiences with toxics tort victim compensation will prove particularly enlightening.

I am pleased also that the Assistant Attorney General, Paul McGrath, will be appearing before the subcommittee today. Among other things I would hope his testimony would enable us to better understand why the administration has chosen to elevate the victim compensation issue to Cabinet-level policymaking, without the hyperbole I have recently seen in reaction to an OMB briefing memo which was selectively leaked some months ago.

Mr. Chairman, I want to commend you for initiating and now continuing the subcommittee's hearings on the victim compensation issue. I know I speak as well for the gentleman from New York, the ranking minority member—Mr. Molinari—in expressing their thoughts.

Victim compensation has, I suspect, proven to be a more complex issue than many of us considered when the subcommittee began its inquiry last July. I have been quite impressed by the testimony of the witnesses I have heard to date. Their views, although sometimes directly contradictory, have enabled the subcommittee to build a substantial record on the subject.

The testimony we have previously heard, together with that we will receive today and tomorrow, perhaps gives us an indication of why it was so difficult and ultimately impossible for the Congress to include victim compensation provisions in the 1980 enacted Superfund legislation. To my knowledge, an extensive record on the issues simply did not exist at that time. It seems to me that this subcommittee hearing, together with those of other committees with jurisdiction over the subject matter, will better enable the Congress to fashion well considered remedial legislation appropriate to the scope of the problem.

With that by way of introduction, Mr. Chairman, I again thank you for scheduling these hearings and look forward to a productive 2 days of hearings.

Thank you.

Mr. LEVITAS. Thank you, Mr. Shaw. Thank you for your comments.

Let me say that our colleague, the ranking member on this committee, Mr. Molinari, who, as you said, was unavoidably not able to be here today, carrying out a mission at the request of the President, truly has taken the lead in this entire matter of hazardous waste. I think it is no understatement to say had it not been for his persistence and urgings, the hearings we began 2 years ago would not have been undertaken. I know he wanted to be here, and I know he appreciates your filling in for him. He told me he had spoken to you about this, and I appreciate your participation.

Does any other member of the subcommittee have an opening statement to make at this time? If not, we are very pleased to have a colleague of ours with us today, a member of the full committee, who for a variety of reasons has played a very key role in our investigation and the whole subject of dealing with toxic wastes. In fact, yesterday, Bob, at a meeting in Memphis which was chaired by our colleague Don Sundquist, you had representation there. I know you wanted to be there, and your name was invoked on several occasions. I am sure that your interest preceded and extends beyond the immediate fact that you happen to represent the district in which Times Beach lies, but without doubt that has given you a special insight, as the citizens and witnesses from Times Beach and Imperial who have already testified before our subcommittee so poignantly demonstrated in their testimony before us. So we are glad to have you back with us today. You may proceed as you so desire.

TESTIMONY OF HON. ROBERT A. YOUNG, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF MISSOURI

Mr. YOUNG. Mr. Chairman, because I want to move through the statement as fast as I possibly can, I don't want to forget to invite you out to the Metropolitan St. Louis area if you feel the subcommittee would be able to do that. I think it would be very interesting.

I would like to take this time to thank you for allowing me to appear before you today and share with you some of the very startling facts about the facts of hazardous waste. I also want to commend you for holding these hearings, because I feel the area of victim compensation due to hazardous waste contamination is an issue which will continue to plague America well into the next century.

I hardly consider myself an expert on the plight of victims. I honestly believe that I do have a contribution to make as I have grown all too familiar with the burdensome ordeal of reconciling one's life after such a catastrophic incident. My congressional district contains several dioxin-contaminated sites, as well as the famous—infamous Times Beach. Over the last year I have had to head the battle for Federal disaster assistance so that the thousands of residents could be placed in temporary housing, be provided with necessary emergency health care for injuries as well as food and clothing after a devastating flood ravaged the area in December 1982.

However, recovering from any flood is always a monumental task; however, to recover from a major toxic contamination is a nightmare.

Ladies and gentlemen, I am not exaggerating when I tell you that I, along with the residents of Times Beach, worked feverishly for months to convince EPA that the residents were exposed to a deadly chemical dioxin. It was an arduous, sometimes bitter, struggle for us to insist that immediate relocation was the only viable alternative until remedial action, whether on a temporary or permanent basis, could be found.

I am very sorry to sit here before you today and tell you that this was only the beginning. At this very moment we are engaged in these hearings, there are Times Beach residents who are still coping or grappling with the devastation of dioxin. It has been over a year since dioxin was discovered in Times Beach, and it is hard to realize that these people are still resolving fundamentals that seriously affect their lifestyle. I am talking about fundamentals which are crucial—fundamentals like a home, their health, well-being of their families, and their future.

As of October 23, 1983, only 43 properties in Times Beach have been bought out. Before anyone gets excited about the high number, I want to quickly add that is 43 pieces of property out of a possible 970. I can almost predict everyone's comment; and that is, EPA, FEMA, and everyone involved is trying to reconcile these issues as quickly as possible. Well, I can speak with authority when I say that I am aware of the efforts of FEMA and EPA and all the others involved, because I have been working right along with them since day one.

But the facts speak for themselves. I wholeheartedly concur when the headline reads "Missourians Forced Out By Dioxin Are Impatient, Distrustful and Worried," for all of these adverbs apply to me as well. The citizens of Missouri and the Nation have become increasingly aware of the serious health threats to the public health by the discovery of dioxin. However, when I have asked health experts in the field just what the health effects are of dioxin, no one, not one single person, could give me a straight specific answer. All I am told is that "it needs to be studied more" and "we're working on it," things like that.

However, I have noticed that the Center for Disease Control still continues to issue health advisories. I just feel that communication needs to be improved and the facts need to be made available now.

In response to several problems of the dioxin contamination, the Governor's task force has issued a final report calling for a super bunker to be built in Times Beach to house some 500,000 cubic yards of soil, or 50,000 truckloads, of dioxin-contaminated soil. I will make that—Mr. Chairman, I would like to make that report a matter of your committee's record.

Mr. LEVITAS. Without objection so ordered.

Mr. YOUNG. I question this proposal for several reasons. Primarily I am concerned about the structure being located in a flood plain or prone area. There are a myriad of other concerns about the construction of the bunker. There is also the very serious matter of the permit to be issued for the project. Cost estimates have been quoted at $200 million. May I remind you that this is $200 million just for a temporary solution?

My final concern is the soundness of the bunker from an engineering standpoint. Is there a danger of another public health hazard in 5 or 10 years?

During your July hearings, Mr. Chairman, many of the Times Beach residents provided a litany of medical problems and ailments that the citizens have linked directly to dioxin. This may—they are asking this subcommittee to provide some kind of victim compensation for taxpayers exposed to hazardous waste. I am aware of the efforts to include these kinds of provisions in prior legislation, and why, because of certain controversies, why they were not included.

It is not my intention to discuss the differences between acute toxicity as opposed to chronic toxicity and the probability of risk to victims genetically transmitting latent diseases or illnesses including cancers or birth defects. That I will leave for this distinguished panel which will appear before you today. However, it is my intention to encourage the subcommittee to arrive at some answer to these questions and provide Congress a clear direction.

Is it really a matter of our lack of scientific data to make these decisions, or is it simply a matter of cost? Does it cost too much money to find out the health effects? I believe building a super bunker will only defer many of these questions and not solve anything with regard to health risks and proper compensation.

It is clear to me that the scientific/medical community has adhered to the position that we do not have enough information to conclusively determine that persons are suffering as a result of toxic contamination. However, there are literally thousands of people, many in my district, that demonstrate medical problems that are at least connected to exposure of high levels of dioxin.

Lawyers and scientists offer no relief on the subject of how difficult it is to present and prove with sufficient evidence a toxic chemical tort case. The only thing a defense attorney can do is to provide enough negative facts and try to relax requirements for hard evidence in order to clear the procedures for a victim's just compensation.

What the subcommittee needs to determine for the American people is—Mr. Chairman, I have about five different questions here, and I think I will skip them because the other members are behind me.

The question of compensation, the long-term effect. And is this problem of such a magnitude that it requires an immediate Federal response? Mr. Chairman, all I can say is that no one knows how large a problem we are really faced with. What I do know is that the problem is large enough that it dictates a public policy be determined on a higher priority basis. In Missouri alone there are some 230 sites which are under investigation. This is a staggering number and easily demonstrates the need for congressional direction.

In conclusion, I want to stress that it is only through the leadership of this committee that I can envision a strong sense of hope which will enable us to grasp the complexities of the issue of dioxin.

Your efforts to continue to diligently monitor and oversee our Nation's Hazardous Waste Program are sorely needed. They will continue to be instrumental and effective in maintaining a policy that protects the health and safety and welfare of Americans.

I hope my remarks have been of value to you, Mr. Chairman, and the committee. And it is my sincerest wish that no other member should ever be faced with this most serious tragedy.

Mr. Bliss is like Dolly Levi, I think. He just spread that thing all over my district, and it just seems almost an impossible task at this point to try to resolve this very serious health problem. I appreciate the time of the committee, Mr. Chairman.

[The statement of Congressman Robert Young and the "Final Report of the Missouri Dioxin Task Force" follow:]

STATEMENT OF HON. ROBERT A. YOUNG

Mr. Chairman, and members of the subcommittee, I would like to take this time to thank you for allowing me to appear before you today and to share with you some very startling facts about hazards of chemical waste which I believe you will find very pertinent to your mission.

I also want to commend you for holding these hearings because I feel that the area of victims compensation due to hazardous waste contamination is an issue that will continue to plague America well into the next century. I hardly consider myself an expert on the plight of victims who have been shattered by the hazards of chemical waste, I earnestly believe that I do have a contribution to make as I have grown all too familiar with the burdensome ordeal of reconciling one's life after such a catastrophic incident.

My congressional district contains several dioxin-contaminated sites, as well as the infamous Times Beach. Over the last year, I have had to battle for Federal disaster assistance so that the thousands of residents could be placed into temporary housing, be provided with necessary emergency health care for injuries, as well as food and clothing after a devastating flood ravaged the area in December, 1982. Recovering from any flood is always a monumental task, however to recover from a major toxic contamination is a nightmare.

Ladies and gentlemen, I am not exaggerating when I tell you that I, along with the residents of Times Beach, worked feverishly for months to convince EPA that the residents were exposed to a deadly chemical—dioxin. It was an arduous and sometimes bitter struggle for us to insist that immediate relocation was the only viable alternative until remedial action, whether on a temporary or permanent basis, could be found. And I am very sorry to sit before you today and tell you that this was only the beginning.

At this very moment, as we are engaged in these hearings, there are Times Beach residents who are still coping and grappling with the devastation of dioxin. It has been over a year since dioxin was discovered in Times Beach and it is hard to realize that these people are still resolving basic fundamentals that seriously affect their lifestyles. And I am talking about fundamentals which are crucial—fundamentals like a home, their health, the well-being of their families, and their futures. As of October 23, 1983, only 43 properties in Times Beach have been bought out. Before anyone gets excited about the high number. I want to quickly add that that is 43 pieces of property out of a possible 907!

Yes, I can almost predict everyones next comment. And that is "EPA, FEMA, and everyone involved is trying to reconcile the issue as quickly as possible." Well, I can speak with authority when I say that I am aware of the efforts of FEMA, and EPA and all of the others involved because I have been working right along with them since day one. But the facts speak for themselves and it is still a traumatizing, almost hopeless ordeal at times for these residents.

I wholeheartedly concur when a headline reads: Missourians forced out by dioxin are impatient, distrustful and worried. For all of those adverbs apply to me as well.

The citizens of Missouri, and the Nation, have become increasingly aware of the serious health threats to public health and the environment posed by the discovery of dioxin. However, when I have asked experts in the field just what are the health effects from exposure to dioxin—no one, and I mean not one single person could give me a straight, specific answer.

All I am told is that it "needs to be studied more" and "we are working on it," etc. However, I have noticed that the center for disease control still continues to issue health advisories. I just feel that communication needs to be improved and the facts need to be made available—now!

In response to the severe problem of dioxin contamination, the Governor's task force has issued a final report calling for a "super bunker" to be built in Times Beach to house some 500,000 cubic yards of soil, or 50,000 truckloads of dioxin contaminated soil.

I question the proposal for several reasons.

Primarily, I am concerned about the structure like this being located in a flood plain or flood prone area. And there are a myriad of other concerns such as the construction of the bunker, again, the health and safety of the public, cost and timing factors, environmental concerns and public reaction. There is also the very serious matter of the permit to be issued for this project. Cost estimates have been quoted at 200 million—may I remind you that this is 200 million dollars for a temporary solution. My final concern is the soundness of the bunker from an engineering standpoint—is there the danger of another public health hazard in 5 or 10 years?

Mr. Chairman, during your July hearings on this subject, the mayor of Times Beach provided a litany of medical problems and ailments that the citizens had linked directly to dioxin. The mayor was asking this subcommittee to provide some kind of victims compensation for taxpayers exposed to hazardous waste. I am aware of the congressional efforts to include these kinds of provision in prior legislation, and why, because of certain controversies, why they were not included.

It is not my intent to discuss the difference between acute toxicity as opposed to chronic toxicity, and the probability of risks of victims genetically transmitting long latency diseases or ailments including cancers, or birth defects, or genetic mutation. That, I will leave for the distinguished panel which will appear before you tomorrow. However, it is my intention to encourage the subcommittee to arrive at some answers to these questions and provide the Congress with a clear direction on what is proper justice for victims that have suffered from toxic waste exposure.

Is it really a matter of our lack of scientific data to make these decisions, or is it a matter of cost—does it simply cost too much money to find out the health effects: I do believe that building a super bunker will only defer many of these questions and not solve anything in regards to health risks and proper compensation.

It is clear to me that the scientific/medical community has adhered to the position that we do not have enough information to conclusively determine that persons are suffering as a result of toxic contamination. However, there are literally thousands of people, many in my district, that demonstrate medical problems that are at least connected to exposure of high levels of dioxin. Lawyers and scientists offer no relief on the subject of how difficult it is to present and prove, with sufficient evidence, a toxic chemical tort case. The only thing a defense attorney can do is to provide enough negiative facts and try to relax the requirements for hard evidence in order to clear the procudures for a victim's just compensation.

What this subcommittee needs to determine for the American people is:

1. Are victims having extreme hardships in receiving compensation for their injuries?

2. What are the scientific and medical requirements associated with demonstrating a cause-and-effect of exposure to dioxin and the injuries from that exposure?

3. What are the institutional and legal barriers that are currently preventing or hindering victims compensation?

4. Is there a problem of such magnitude and urgency that it requires an immediate Federal response?

5. If a Federal response is needed, what is the appropriate and judicious response we in Congress can provide.

Mr. Chairman, all I can say is that no one knows how large a problem we are really faced with. What I do know is that the problem is large enough that it dictates that a public policy be determined on a high priority basis.

In Missouri alone, there are some 230 sites which are under investigation or which have been investigated for dioxin. Those staggering numbers should easily demonstrate the need for Congress' direction.

In conclusion, I want to stress that it is only through the leadership of this committee that I can envision a strong sense of hope which will enable us to grasp the complexities of the issue of dioxin.

Your efforts to continue to diligently monitor and oversee our Nation's hazardous waste program are solely needed. They will continue to be instrumental and effective in maintaining a policy that protects the health, safety and welfare of Americans.

I hope my remarks have been of value to you. And it is my sincerest wish that no other Member of Congress should ever be faced with this most serious tragedy.

FINAL REPORT OF THE MISSOURI DIOXIN TASK FORCE

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submitted to
Governor Christopher S. Bond

OCTOBER 31, 1983

FINAL REPORT OF THE
MISSOURI DIOXIN TASK FORCE

SUBMITTED TO
GOVERNOR CHRISTOPHER S. BOND

October 31, 1983

October 31, 1983

Honorable Christopher S. Bond
Governor
State of Missouri
State Capitol
Jefferson City, MO 65101

Dear Governor Bond:

I hereby convey to you the final report of the Dioxin Task Force as requested in your executive orders 83-5 and 83-13.

In the eight months since its inception in February of this year, the Task Force has studied the volume of information on dioxin in the literature and as presented by experts in the field. The recommendations within this report reflect that study.

Please accept this report as adopted unanimously by the Dioxin Task Force.

Respectfully submitted,

James A Finch Jr

James A. Finch, Jr.
Chairman
Governor's Dioxin Task Force

JAF:brd

Definition of Dioxin

This report deals with one extremely toxic member of a class of compounds commonly called "dioxins". Within the 75 possible chlorinated dioxins, there are 22 isomers of tetrachlorodibenzo-p-dioxin (TCDD). In Missouri, the contamination is almost exclusively with one highly toxic member of this family, the member "2,3,7,8-tetrachlorodibenzo-p-dioxin". For simplicity and unless otherwise stated, this report uses the terms "2,3,7,8-TCDD", "TCDD", and "dioxin" interchangeably, all referring to one member of a family of compounds.

INTRODUCTION

On February 14, 1983, Governor Bond, by Executive Order, established the Governor's Task Force on Dioxin. It will be dissolved upon submission of this Final Report.

The Executive Order forming this Task Force included a charge to: "examine and evaluate all known methods, processes and technologies for the destruction and containment of dioxin, particularly as it exists as a contaminant in soil and sediment at various locations in Missouri;" and to "examine and evaluate the public health dangers associated with dioxin, particularly with respect to dioxin as it exists as a contaminant in soil and stream sediment at various locations in Missouri." The Task Force was then charged to "recommend a practical and effective plan of action for implementing comprehensive and permanent solutions to the public health and environmental problems caused by dioxin contamination in Missouri." The Executive Order mandated an interim report concerning the site at Times Beach as well as this final report.

In fulfilling the charges to examine public health and environmental problems and to examine and evaluate methods, processes and technologies the Task Force has examined extensive scientific literature and has heard testimony by a wide spectrum of experts from universities, governmental agencies, industries, and the public. It has heard over 50 hours of expert testimony and been provided with 10,000 pages of reports, scientific documents, and other related reading materials. In addition, in order to gain a better understanding of the Missouri problem, the Task Force made site visits to the St. Louis and Verona areas.

On June 1, 1983, the Task Force submitted its Interim Report to Governor Bond. It represented the preliminary opinions of the Task Force concerning immediate, yet temporary, remedial measures to be applied at Times Beach. In addition, the Report addressed the Minker and Stout sites because the Task Force believed there was sufficient urgency for interim actions at those sites. The report included important background materials and assessments of proposed dioxin destruction and remedial technologies.

This Final Report addresses the Governor's directions by giving an assessment of health effects based on currently available knowledge and by recommending remedial actions for all known sites, plus suggesting future research and study. In developing these recommendations careful consideration was given to the psychological and economic impact of the dioxin problem and proposed solutions on affected Missouri citizens.

The report includes additional background materials and acknowledgement of all experts who have been heard. These are attached as appendices. Transcripts of testimony heard and of proceedings of the Task Force meetings are available through the office of the Department of Natural Resources.

The Task Force wishes to thank the Directors of the Department of Natural Resources (DNR) and the Division of Health (DOH) and their staff members for their excellent cooperation in obtaining and providing literature, for arranging for witnesses whom the Task Force desired to hear, and for providing necessary staff work.

Based on our evaluation of all the testimony and data received, the Task Force recommends the following: (For explanation and amplification of these recommendations see the Section entitled Rationale for Recommendations).

RECOMMENDATIONS

I. The Task Force recommends that secure central storage of Missouri's soil contaminated with dioxin exceeding acceptable limits should be provided until proven technology is available to assure a comprehensive and permanent solution to dioxin contamination with minimum risk to public health and the environment.

II. The Task Force recommends that health studies on Missouri citizens potentially exposed to dioxin in residential, manufacturing or other occupational settings should be continued and expanded to assess the long-term public health effects due to dioxin.

III. The Task Force recommends that the Missouri Dioxin Strategy for secure storage of dioxin contaminated soils and for assessment of health effects, as stated in this report, be adopted and thereafter periodically updated. The Strategy includes the following components and provisions:

A. The Missouri Dioxin Strategy must include all sites regardless of CERCLA status.

B. High priority should be given to development and publication of an expedited schedule for sampling and analysis at all suspected sites, and for extensive grid sampling at known sites.

C. High priority should be given to the siting for and construction of a central storage facility and interim storage as required.

D. The central storage facility should be utilized only for dioxin contaminated solids.

E. A primary aim should be the reconstruction and reinhabitation of all sites.

F. The state should establish criteria and follow a uniform policy regarding residents and owners of dioxin sites.

G. The experience gained at Seveso, Italy, should be utilized to the extent possible.

H. The residual level of dioxin allowed to remain after excavation and decontamination must be specified.

I. The State should begin immediately to communicate the criteria for safe transportation of dioxin contaminated soils on Missouri roads and highways to address public concern.

J. A Missouri Program in Toxicology should be established.

K. An advisory panel, similar in composition to this Task Force, but including two dioxin site resident representatives, should be formed to advise the Director of DNR.

HEALTH RISKS

In order to responsibly assess the possible remedial actions, it is necessary to understand the public health dangers associated with dioxin. For purposes of this report, the health effects of dioxin are classified as acute, chronic and carcinogenic. There is some overlapping of these areas which means that the symptom-complex interrelationships also must be examined. The following is a brief summary of each of these areas:

1. Acute Toxicity. The acute toxicity of relatively large doses of dioxin incident to industrial accidents is well documented. Chloracne appears within several weeks and may persist for many years. It is disfiguring and may produce psychological stress. Liver disease may also be found but is not consistently evident in man. Porphyria cutanea tarda is another condition which may be produced by exposure.

Cell mediated immunity is decreased by dioxin in mice. There is minimal evidence on whether dioxin exposure causes immunosuppression in humans. In a study of 45 children from zone A, the area of highest dioxin concentration, of Seveso, Italy, Pocchiari, et. al. showed in four repeated examinations over the first year following the accident that there were no immunological differences between the exposed children (21 of which had chloracne) and a minimally exposed comparison population.

Dioxin is a potent inducer of aryl hydrocarbon hydroxylase (AHH) and at least a dozen other enzymes in animals.

2. Chronic Effects. The most obvious chronic effect is persistent chloracne. Other effects such as porphyria cutanea tarda, liver dysfunction, and functional changes in kidneys, pancreas, blood and lipid metabolism, have been reported following accidental exposure to dioxin and related compounds but it is difficult to determine whether the signs and symptoms observed were due to dioxin or other chemicals involved.

Neuropathologic findings including nerve conduction defects and demyelinization of nerves, particularly in the lower limbs, have been demonstrated. Numerous neuropsychiatric symptoms such as fatigue, depression and headache have been described, but cause and effect relationships with dioxin are difficult to validate with present methods.

Reproductive effects of dioxin such as fetotoxicity and teratogenesis have been shown in a number of animal species. However, human epidemiologic studies in Germany, the United States, Australia, and at Seveso have shown no apparent derangements of gestation, no fetal loss, no gross malformations and no cytogenetic abnormalities. A study in Oregon alleging an increase in abortions among exposed women was subsequently declared valueless by reviewers from Oregon State University and from Australia, New Zealand and the United States government. A North Vietnamese study alleging increases in birth defects in the children of exposed Vietnamese soldiers cannot be evaluated because the study itself is not available.

3. Carcinogenesis. Probably the most important and yet the least understood possible effect of dioxin is carcinogenicity. It is generally agreed that dioxin is an inducer of cancer in rats and mice. Therefore, it has a potential for causing cancer in humans. It is unnecessary, for the purposes of this report, to decide whether dioxin might be a cancer initiator, a cancer promoter, or whether its action is exerted through enzyme or cellular immunity changes.

Two methodological problems in the assessment of the possible carcinogenic action of dioxin are the long "latent period" of most cancers and the small numbers of exposed individuals from industrial accidents that occurred more than twenty years ago.

Studies in Sweden and testimony before the U.S. Congress by the Director of the NIOSH Division of Surveillance, Hazard Evaluations, and Field Studies have indicated a higher risk of soft tissue sarcomas among those workers exposed to dioxin and the phenoxy acids. The question of an increase in cancer rates among Vietnamese is not fully resolved. A study of participants in "Operation Ranch Hand" of the Air Force has not implicated dioxin in carcinogenesis nor have the studies in Finland and New Zealand. Definitive results from the Air Force study cannot be expected until a sufficient period of time has passed to rule out or implicate dioxin as a carcinogen among those exposed to "Agent Orange".

An EPA panel of experts in July 1983 prepared a draft report after considering all available evidence which stated dioxin must be considered a probable human carcinogen.

4. Dioxin Symptom-Complex Interrelationships. It is customary, from the clinical point of view, to describe a number of symptoms related to an infection or an environmental exposure as a symptom-complex with various stages of the disease. In the case of dioxin exposure there are those who would insist that chloracne is the "stage one" or hallmark of all subsequent chronic or carcinogenic effects and that without chloracne, no subsequent effects would be expected. However, considering the various routes of exposure, the degree of exposure, and the shortness of the time since such exposures occurred, it is impossible to state unequivocally that chloracne is a prerequisite for dioxin intoxication.

There is some published evidence that soft tissue sarcomas may occur in persons exposed to dioxin who had no preceding chloracne. The question of other adverse health effects such as nerve conduction defects and reproductive problems occurring without prior chloracne is not resolved. However, there is certainly enough concern about this issue to warrant continued investigation. An interrelated problem requiring further research is the reported background adipose tissue levels of dioxin among the "normal" population which varies from 0.007 to 0.020 ppb. These background levels have been established in studies by the U. S. Air Force, Veterans Administration, and Canadian government. Therefore, it is possible that there are other sources of dioxin in the environment. Such sources may include incineration and chemical derivatives of the trichlorophenol synthesis such as hexachlorophene and 2,4,5-T. The possible sources are not yet well defined.

MISSOURI HUMAN HEALTH RISK ASSESSMENT

The problem of dioxin in Missouri is that the potential for exposure of the affected population consists of protracted contact with soils contaminated by spraying with a mixture of oil and dioxin more than ten years ago. The only evidence presented to the Task Force of any acute human effects occurred at a horse arena where the level of dioxin was measured at 32,000 ppb - a level much higher than that measured at any of the residential sites. The fact that horses, pets, and birds died immediately following the road and horse arena spraying may be due either to species differences in sensitivity to dioxin in these animals, or to a high dosage and close contact with the sprayed material, the soil and the dust, or to other unknown contaminants in the sprayed material.

For most Missourians the likely exposure has been limited to dioxin in the soil, the dust emanating from the soil, and the erosion and mobilization of the soil as sediment in surface waters and the resulting contaminated fish. There is tight binding of dioxin to soil particles and it has low solubility and vapor pressure which reduces its solubilization and percolation into ground waters or vaporization into the atmosphere. Therefore, the risk assessment in Missouri is confined largely to the possible chronic and carcinogenic effect of low dosage exposure to dioxin in soil over a period of many years.

Bioaccumulation of dioxin in fish from contaminated streams has been shown in Missouri. It is believed that there is limited uptake into food plants. Therefore, the human food chain may be affected.

It must be cautioned that a complete assessment of the health risk at this time is not possible for the following reasons:

1. Recent health studies undertaken by the Missouri Division of Health and NIOSH are not complete. These reports when submitted must be carefully examined.

2. Further study is needed concerning the rate of absorption of dioxin from contaminated soils. Bioavailability of dioxin may be limited by soil binding which appears to increase over time.

3. The soil sampling procedures used by EPA to date have primarily been designed to detect contaminated areas and not to evaluate habitant exposure.

4. Much of the available information of health effects has come from exposure at industrial accidents where the level of the actual exposure was largely unknown and the route of exposure was quite different from soil contact.

5. The knowledge of background levels of dioxins in humans is inadequate at present.

6. The actual past dioxin exposure histories of Missouri residents are only gross estimates. Exact determination of an individual's exposure to dioxin is extremely complex and currently can only be estimated. In the future

it is expected that tests to measure tissue and blood levels of dioxin will be available and make human risk assessment more precise and simpler. For the present, using worst case assumptions, experts from the Centers for Disease Control have decided that a concentration of 1 part per billion in composite soil samples should be considered the upper limit of safe exposure in residential settings. It is important to note that children would have the most exposure to soil and dirt by ingestion and would probably receive the greatest dose per body weight. In adults with less soil contact the risk could be significantly less. Continuous and extended exposure to this soil level would be expected to increase a person's risk of cancer by one in one million in six to seven years. A review of this standard is being conducted by the Centers for Disease Control. A tentatively proposed maximum dioxin level in water is at the parts per quintillion (10 raised to the minus eighteenth power) level, based on the biologic concentrating capacity of aquatic organisms which may be found in the human food chain. It should be added that such levels cannot yet be analytically determined.

For the present, therefore, we have no choice but to use the limit of 1 ppb of dioxin in residential soils as the upper limit of allowable soil concentration, with the resulting stream sediment, water contamination and food chain content being some smaller concentration.

HEALTH CONCLUSIONS

The Task Force reaches the following conclusions on the health effects of dioxin exposure:

1. Dioxin is acutely and chronically toxic to humans as demonstrated in accidental large, but unknown dosage exposures.

2. Dioxin may be absorbed in the human by skin contact, by inhalation, and by ingestion.

3. In Missouri, the potential dosage exposure is complicated by the tight soil binding proclivity of the compound, the long-term (more than 10 years) exposure to soil levels ranging from one to more than one thousand parts per billion, and the variations in exposure to the contaminated soils due to differences in age, occupation, and living environment.

4. The review group convened in July 1983 by EPA and the Director of the Division of Surveillance, Hazard Evaluation, and Field Studies of NIOSH of the USPHS have characterized dioxin as a probable carcinogen.

5. Present risk assessment procedures based upon the reproductive and carcinogenic effect appear to suggest a maximum safe level of 1 ppb in residential soils of Missouri. Levels in water and human food chain vegetation, mammals and fish may be required. CDC and EPA are encouraged to establish standards for allowable levels of dioxin as soon as possible.

6. Little is presently known about: adipose tissue levels of dioxin in "normal" or unexposed individuals in comparison to those with known exposure; the absorption and metabolism of dioxin and related compounds; and the long-term effects of various dosages, entry into the human, and the biological availability of the compound in the various soil, water and air conditions in which dioxin is now being found.

7. Human contact and exposure to contaminated soil should be minimized.

8. Sufficient funding should be provided for appropriate follow-up studies to be conducted on the exposed populations.

9. Careful consideration should be given to dealing with the emotional stress of living in and near contaminated areas. Effective methods should be developed to consistently advise and counsel residents of contaminated areas.

10. There should be continuation of health education for the general public about dioxin. There should be continual review of all research concerning dioxin. Uncertainty about the health effects of exposure to dioxin has caused fear and psychological and emotional stress. Health education techniques, including the dioxin hotline, are essential to the well being and rational activity of our citizens.

11. There is no human health benefit attributable to dioxin which is usually produced as an unwanted by-product in chemical manufacturing processes.

A primary concern of the Task Force was the determination whether the dioxin contamination of the soils of various Missouri communities since 1970-71 required only continued surveillance of the health of the individuals exposed or whether the past decade of such soil-dioxin exposure had been, and continues to represent, a sufficient health and environmental hazard to require remedial action. Weighing the testimony given, and other information made available to us, it is the conclusion of the Task Force that the dioxin contaminated soil in Missouri does constitute a hazard to the health of citizens exposed to such contaminated soil. The degree of the hazard is not presently determinable; therefore, deliberate, prudent remedial action should be taken to eliminate future exposure and health studies should be continued and expanded.

RATIONALE FOR RECOMMENDATIONS

I. The Task Force recommends that secure central storage of Missouri's soil contaminated with dioxin exceeding acceptable limits should be provided until proven technology is available to assure a comprehensive and permanent solution to dioxin contamination with minimum risk to public health and the environment.

After considering all of the scientific literature and testimony provided, the Task Force concludes that a permanent solution to the dioxin contamination in Missouri cannot be recommended at this time because there is no proven technology for the safe destruction of dioxin in soils. Secure storage should be provided until destruction technology becomes available.

Secure storage will require about five years to accomplish. Based on the Seveso experience with excavation and storage, costs could be about $70.00 per cubic yard. However, after considering all available information on remedial technologies, health effects, and impact on Missouri and its citizens, the Task Force believes that this action must now be taken.

In the Task Force's interim report it was concluded that no proven technology then existed for the destruction of dioxin. (See Chapter 6 of that report for an assessment of available technologies.) The Task Force now concludes that adequately proven technologies for the destruction of dioxin in soils will not be available for several years.

Incineration is probably the nearest to being a proven technology, but it appears that perhaps five years of laboratory and EPA permitted pilot studies would be required before construction of an incinerator could begin. Also, it is not clear that an exemption could be obtained from the EPA requirement that residues from destruction of acutely hazardous wastes must be stored in a facility meeting the same requirements as the material before destruction. Since the incineration of soil would not drastically change its volume, a storage facility, as recommended above, might still be required. Incineration, then, is not a short term option, and all other technologies appear less likely to be applicable within the next decade.

The Task Force also concludes that technologies which require extensive processing, such as solvent extraction, are inappropriate at this time. These approaches create more waste than originally existed, are inherently expensive, and present considerable risk of environmental release and human exposure.

II. The Task Force recommends that health studies on Missouri citizens potentially exposed to dioxin in residential, manufacturing, or other occupational settings should be continued and expanded to assess the long-term public health effects due to dioxin.

The Task Force recommends that health studies should be expanded to include all identifiable Missouri citizens who potentially have been directly exposed to dioxin contaminated materials. In addition, Missouri should participate in studies similar to those recently reported on the dioxin content of human adipose tissue to assist in determining background levels due to other exposures.

III. The Task Force recommends that the Missouri Dioxin Strategy for secure storage of dioxin contaminated soils and for assessment of health effects, as stated in this report, be adopted, and thereafter periodically updated.

The widespread distribution of dioxin in Missouri is an unfortunate reality which has presented serious health concerns and a serious social and economic problem to the State and to many Missouri citizens. Lacking a technology for destruction of dioxin contaminated soil, a definite end to potential exposure of citizens and the environment should be provided by secure storage which would permit return of the affected areas to productive use.

Of particular concern to the Task Force is the public perception of inactivity followed by abrupt actions. Therefore, it is recommended that this strategy for secure central storage, as herein outlined and recommended, should be adopted by the DNR, DOH and the EPA immediately. To the extent possible, the State should assume leadership in the implementation of this strategy, even if some additional costs are incurred by the State. The public must know that a single coordinated strategy is being followed to achieve an attainable resolution to contamination in Missouri, and that sufficient funds are available to implement it. To address the concerns of Missouri citizens, implementation of this strategy should include an opportunity for public comment and public hearings. Once adopted, this strategy should be periodically updated to keep Missouri citizens informed and to permit their comment on actions to be taken.

The Task Force believes that specific responsibility should be assigned the Director of DNR to assure that priority is maintained on this problem until closure of the secure storage facility. A system for information release should also be established so Missouri speaks with one voice to residents and owners of dioxin sites and to the public. Such information release might be accomplished by an expanded Dioxin Update and periodic press releases. Regular listing of the implementation status at each site should be included. To the extent possible, information from State agencies should be supplemented with sufficient explanation to permit the public to view the information in the context of this Strategy. Information released or published by EPA, Centers for Disease Control (CDC), Veterans Administration (VA), National Institute for Occupational Safety and Health (NIOSH), or others should be summarized, evaluated, and commented upon to help the public to assess its impact on the Missouri Dioxin Strategy.

It is the opinion of the Task Force that the State of Missouri has a public image as the dioxin center of the USA. That problem should be addressed and solved, even if the solution requires the expenditure of some funds currently not available. Otherwise, that image will continue to disrupt the lives of its citizens and plague the economic well being of the State. A decisive plan and responsible action will clearly demonstrate to the public that Missouri is willing to make and execute the necessary decisions to ensure the health and well-being of her citizens and to provide a good environment in which to live. It is believed that this was the experience of the Seveso Authority, whose representatives presented to the Task Force a description of their response. Some of their decisions may prove to be inadequate or excessive, but the Seveso Authority took action according to a publicly stated plan and subsequently reinhabited the area, permitting life to go on until an ultimate answer can be found.

The extremely toxic properties and potential health effects of dioxin have caused an enormous effort to be mounted to rid the environment of this hazardous substance. While this high level of concern is certainly justifiable, dioxin also has several properties which greatly reduce exposure to humans and migration in the environment. These properties include its extremely low solubility in water, extremely low volatility, and tight binding to soils. While dioxin is extremely toxic, dioxin as it exists in Missouri soil may not be as serious a problem as other contamination by very toxic and carcinogenic hazardous wastes. Our zeal to rid the State of dioxin should not deplete financial and human resources to the exclusion of the other very important hazardous waste problems in Missouri.

In summary, the Task Force concludes that an orderly, planned removal of dioxin contaminated soil to a secure storage facility best serves the interests of Missouri and its citizens. Recognizing the potential effects of dioxin, it seems prudent to take decisive, but not panic-driven actions to eliminate potential exposure and control the spread of dioxin in Missouri. A planned approach will require several years to implement and will require delay at some sites relative to others. Some sites will require short term remedial actions due to relative risks of exposure or probable spread of contamination. Most other sites should be left intact until removal and storage can be accomplished. For a few sites it seems possible that the health and environmental risks may be eliminated without excavation. These sites might be left intact with only long term surveillance and maintenance.

A. The Missouri Dioxin Strategy must include all sites regardless of CERCLA status.

The majority of the confirmed sites in Missouri contain contaminated soil which can be treated in a similar manner. In the Task Force's opinion, these sites should be handled as one problem under the Comprehensive Emergency Response, Compensation, and Liability Act (CERCLA). If this is not possible under Federal law, Missouri should nevertheless follow a comprehensive strategy for all sites.

Under the present CERCLA Law all hazardous waste sites undergo a site evaluation and ranking procedure. Based on a large number of parameters, each site is assigned an overall priority number which determines if that site qualifies for remedial monies under CERCLA. Currently only 6 of 33 dioxin sites in Missouri qualify for these funds, which are being administered on a site by site basis.

To remedy this situation, the State has requested that EPA combine the 33 sites for consideration as a single CERCLA site and currently is conducting negotiations with EPA to that end. The Missouri dioxin problem is unique in that while the sites are not contiguous, they are linked together because of the same type of contamination, namely dioxin in soil, the common origin of much of the material, and uniformity of the method of disposal. Individual listing of the sites causes extreme duplication of effort within a number of state agencies in every facet of handling of the sites, including health studies, remedial actions, and attempts at cost recovery. Feasibility studies being done for EPA on several of the CERCLA sites are identifying the same range of solutions. Considering each of the sites separately adversely affects the type of solution selected and the cost of that solution. Since in Missouri the total cost of transporting materials off-site appears to be a small fraction of the total cost of remedial actions, a policy of treating all non-contiguous sites separately is not cost effective or logical. Therefore, it is recommended that the State continue to make every effort to have the 33 sites ranked as a single CERCLA site.

Should this primary course of action not be possible, the state of Missouri should still follow a uniform strategy to fully address the dioxin problem. The residents of non-CERCLA sites are no less concerned with the potential health problems, loss of property use, land values, and the overall psychological effects of the problem than those on sites which have higher CERCLA risk assessment point totals. If federal law cannot address their plight, the state of Missouri must provide the necessary uniformity of action.

B. High priority should be given to development and publication of an expedited schedule for sampling and analysis at all suspected sites, and for extensive grid sampling at known sites.

It is recommended that adequate sampling and analytical capabilities should be established or contracted by the State to provide this critically needed information as soon as possible.

An expedited timetable for comprehensive sampling of all sites, and prompt analysis of samples taken, should be developed to define the total problem in Missouri. It is recommended that the State should supplement its own resources with contract sampling and analyses to provide timely response. It is also recommended that a careful study by industrial hygiene experts should be made to determine the protective equipment appropriate to the sampling activity. Workers should be protected from expected danger, but residents should not be unnecessarily alarmed by overemphasis on worker protection.

C. High priority should be given to the siting for and construction of a central storage facility and interim storage as required.

A timetable for selection and construction of the facility and estimates of the period of time required to accomplish secure storage should be given at the earliest possible date.

The Task Force recommends that several potential storage sites should be selected and critically evaluated immediately. The criteria for the facility should include complete encapsulation of the dioxin contaminated soils and solid debris; collection, monitoring and treatment of water from the facility prior to release to the environment; prevention of rupture of the facility due to thermal expansion and contraction or moderate earthquake activity; and separation from the existing soil and ground water system. Criteria for the site should include State ownership; low land usage but with parks and wilderness areas excluded from consideration; accessibility for soil transportation; minimal risk of public exposure; and utility of the site following facility closure.

The Task Force specifically recommends that Times Beach should be included in the evaluation of potential sites. In spite of obvious concerns about flooding, Times Beach has several advantages as a location for a well constructed facility. It is estimated that about ninety percent of Missouri's dioxin contaminated soil is located in eastern Missouri with about sixty percent being in the Times Beach roadways. Potential for exposure and potential for spread to the environment and total cost could all be minimized by locating the storage facility on this site.

If about sixty acres of Times Beach were filled with clean soil to above maximum flood level, an above ground facility, similar to those built at Seveso, could be constructed to safely collect and control water from the facility. The construction of a levee, which may be required to permit excavation and removal of soil to another site, would provide additional protection for the facility. Following closure and replanting, the area should be made available for public use.

D. The central storage facility should be utilized only for dioxin contaminated solids.

It is recommended that alternate technologies, such as incineration, should be utilized for the small amounts of existing liquid wastes containing dioxin. The central storage facility must be constructed exclusively for the containment and control of dioxin contaminated soils and associated solid debris. The Task Force concludes that existing or proposed hazardous waste landfills are not acceptable alternatives.

The high probability that ultimate destruction of dioxin contaminated wastes may be required and become feasible in the future dictates that only solid dioxin wastes should be included in this facility. Non-contaminated solids, such as houses, trailers and trees, should not be stored in the dioxin facility simply because they are located on a dioxin site.

E. A primary aim should be the reconstruction and reinhabitation of all sites.

Decisions should be made for each site based on total assessment of risks of the spread of dioxin in the environment and risks to the health of residents and the public. Special efforts should be made to maintain the integrity of affected communities, and minimize the financial burden.

The Task Force recommends that the preservation of neighborhoods and communities should remain a high priority. The Missouri Dioxin Strategy must recognize that disruption of residents' lives has already occurred, with accompanying emotional stress, and that actions taken should be sensitive to residents' concerns. Property purchases do not solve the problem.

F. The state should establish criteria and follow a uniform policy regarding residents and owners of dioxin sites.

Included should be criteria for temporary relocation to reduce exposure, temporary relocation during excavation and reconstruction, level of reconstruction, replacement of personal property, title clearance on reconstructed sites, and maintenance and control of sites stabilized and left intact.

To build and maintain public confidence in the Missouri Dioxin Strategy, a uniform policy must be communicated and followed. While it is recognized that different actions will be required at different sites, and that all residents' concerns and expectations cannot be universally satisfied, actions that are taken must be administered fairly and equitably. In particular, during relocation of residents, adequate consideration must be given to protecting family structure and minimizing psychological and economic impact.

G. The experience gained at Seveso, Italy, should be utilized to the extent possible.

Much information is available on worker protection, sampling techniques and construction engineering data. This should be utilized to minimize response time and costs in Missouri.

The Task Force was impressed by the efforts of the Lombardi Government and its agent, the Seveso Authority, in addressing the problem at Seveso. A great deal of information was generated and should be utilized by the State to the extent possible. One of the problems they encountered was the delay in obtaining analyses during excavation. It is recommended that the State should address that problem before beginning excavation since it strongly affects both the timeliness and cost of excavation.

The Task Force recommends that a summary of the Italian health studies should be provided to Missouri citizens, along with their findings on dust monitoring and other aspects of excavation and storage.

H. The residual level of dioxin allowed to remain after excavation and decontamination must be specified.

The Task Force recommends that CDC develop residual levels of clean up which will permit return of residential and agricultural properties to unrestricted land use as soon as possible. In addition, certain other residual levels, which may be site specific, should be developed for restricted use in areas such as commercial and industrial settings. Contaminated soil, excavation and processing must take into account the health and safety of workers and the local population.

The Task Force concludes that properties should be returned to unrestricted use, and removed from the dioxin registry, at the earliest possible date. To accomplish this, acceptable residual levels for clean up must be established.

I. The State should begin immediately to communicate the criteria for safe transportation of dioxin contaminated soils on Missouri roads and highways to address public concerns.

A mechanism must be developed for educating and reassuring residents and the public near any proposed route over which transportation of dioxin contaminated soil will occur. The concerns of these citizens are no less important than those of residents, workers, etc. The Task Force believes that state-of-the-art technology can provide all of the needed protection and that a format must be found for conveying this information.

J. A Missouri Program in Toxicology should be established.

It must be recognized that dioxin is by no means the last toxic material which may threaten the health and well being of Missouri citizens. To assist in final decisions regarding dioxin destruction and to provide a system for informed and rational future responses to toxic substances, a State Program in Toxicology should be established. That program should include an efficient system for gathering information, state-of-the-art analytical development capabilities, and appropriate animal toxicological research facilities.

The Task Force recognizes that a program in toxicology cannot be implemented to provide immediate response to the present dioxin problem. However, once secure storage of dioxin contaminated soils is provided, many dioxin questions will still need to be addressed as the ultimate destruction of dioxin is considered. This program will assist in the continuing evaluation of dioxin and will provide for an early response to future toxic waste issues. The Task Force strongly recommends that such a program should be established as a State facility, utilizing, to the extent possible, the capabilities that already exist in the universities and industries in Missouri through cooperative programs. The recently enacted hazardous waste legislation will have limited success until it is possible to define and quantitate compounds and define their degree of hazard to human health through toxicological testing and research.

K. An advisory panel, similar in composition to this Task Force, but including two dioxin site resident representatives, should be formed to advise the Director of DNR.

This panel should exist only until secure storage site closure is accomplished. It should make recommendations concerning final remedial actions and long term health studies. The Task Force believes that such an advisory panel with citizen representatives from academia, public groups, industry and dioxin site residents could provide valuable public input and oversight of the implementation of the Missouri Dioxin Strategy.

SITE SPECIFIC RECOMMENDATIONS AND PRIORITIES

Recommendations on remedial actions to be taken at each site and the relative site priorities are based on the information presently available, and should be altered as new facts, particularly from more intensive sampling, become available. Recommendations are based on the assumption that the Missouri Dioxin Strategy for secure storage will be adopted and that necessary funding will be provided to implement the plan under CERCLA or by the State.

In developing recommendations and priorities, the following factors were given primary consideration by the Task Force: overall risk to residents, workers and the environment; exposure risks to residents (especially children) and workers; potential for erosion and dusting; maintenance of neighborhoods and property values; and long term effects on Missouri and its citizens.

Due to the similarity of circumstances at some sites, and the widely varying circumstances at others, the Task Force chose to assign priorities and make recommendations based on groups of similar sites. (Any future confirmed sites must be separately evaluated and acted upon.)

Our recommendations are as follows:

Group I: Sites where short term excavation and interim storage of excavated soils should be considered to prevent exposure and restore the neighborhood.

Sontag Road
Quail Run

It is recommended that extensive sampling, temporary relocation of residents, excavation of soil with removal to an off-site interim storage facility, reconstruction and reinhabitation should be planned and implemented as soon as possible.

Group II: Sites where short term stabilization actions to prevent exposure or erosion should be taken, with the site remaining intact until secure storage can be accomplished.

Minker
Stout
Saddle and Spur Club
Cashell Residence
Ruth Sullins Property
Romaine Creek

It is recommended that short term actions should be taken to determine the potential for exposure and erosion, then provide temporary protection, possibly including off-site storage, until permanent removal, secure storage, reconstruction and inhabitation can be accomplished. (See recommendations on Minker-Stout in the Interim Report of the Task Force.)

Group III: Sites which should remain intact until secure storage can be accomplished.

- Spring River
- Denney Farm
- Shenandoah Stables
- Bliss Farm Road
- Wall Property and Piazza Road
- Bubbling Spring Arena
- Timberline Stables
- Erwin Farm
- Rusha Farm
- Times Beach
- Methodist Church
- Bliss Oil Frontenac
- Mid-America Arena
- Sand Cut Road
- Baxter Gardens

It is recommended that these sites should be continuously assessed as new sampling information becomes available.

Group IV: Sites which should be assessed for potential exposure, dusting, erosion and the presence of utilities in affected areas. Depending on that assessment, the areas should be paved and excavation and use controlled, or they should be included in Group III for excavation and secure storage.

- Southern Cross Lumber Company
- Hamill Transfer Company
- Overnite Transfer, Inc.
- Jones Truck Lines

Group V: Sites where other remedial actions are in progress or being negotiated.

- Syntex Verona
- Syntex Springfield
- Neosho Digester, Trench, Tank, Spill Area and Wastewater School
- Bill Ray Farm

It is recommended that the current negotiations and remedial actions should continue with the State providing secure storage, as required, to expedite resolution.

Group VI: Sites where sufficient data is not available for recommendation.

- Bull Moose Tube Company
- Community Christian Church

RECOMMENDATIONS FOR FURTHER RESEARCH

The Task Force recommends that the following scientific studies should be done to expand the available data base on dioxin with emphasis on ultimate disposal solutions:

1. Physical properties of dioxin in water and other solvents should be determined.
2. The binding mechanism of dioxin to a variety of soils, and the soil water distribution coefficients should be determined.
3. The method(s) by which dioxin moves in the environment should be determined and quantified.
4. The natural decay rate of dioxin in water and a variety of soils should be determined.
5. The temperature-vapor pressure relationship for soil bound dioxin should be quantified, and the ambient rate of volatilization from soil determined.
6. Parameters for the volatilization of dioxin bound to soil and for incineration of volatilized dioxin should be determined.
7. The ash from incineration of dioxin bound to soil should be analyzed to determine if the resulting ash can be delisted under RCR'.
8. Continuing work should be done on insitu treatment of dioxin contaminated soils (e.g. chemical destruction).
9. Microbial degradation studies to find or develop an organism which can safely and economically degrade dioxin under field conditions should be continued.
10. Acute and long term health studies in multiple species to clarify the:
    a. Absorption of dioxin from soils and other media and from the food chain.
    b. The metabolism of dioxin.
    c. The toxicology and carcinogenic potential of a prolonged period of low dosage of dioxin.
11. The determination of tissue levels of dioxin to include: the normal or "base line" levels of dioxin in the tissue of "unexposed" humans; the possible low dosage cumulative tissue concentrations over time; the change in such tissue levels over time in highly exposed individuals; and the correlation of blood and tissue levels of dioxin with different types of exposure.

12. Continuation and expansion of the current epidemiologic and clinical studies of the individuals exposed to dioxin in industrial accidents; exposed through protracted soil contact such as in Missouri; and in groups otherwise exposed in the past and at present to other chemical product derivatives of trichlorophenol, possibly contaminated with dioxin.

We, as members of the Governor's Dioxin Task Force, do hereby approve the Task Force report this 31st day of October, 1983.

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James A. Finch, Jr. Chairman

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Betty Wilson
Betty Wilson

GLOSSARY OF MEDICAL TERMS

Acute: Having a short or relatively severe course.

Adipose tissue: Fatty tissue.

Aryl hydrocarbon hydroxylase (AHH): A normally occurring liver enzyme, the function of which includes the metabolism or break-down of borth natural and poisonous substances in the body. In certain laboratory animals, this enzyme has changed some substances into carcinogens.

Carcinogenic: Producing cancer.

Cell mediated immunity: Specific acquired immunity in which the role of small white blood cells called T-lymphocytes is predominant; it includes certain aspects of resistance to bacterial and viral infections, to cancer, and to organ transplants, and has a role in certain allergies.

Chloracne: Skin eruptions that are somewhat similar to but sometimes more severe than teenage acne, but unlike teenage acne are due to exposure to organic compounds containing chlorine or other related chemicals.

Chronic: Persisting over a long period of time.

Cytogenesis: The formation and development of cells.

Demyelinization: To remove the myelin which covers some nerve fibers. The myelin is a white fatty substance which forms a sheath around certain nerves.

Fetotoxicity: The quality of being poisonous to the fetus.

Immunosuppression: Diminution of the immune response.

Lipid: Any one of a group of complex compounds which include fats, oils, fatty acids, waxes, etc.

Neuropathologic: Pertaining to diseases of the nervous system.

Neuropsychiatric: Pertaining to diseases that may have both a neurologic and psychiatric basis.

Porphyria cutanea tarda: A disease in which the metabolism of porphyrins (a chemical produced in the liver) is disturbed resulting in chronic skin lesions and an enlarged liver.

Soft-tissue sarcomas: A type of cancer found in muscle, fat, nerve, or connective tissue.

Synthesis: The formation of a complex chemical compound by the combining of two or more simple sompounds.

Teratogenesis: The production of physical defects in the developing fetus in utero.

EXECUTIVE ORDER
83-5

WHEREAS, extensive laboratory testing has confirmed the presence of dioxin at several locations in Missouri; and,

WHEREAS, dioxin is a potentially deadly chemical which threatens the health and safety of thousands of Missourians,

NOW, THEREFORE, I, CHRISTOPHER S. BOND, GOVERNOR OF THE STATE OF MISSOURI, by virtue of the authority vested in me by the Constitution and laws of Missouri, hereby establish the Governor's Task Force on Dioxin (hereinafter the "Task Force") which shall be composed of such residents of Missouri as from time to time may be appointed by the Governor. The Task Force shall include representatives of Missouri higher education institutions and Missouri companies which have expertise regarding hazardous wastes, as well as concerned citizens. The Chairman of the Task Force shall be designated by the Governor.

The purposes of the Task Force shall be as follows:

1. To examine and evaluate all known methods, processes and technologies for the destruction and containment of dioxin, particularly with respect to dioxin as it exists as a contaminant in soil and stream sediment at various locations in Missouri;

2. To examine and evaluate the public health dangers associated with dioxin, particularly with respect to dioxin as it exists as a contaminant in soil and stream sediment at various locations in Missouri;

3. To prepare and present, not later than June 1, 1983, an interim report which, *inter alia*, provides specific recommendations for solving those public health and environmental problems occurring in Times Beach, Missouri which are the result of dioxin contamination exacerbated by widespread flooding; and

4. To prepare and present, as soon as practicable but not later than October 1, 1983, a final report which includes the evaluations set forth in paragraphs 1 and 2 above, and which recommends a practical and effective plan of action for implementing comprehensive and permanent solutions to the public health and environmental problems caused by dioxin contamination in Missouri.

The Director of the Department of Natural Resources and the Director of the Division of Health shall make available such staff and support resources as are needed by the Task Force in the course of its deliberations. All meetings of the Task Force shall be open to the public, and a record shall be kept of all Task Force proceedings.

EXECUTIVE ORDER
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Members of the Task Force shall receive no compensation for their services, but shall be reimbursed from funds provided through the Department of Natural Resources for their reasonable and necessary expenses. The Task Force may contract for the services of such consultants and experts as it deems necessary and shall pay for such services from funds provided through the Department of Natural Resources or the Division of Health.

The Task Force shall meet at the call of the Chairman, or at the call of the Governor or his designate, and shall dissolve upon the issuance of its final report, unless extended by subsequent Executive Order.

IN TESTIMONY WHEREOF, I have hereunto
set my hand and caused to be affixed the
Great Seal of the State of Missouri, this
14th day of February, 1983.

Christopher S. Bond
GOVERNOR

ATTEST:

James C Kirkpatrick
SECRETARY OF STATE

EXECUTIVE ORDER

83-13

WHEREAS, the Governor's Task Force on Dioxin was created by Executive Order 83-5 on February 14, 1983; and,

WHEREAS, said Executive Order provided that said Task Force was to prepare and present, as soon as practicable but not later than October 1, 1983, a final report, and that said Task Force was to dissolve upon issuance of said final report, unless extended by subsequent Executive Order; and

WHEREAS, need has arisen for said Task Force to obtain and evaluate additional information prior to preparation and presentation of said final report,

NOW THEREFORE I, CHRISTOPHER S. BOND, GOVERNOR OF THE STATE OF MISSOURI, by virtue of the authority vested in me by the Constitution and laws of Missouri, hereby extend the existence of the Governor's Task Force on Dioxin, as established by Executive Order 83-5 issued on February 14, 1983, until completion of its final report or until November 1, 1983, whichever sooner occurs, unless extended by subsequent Executive Order.

IN TESTIMONY WHEREOF, I have hereunto set my hand and caused to be affixed the Great Seal of the State of Missouri, this 31st day of August, 1983.

Christopher S. Bond
GOVERNOR

ATTEST:

James C. Kirkpatrick
SECRETARY OF STATE

GOVERNOR'S DIOXIN TASK FORCE

The Honorable James A. Finch, Jr.
Judge
Chairman, Governor's Dioxin Task Force

Mr. Dwight Douglas
Douglas, Douglas & Johnson Law Offices

Mr. Ray Forrester
Engineering Manager & Dioxin Project Supt.
Syntex Agribusiness, Inc.

Dr. Perry King
Director, Quality Assurance Chemicals
Mallinkrodt, Inc.

Dr. Robert L. Powell
Assistant Professor of Chemical Engineering
Washington University

Dr. Carl J. Marienfeld
Professor of Family & Community Medicine
Environmental Health Surveillance Program
Sinclair Research Farm

Mrs. Sharon Rogers
Warren Countians Against Hazardous Waste

Dr. George Roush
Director, Dept. of Medicine & Environmental Health
Monsanto

Dr. James Shaddy
Associate Professor of Ecology
Northeast Missouri State University

Mrs. Betty Wilson
Environmental Quality Chairperson
League of Women Voters

TASK FORCE STAFF SUPPORT

Department of Natural Resources

Fred Lafser, Director
Ronald Kucera, Deputy Director

Robert Schreiber, Director - Division of Environmental Quality
Linda James, Assistant Director

David Bedan, Director - Waste Management Program
Beth Rice, Environmental Engineer
Dan Tschirgi, Environmental Engineer
Dianne Luebbert, Clerk Steno
Martha Siegel, Clerk Typist

Mary Still, Director - Office of Public Affairs, Division of Management Services
Nancy Guyton, Office of Public Affairs

Department of Social Services

Dr. Robert Hotchkiss, M.D., Director - Division of Health

Dr. Denny Donnell, M.D., MPH, Director - Disease Prevention Section
Dr. Patrick Phillips, DVM, MSPH
Dr. Thomas Satalowich, DVM, MSPH
Jeff Staake, Dioxin Activities Coordinator

Mr. LEVITAS. Thank you very much, Congressman Young, for your additional contribution. Let me just say that I think you are right in that we have got to be diligent about this. Unfortunately, this is one of those areas it seems to me where the public goes back to sleep 2 months after Times Beach is over, and they only wake up when the next ticking time bomb goes off and there is another Times Beach someplace else. And then they go back to sleep again.

Fortunately there are more and more people such as yourself, such as the panel of witnesses we will be hearing this morning, and others, who are beginning to make this an ongoing issue rather than one which comes and goes as do the headlines. But I think until we do that, the enormity of the problem, the number of sites we are talking about, the now estimated $50 billion it is going to take to clean them up, the fact we haven't even identified some of the worse sites much less cleaned up those we already have identified, nor will we by the end of this century, and the tragedy of what happens to the people who are affected by hazardous waste sites, means that if we aren't persistent and if we don't persevere, the task will not be done. I think that would be a terrible indictment of all of us.

Mr. YOUNG. Mr. Chairman, if I might respond to your remarks, we are still wrestling with low-level radioactive uranium mill tailings in my district. A result of the activity of the creation of the atomic bomb. I still have many, many sites throughout my district. And here we are 40 years later and we have no resolving of that question. And it is a serious question to the people of my district and the State of Missouri.

I have got enough for everybody. If you could just spread it around a little bit like Dolly Levi.

Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you. Mr. Shaw?

Mr. SHAW. I would like to add my words of welcome to you, Bob. I am privileged to serve with the gentleman from Missouri as his ranking member on Public Grounds and Buildings. I know of his continuing concern for the problems that his district has had to bear with regard to toxic waste. Unfortunately, we are just beginning to realize at this time the full impact this type of disposal has on the health of communities around the country.

I am sorry that Mr. Young's district has borne such an extraordinary waiting period of not knowing, and of course the potential health damage that this situation has caused. I know of your concern and work in this particular area, and I appreciate your coming before the committee this morning.

Mr. YOUNG. Thank you very much, Mr. Shaw.

Mr. LEVITAS. Mr. Rowland.

Mr. ROWLAND. No questions.

Mr. LEVITAS. Mr. Wise.

Mr. WISE. No questions.

Mr. LEVITAS. Without objection your entire statement will be made part of the record.

Mr. YOUNG. Thank you. Mr. Chairman, this is an important committee to the public health of this country. I am just real pleased that we had the foresight to move ahead in the investigation and oversight on this right from the beginning.

Mr. LEVITAS. Thank you. I appreciate your being here, Bob.

Mr. YOUNG. Thank you very much.

Mr. LEVITAS. Now, we look forward to hearing from a panel of State legislators who are going to share with us their experience. It is not just because I served for 10 years in the Georgia Legislature that I look forward to this testimony. But it is also because I know from experience here as well as the State level that most of the sound answers we get to the problems this Nation faces come from the innovative experience that our States bring to us.

So it is in no way coincidental that we have today as our next panel of witnesses three State legislators who have played a role in this entire matter. I would like to ask the panel of witnesses to come forward.

Also, I note the presence of our colleague Congressman Matsui from California, who is accompanying Hon. Lloyd Connally from the California Assembly. You may come forward, too, if you would, please.

Assemblyman Connally, Representative Joe Hackney of North Carolina, and Representative Dee Long of Minneapolis, MN.

TESTIMONY OF HON. LLOYD G. CONNALLY, CALIFORNIA ASSEMBLYMAN, SACRAMENTO, CA; HON. JOE HACKNEY, NORTH CAROLINA HOUSE OF REPRESENTATIVES, CHAPEL HILL, NC; HON. DEE LONG, MINNESOTA HOUSE OF REPRESENTATIVES, MINNEAPOLIS, MN

Mr. MATSUI. Mr. Chairman, I merely would like to come to just introduce my colleague.

Mr. LEVITAS. Please have a seat, and do so.

Mr. MATSUI. I would just like to introduce my colleague Assemblyman Lloyd Connally from Sacramento. He and I had served in local government, city council, for 5 years. Then I came back to Washington, of course, and then he went to the State capital.

He has been one of the leaders in the whole area of toxic waste disposal in the State of California. He has knowledge of victim compensation legislation that I am sure you are all interested in hearing about. I think his expert testimony today will be very helpful to the committee and the Congress.

I thank the chairman for inviting him, and myself as well.

Mr. LEVITAS. Thank you, Bob. We appreciate your coming here, and look forward to hearing from Mr. Connally as to his experiences in California.

Thank all of you for being here. I think the best way to proceed would be to have each of you make a brief opening presentation, and then we would like to put some additional questions to the panel and to each of you following your own presentation of your experiences in your State.

So why don't we start with Mr. Connally?

Mr. CONNALLY. Mr. Chairman, members, thank you for inviting me. Mr. Levitas, I don't know how you do it, but you started your committee meeting on time. I wish I could take you back to California. We never start on time; so whatever your magic is, it is good magic.

During the last week and a half or so I had occasion to peruse some of the testimony presented to you folks last summer. It is very impressive indeed. You have obviously put in substantial work. You are to be commended. It is a problem not only in California, as I can tell by the testimony, but indeed throughout the whole country.

California started in 1980 on a similar process in terms of evaluating the issue of compensation, and specifically folks who suffer health problems as a result of toxics. And as a result of a hearing process that extended over the better part of a year, we came to eventually two conclusions: One, that indeed there was a significant number of people, difficult to quantify but a large number of people who are exposed to toxics and suffer health effects as a result of that; two, we came to the conclusion that those folks who suffer serious problems—that is, the ones that are really damaged—have fairly effective remedies available now through the common law tort system, particularly in California where we are fairly progressive with regard to our plaintiff law.

However, we found that the smaller injury, the person who was in many instances isolated and suffered damages of a smaller character had no effective remedy. The costs of proving the causal link between the toxics and health problems they were experiencing was inordinately high; one that would not be borne by private attorneys, one that the individual could not pay.

As a result, in September 1981 the California Legislature adopted and the Governor signed a two-tier system of victim compensation recovery. We didn't know it was two tier at the time because the study group term "two tier" hadn't come into existence, but essentially the same thought pattern was there. One, it was designed so that the larger injury person would proceed under the normal tort action in the State. Second, for the person who suffered the smaller isolated case, there would be an administrative remedy through our own local or State superfunding mechanism whereby they could file a claim and seek the damage recovery from the harm that they had suffered.

Specifically, we set aside $2 million a year for that purpose, and we put forward the following system: First, we required that the folks either had to suffer the health effects from a source that they did not know where it originated from. For example, they were exposed to toxics at some point so far in the past that they didn't remember where that exposure point occurred, or it was the proverbial orphan site. In the alternative, they had to go to court and seek a judgment, and get a judgment against a nonpolluter, a nonsource, and then be able to collect that judgment either as a result of insolvency or because the person was no longer in business, or what have you. That was the first condition. The second condition was that they had to prove by the weight of evidence that the health harm they suffered was proximately caused by this exposure to toxics. Those are the two conditions they had to meet in order to come under the jurisdiction of the Superfund in order to file that claim.

Once they met those conditions they could recover up to 100 percent of their medical costs for 3 years, up to 80 percent of the lost salary they incurred up to 3 years, and there was the usual language with regards to double recovery.

This law, with the support of business and industry throughout the State, was enacted. Immediately after its enaction, the State moved aggressively to publicize the law. We sent out 60,000 letters to doctors throughout the State of California, to the bar; notices were required to be posted in hospitals and employers that employed more than 50 people.

We have received literally hundreds of inquiries about this law. Almost 30 inquiries a day come into the board of control in terms of how you file a claim. The bottom line is whether or not this law is working. And the answer to that question is that it is not.

The California experience, the law that I have identified, is so squeaky tight that anything short of calling it a failure would be misleading you. Out of the hundreds of inquiries we have received only two people have filed claims to date. Out of the $2 million setaside for the last 2 years not $1 has been distributed to claimants who filed these claims.

I think there are two reasons for this failure. Hopefully, from our experience here, you will not make the same mistake in the Federal legislation. The first mistake was the requirement for those folks who know what the source is to proceed to get a judgment before they can file a claim. That is, proceed to get a judgment, then be unable to claim it. If they are in a position to proceed to expend the funds required to prove they are in fact suffering from health effects caused by toxic substances, they or their counsel will make a determination at the outset whether or not there is going to be a deep pocket, a source to recover at the other end of that process. So those people don't come to our Superfund for claims.

The second group, these are the folks who don't know the source, the orphaned site group, have to meet the same burden of proof, essentially the same burden of proof that they have to meet in a court of law. That is, they have to prove by the weight of evidence that their health problem was proximately caused by the exposure to the toxic substance. To meet that burden of proof, if they have to do that, they incur the same kinds of costs, the same kind of difficulty and time that the normal process, or normal tort process puts in front of them.

There was in this past year by State Senator Torres, and I joined in this effort, an attempt to change the California law in these two areas so that our victims' comp process would work more effectively. The same groups that had supported the original law, supported the 1981 victims' comp law, opposed the amendments that would make the changes required so that it would work. If there is a lesson here that I could convey to you, it would be that in the fashioning of the Federal remedy you be extremely careful that you do not identify properly that there is not an effective remedy now and then set forward another remedy, as I think we have done in California, that is equally ineffective.

The single most important thing that you can address in that regard is if you move forward with this two-tiered concept in this victims' comp recovery mechanism is to put in some kind of rebuttable assumption so you don't have the same causal proof burden. I find almost unbelievably a consensus vote, one mechanism to provide that presumption. But it ought to be there, some way that is short of the extensive epidemiological studies required to prove that causal connection now, a person who suffers a small injury can make a claim and recover.

Mr. Chairman, that concludes the remarks I have. I did bring with me the California version of the study group. It is a report prepared by our hazardous waste management council, very similar to your report. It was published just this past month. Talks about the California law, the difficulty we are having; makes suggestions for revisions in the California law, which I know will have some applicability to the Federal legislation as well.

Mr. LEVITAS. Thank you very much, Mr. Connally. We would like to have a copy of that.

I have some questions for you, but I think it might be helpful at this time to hear from the other State legislators on the panel.

[The report of the Hazardous Waste Management Council follows:]

HAZARDOUS SUBSTANCE VICTIM COMPENSATION APPLICATION BROCHURE

The State of California has a fund to reimburse any person who has suffered out of pocket medical expenses or lost wages or business income resulting from injuries caused by exposure to releases of hazardous substances. The program is administered by the State Board of Control.

Where to obtain applications or additional information: Contact the Board of Control, 926 J Street, Suite 300, Sacramento, CA 95814-2772. Phone number (916) 324-1342.

Qualifications for filing an application: Applicant must have incurred a medical expense or loss of wages or business income as a result of injury caused by exposure to a hazardous substance which was released in California.

The following conditions must also be met:

(a) The source of the release of the hazardous substance or identity of the party liable for damages in connection with the release or the responsible for the cost of removal of the substance is unknown or cannot with reasonable diligence be determined. This means you must have made some effort to identify the responsible party before filing a claim for compensation with the Board of Control.

(b) The loss was not compensable pursuant to law, including Chapter 6.5 (commencing with Section 25100), because there is no liable party or the judgement could not be satisfied in whole or in part against the party determined to be liable. This means no one was able to identify who caused or was responsible for the release or after identifying the responsible party you were unable to get reimbursement for your damages from them. Lack of recovery could be due to the liable party being insolvent or no longer in existence or cannot be found by reasonable means.

Information that must be submitted when filing a claim under the hazardous substance compensation program:

The law requires that the Board obtain certain information from an applicant before considering the application. This includes the following:

(1) Certification by the claimant of dates and places of residence for five years preceding the date of the claim.

(2) Certification of the medical history of the claimant for the five years preceding the date of claim.

(3) Certification of the alleged physical injury or illness and expense for that injury or illness. The certification must be made by hospitals, physicians or other qualified medical authorities.

(4) Copies of the claimant's federal income tax returns for the three years preceding the incident to establish lost wages or income.

(5) A sworn statement by the claimant attesting to the authenticity of the claim to the best of his or her knowledge.

(6) A full description of the incident supported by appropriate evidence from government agencies of the release of the hazardous substance claimed to be the cause of injury or illness.

Losses compensable under this program:

(a) One hundred percent of uninsured out-of-pocket medical expenses for up to three years from onset of treatment.

(b) Eighty percent of any uninsured actual lost wages or business income caused by injury to the claimant or claimant's property not to exceed fifteen thousand dollars ($15,000.00) per year for three years.

The limits for filing a claim: A claim must be present to the Board under this program within three years of the date of discovery of the loss, or from January 1, 1982, whichever date is later.

Board decisions:

All decisions rendered by the Board under this program shall be in writing and shall be rendered within 90 days of submission of a claim to the Board, unless an extension of time is agreed to in writing by all parties to the claim.

The decision of the Board shall be considered a final agency action for the purposes of judicial review.

Payment of claims: Claims approved by the Board under this program will be paid from the Hazardous Substance Compensation account.

Miscellaneous information:

No compensation or decision by the Board under this program is admissible as evidence of any issue of fact or law in any proceeding brought under any other provision of law.

Compensation for any loss claimed under this program by any other source shall preclude compensation from this program for that identical loss.

Compensation of any loss under this program is subject to the state acquiring by subrogation all rights of the claimant to recover such loss from the party determined to be liable.

The Board has the authority to adopt and revise all rules and regulations necessary to implement the provisions of this program including regulations which specify the proof necessary to establish a loss compensable under this program.

What constitutes a hazardous substance: There are many types of hazardous substances listed under United States Codes and Environmental Protection Agency actions. If you have any questions about the agent which caused your injuries, refer to Section 25316 of the California Health and Safety Code for reference to determining which specific agents are considered hazardous substances under this program.

What the term "hazardous substance" does not include under this program: Refer to Section 25317 of California Health and Safety Code for specifics of agents not considered hazardous substances under this program. In general, they include petroleum, natural gas, liquid gas, or synthetic gas used for fuel or the ash produced by a resource recovery facility using a municipal solid waste stream. Also included is non-toxic, non-flammable, non-corrosive stormwater runoff drained from underground vaults, chambers or manholes into gutters or storm sewers.

What does the term "release" mean: It refers to any spilling, leaking, pumping, pouring, dumping or disposing into the environment.

What the term "release" does not include under this program:

(a) Any release which results in exposure to persons solely within a workplace, with respect to a claim such exposed persons may assert against their employer.

(b) Emissions from engine exhaust of a motor vehicle, rolling stock, aircraft, vessel or pipeline pumping station.

(c) The normal application of fertilizer, plant growth regulants and pesticides.

(d) Release source, by-product or special nuclear material from a nuclear incident. (See California Health & Safety Code Section 25321(c) for specific details.)

Air pollution: Section 25375 of the California Health & Safety Code prohibits the payment of a claim under the Hazardous Substance Compensation Program when that claim is the result of long-term exposure to ambient concentrations of air pollutants, commonly referred to as smog.

Submitting information: Submit only information that is accurate to the best of your knowledge as Section 25373(f) of the Health & Safety Code provides up to a $5,000.00 fine or up to one year imprisonment for knowingly giving or causing to be given false information as part of a claim under this program.

Mr. LEVITAS. Mr. Hackney, why don't we hear from you next, from the State of North Carolina.

Mr. HACKNEY. Thank you, Mr. Chairman. It is a pleasure to be here and to share with you some of our experiences in North Carolina as we grapple with the same problem that is facing my colleagues here.

I guess to describe what is going on in our State one would say that the main battle during the last legislative session was actually over what can be landfilled and what cannot be landfilled. The victim compensation issue was pretty much pushed to the back burner and only got on the calendar along about June or July. Nevertheless I will confine my comments to just that issue; that is, who will pay the damages.

In our State there was a study by our Governor's waste management board in 1981. Thereafter we established a permitting system to meld with RCRA in order to regulate hazardous wastes in our State in a safe manner. That legislation then called for a study, which was done. And that study did recommend adoption of the strict liability concept for hazardous waste occurrences. I have a copy of the study here.

It was found that the law, as my colleague Mr. Connally has so well described, simply is not adequate for the new concepts involved in proving toxic torts—it is often impossible to prove negligence in such a situation—and to define what a hazardous waste handler's duty might be under the negligence system. On the other hand, the strict liability system spreads responsibility for all injuries proximately caused by the "hazardous waste occurrence"—as we have defined it.

Strict liability does not, without some alteration of its pure form, solve the problem Mr. Connally described of proving causation. Sometimes you just can't do it. That is why you do need the Superfunds and governmental assistance. Likewise, strict liability in its purest concepts I suppose does not include the rebuttable presumptions that he mentioned.

I would like to tell you a little about our bill as it was introduced. It has not yet passed. It passed our house and is pending for possible action in our State senate when we meet again next June. As introduced, H.B. 738 contained these provisions:

One. It created a separate system of tort liability for "hazardous waste occurrences," based on strict liability. That is, it placed hazardous wastes, as defined by the Federal laws, in the same category with the ultrahazardous or abnormally dangerous substances that you find in the Restatement of Torts with respect to strict liability.

Two. This strict liability standard applied to generators, transporters, treaters, storers, and disposers, and the bill created a system by which, if the waste were passed on safely to the next person, under the manifest system set up by RCRA—except for the generators—then one was absolved of liability—thereby, we hoped, encouraging safe disposition of the materials.

Defenses allowed were assumption of risk, act of God and act of war, and no others. No punitive damages were allowed. The plaintiff could still elect a negligence theory and pursue that if that was desired. Statutes of limitations—more correctly, under the way our laws are written, called statutes of repose—a 10-year statute of repose in North Carolina was thought to be a bar and that was abolished in the original legislation.

During the committee hearings several objections were raised. Those are noted in my prepared statement, and some of those were credited and some not. There was put in a 30-year statute of repose to change the aforementioned 10-year statute of repose that is applicable in ordinary occurrences in North Carolina.

There was great debate about the subject of contributory negligence, as there is in the literature generally in the strict liability area. It was urged—in fact, the Governor's waste management board report suggested that contributory negligence be a defense. Those of us on the committee felt that that was totally inconsistent with the concept of strict liability and that assumption of a known risk was the proper defense to allow. That view prevailed in committee, and prevailed when the bill passed the House.

What to do about the State's liability was a major problem for us simply because there were no funds set up, as in some other States, for payment for these injuries. So we simply channeled those under the regular Tort Claims Act or State substitute for the workers' compensation.

There was discussion of an additional defense for acts of terrorism and vandalism and whether the strict liability standard, if adopted, ought to be the exclusive remedy.

I have indicated at the bottom of the second page of my statement the form in which the bill did pass the house of representatives. I have several copies of it here as it passed and would be happy to make one of those part of your record.

The emerging legislation we hope will be a part of the puzzle of victim compensation. We do not by any means consider it to be a complete remedy. I certainly support the formation of funds for compensation of victims. South Carolina has done this, and I have a copy of their legislation here if the members of the subcommittee have not seen it.

I do think that victims' compensation in the area of toxic torts requires the adoption of a strict liability standard at a minimum. We must not go into the late eighties and the nineties and expect victims to use the tort system as their principal means of recovery, and not have this strict liability standard. So I would urge the committee to consider some Federal incentive toward States moving their State laws in this direction.

I appreciate the opportunity to come here to tell you what small progress we are making in our State, and I appreciate the efforts of the members of the committee to struggle with this particular problem.

Mr. LEVITAS. Thank you, Representative Hackney, for sharing your experience with us. We, will as I indicated, have some questions in just a moment.

[The statement of Mr. Hackney follows:]

STATEMENT OF REPRESENTATIVE JOE HACKNEY OF NORTH CAROLINA

I have been asked to come to Washington today to tell of our efforts to enact legislation in the State of North Carolina imposing a standard of strict liability for accidents involving hazardous wastes. H.B. 738, after several substantive changes, passed our House of Representatives in July of this year, and is pending for consideration by the Senate in our June, 1984, session of the General Assembly.

H.B. 738 had its genesis in a recomendation by our Governor's Waste Management Board that the system of compensation for injuries in effect, the ordinary negligence system, was not sufficient to protect the public interest. There are many reasons why this is so. North Carolina has not yet adopted comparative negligence, and so any act of contributory negligence, however slight, completely bars any recovery. Secondly, the chances of proving negligence on the part of the hazardous waste handler are always problematical, and especially so in a case where the act of negiligence may have occurred many years previous to the injury.

In addition, current applicable statutes of limitations are simply not intended for present day circumstances. Thus our Governor's Waste Management Board recommended that our State adopt a standard of strict liability for hazardous waste occurrences, and H.B. 738 was introduced.

As introduced, H.B. 738 contained these provisions:

1. It created a separate system of tort liability for "hazardous waste occurrences", based on strict liability.
2. This strict liability standard would apply to generators, transporters, treaters, storers, and disposers.
3. Defenses were assumption of risk, act of God, and act of war.
4. Safe delivery to a hazardous waste facility would guarantee that the generator or transporter would not be liable, thus encouraging disposal in presumed safe depositories.
5. No punitive damages would be allowed.
6. A plaintiff could elect to pursue this remedy for negligence, if desired.
7. The statute of limitations was abolished for actions brought under the strict liability standard.

A series of committee and subcommittee hearings ensued, at which the usual environmental and business lobbies squared off against each other. Committee members heard a series of business objections to the bill:

1. It is unreasonable to have no statute of limitations at all, and industries may not be able to insure against the risk.

2. Not allowing contributory negligence as a defense will allow those at fault to recover. For example, a drunk could drive his automobile into a truck carrying hazardous waste, and still recover from the transporter and the generator.

3. It is unfair for the State to be liable for only $100,000.00 of damages under our Tort Claims Act, and industry to have no cap at all on damages.

4. An additional defense for acts of terrorism and vandalism should be included.

5. If the strict liability standard is adopted, it should be the exclusive remedy.

Our committee substitute bill which finally passed the House attempted to address some of the concerns expressed, but rejected the major ones as groundless:

1. Any statute of limitations would constitute a serious limitation of liability when injuries may become apparent 20-30 years after exposure. Nevertheless, in the committee substitute, there was inserted a 30 year statute of limitations.

2. Contributory negligence is manifestly inconsistent with the concept of strict liability, and its adoption as an available defense in the strict liability bill would make the bill ineffective.

3. The State should be liable in a limited way just as in other tort situations, but with a substituted standard of strict liability.

4. Providing an additional defense for acts of terrorism and vandalism acts as a disincentive to precautions to defend against such acts.

5. The dual remedies should remain, so that a plaintiff who can prove negligence within the applicable limitation period is free to pursue his remedy without the "no punitive damages" limitation included in the strict liability concept.

6. The act was clarified to assure that hazardous materials not yet "waste" were not covered.

7. A defense was established for intentional acts or omissions of third parties not connected to the defendant by an agency or contractual relationship.

Floor amendments which attempted to alter certain of these provisions were all defeated.

The difficulties of molding our traditional tort remedies to fit the modern requirements of victim compensation in the world of hazardous wastes are obvious. Yet this must be done, in fairness to the consuming public.

I fully support attempts to set up victim compensation funds, as South Carolina had done, from which damage awards can be paid. But a serious disaster can wipe out such a fund, leaving many victims compensated in small part or not at all. In my view, adoption of the strict liability standard should be an integral part of any attempt to solve the victim compensation problem.

The tort system, as modified by such legislation as H.B. 738, is a powerful incentive toward guaranteeing safe handling of hazardous wastes to prevent injuries and deaths from occurring. Victim compensation funds are essential to compensate those victims left out when even the tort system fails, as in the case of bankruptcy or inadequate insurance protections. And federal disaster relief will continue to be necessary when the hazardous waste occurrence is a true disaster.

As a state legislator who has struggled with the complexities of the problem, I applaud the efforts of the members of the committee to find a comprehensive federal approach. Your efforts, if successful, will provide a legacy of safe handling of hazardous wastes for our children and grandchildren.

GENERAL ASSEMBLY OF NORTH CAROLINA

H

SESSION 1983

2

HOUSE BILL 738
Committee Substitute Favorable 7/8/83

Short Title: Strict Liab. Hazardous Wastes. (Public)

Sponsors: Representative

Referred to: Water & Air Resources.

April 7, 1983

A BILL TO BE ENTITLED

AN ACT TO PROVIDE FOR STRICT LIABILITY FOR DAMAGES RESULTING FROM HAZARDOUS WASTES IN NORTH CAROLINA.

The General Assembly of North Carolina enacts:

Section 1. Short title. This act may be referred to as the Hazardous Waste Strict Liability Act of 1983.

Sec. 2. Purpose. The purpose of this act is to provide for a workable strict liability system for hazardous wastes in North Carolina as recommended in the 1983 Report of the Governor's Waste Management Board.

Sec. 3. Creation of a strict liability system for hazardous wastes. Chapter 130 of the General Statutes as it appears in Volume 3B of the General Statutes (and the Cumulative Supplement thereto) is amended:

(1) By adding the following at the end of G.S. 130-166.16:

"(23) 'Claimant' means a person damaged by a hazardous waste occurrence.

"(24) 'Hazardous waste occurrence' means any sudden or non-sudden occurrence in which damages result from any quality or characteristic of a solid waste, as defined in G.S. 130-166.16(16), which quality or characteristic causes the waste to be a hazardous waste under this Article and the rules and regulations adopted pursuant to it.

"(25) 'Strictly liable' means liable without regard to the defendant's negligence or fault. The defendant's foreseeability of danger or risk arising from or injury or other consequences caused by a hazardous quality or characteristic of the waste either at or prior to the occurrence shall not be relevant to a determination of liability.

"(26) 'Caused by' means caused in fact. If the evidence offered by any party tends to show that the damage of which the claimant complains was caused by both a hazardous waste occurrence and other causes, liability under G.S. 130-166.21G shall be limited to damages attributable to the hazardous waste occurrence."

(2) By adding a new section G.S. 130-166.21G to read as follows:

"§ 130-166.21G. Hazardous waste liability.--(a) This section applies only to hazardous waste occurrences caused wholly or partially by a defendant's operations or activities occurring after the effective date of this act. For the purpose of this section, waste polychlorinated biphenyls shall be considered a hazardous waste. Furthermore, for the purposes of this section, governmental immunity from strict liability for bodily injury or property damage caused by a hazardous waste occurrence is hereby waived, but only to the extent that damages do not exceed the amount of damages authorized by the North Carolina Tort Claims Act, G.S. 143-291. Any agency of the State of North Carolina that generates, transports, treats, stores, or disposes of hazardous waste is hereby authorized and empowered to procure proper insurance against such liability..

(b)

(1) Subject only to the defenses set forth in subsections (c) and (e) of this section, the generator, transporter, treater, storer or disposer in control of a hazardous waste at the time of a hazardous waste occurrence shall be strictly liable for bodily injury or property damage caused by the occurrence.

(2) Subject only to the defenses set forth in subsections (c) and (e) of this section, the generator of a hazardous waste, whether or not in control of the hazardous waste at the time of an occurrence, shall be strictly liable for bodily injury or property damage caused by hazardous waste occurrences arising from the hazardous waste generated. Under this subdivision, the generator shall be jointly and severally liable together with the transporter or any other person in control of the hazardous waste at the time of the occurrence. For purposes of this subsection, any person that accepts a hazardous waste from a transporter for storage and causes the hazardous waste to be again

transported shall be considered a generator.

(c) There shall be no liability under subsection (b) for a person otherwise liable who can establish by a preponderance of the evidence:

(1) that the claimant had knowledge of the danger and voluntarily and unreasonably encountered that danger; or

(2) that the hazardous waste occurrence was caused solely by any one of the following:

(i) an act of God;

(ii) an act of war or sabotage; or

(iii) an intentional act or omission of a third party (but this defense shall not be available if the act or omission is that of an employee or agent of the defendant, or if the act or omission occurs in connection with a contractual relationship with the defendant).

(d) Nothing in this section shall bar or otherwise affect the transfer of liability to the Post Closure Liability Fund, under Section 232 of the Federal Comprehensive Environmental Response, Compensation and Liability Act of 1980.

(e)

(1) Subject to the provisions of subsection (i), any defendant who proves that hazardous waste was transferred to and accepted by a hazardous waste facility duly permitted by appropriate regulatory agencies of which he is not the beneficial owner or

operator and that such transfer and acceptance is in compliance with applicable federal and State requirements existing at the time of the transfer and acceptance shall not be liable under subsection (b) for bodily injury or property damage caused by a hazardous waste occurrence after such transfer and acceptance.

(2) When signatures are admitted and established, production of a copy of a hazardous waste manifest entitles a defendant, who according to the manifest generated or transported the waste to a hazardous waste facility, to the defense under this subsection unless the claimant establishes that the waste was not in fact transferred to and accepted by the hazardous waste facility prior to the hazardous waste occurrence.

(3) Each signature on a hazardous waste manifest is presumed to be genuine or authorized unless the party opposing its admission offers prima facie evidence that it is not genuine or authorized.

(f) No punitive or exemplary damages shall be awarded in any action under subsection (b).

(g) Nothing in this section shall deprive a claimant from electing to pursue any other cause of action based upon a hazardous waste occurrence that may exist under statutory or common law, or to deprive a person liable under subsection (b) of any right of contribution or indemnity he may have under law in existence at the time of the occurrence, nor shall anything in this section restrict any right which any person (or class of persons) may have under any statute or common law to seek enforcement of any standard or requirement or to seek the imposition by any State and federal authorities of civil and criminal sanctions. If the principal action was decided on the basis of strict liability, the principles of strict liability shall also apply in any action for contribution or indemnification arising out of the same hazardous waste occurrence.

(h) If any provision of this section or its application to any person or circumstances is held invalid by any court of competent jurisdiction, the invalidity will not affect other provisions or applications that can be given effect without the invalid provision or application; and to this end the provisions of this act are severable."

Sec. 4. G.S. 1-52(16) is amended by changing the second sentence to read as follows:

"Provided that no cause of action shall accrue more than 10 years from the last act or omission of the defendant giving rise to the cause of action; except that for any cause of action arising out of a hazardous waste occurrence as defined in G.S. 130-166.16(24) no cause of action based on strict liability shall accrue more than 30 years from the last act or omission of the defendant giving rise to the cause of action."

Sec. 5. This act shall become effective October 1, 1983.

Mr. Levitas. The final member on the panel we would like to hear from is Ms. Dee Long of the Minnesota House of Representatives.

Ms. Long, we look forward to your presentation.

Ms. Long. Thank you, Mr. Chairman, members of the committee. I want to thank you for the opportunity to be here today to discuss this very substantial issue of great importance to all of us.

I have been asked to address a bill which passed the Minnesota legislature last spring and was signed by the Governor and is now law. The Minnesota bill addresses victim compensation in terms of statutory mechanisms rather than an administrative fund remedy.

Minnesota first considered a State "mini-Superfund," if you will, in 1981. The bill introduced at that time was patterned after S. 1480, the Federal Superfund bill which was not adopted by Congress. This bill contained liability provisions not only for cleanup of hazardous waste sites but personal injury and economic loss damages as well. That bill was heard that particular session in only one committee of the Minnesota House and one committee of the Minnesota Senate.

In 1982, after extensive work over the interim, the bill was heard in 12 committees and subcommittees and passed both the Minnesota House and Senate by wide margins, but was vetoed by then Governor Quie. Again after extensive interim work a revised bill again passed the legislature in 1983 and was signed into law by a new Governor on May 10 of this year.

From the time the bill was first introduced until it was finally enacted, the bill underwent intensive debate, fairly extensive amendment, and was the object of fairly significant controversy of whether we needed the bill.

The citizens in Minnesota have become increasingly aware of the serious threats to public health and the environment posed by the discovery of hazardous wastes not only at sites such as Love Canal in New York, but at sites within our own State. The FMC site immediately north of the city of Minneapolis was rated by the Environmental Protection Agency as the No. 1 site in the Nation.

Following passage of this act, that site has been cleaned up. At the time the bill was being considered by the 1982 legislature, the Minnesota Pollution Control Agency had identified 49 contaminated hazardous wastesites within the State in need of cleanup; that number has now grown to 61, and it is anticipated it will continue to grow. Some of these sites are causing serious pollution to ground water now. Many others will do so in the future if remedial action is not taken.

The Pollution Control Agency estimates it will cost $50 million to cleanup these sites. But that is only part of the problem. Injured victims also face substantial costs. The State Superfund Act addresses these problems, first, by giving the State authority to clean up hazardous waste dumps when there is no one else willing or able to do so. Second, it creates a fund to finance that cleanup. Third, it establishes a clear standard of strict liability for hazardous waste releases in order to make sure that dump sites are cleaned up and victims are compensated for their losses or injuries.

There was actually very little disagreement over the need to cleanup or need for a funding mechanism. The controversy centered specifically on how to deal with the issue of strict liability and over the retroactive application of this liability.

Industry in Minnesota did not seem to oppose applying strict liability for cleanup, but they did object to being held strictly liable for the injuries they cause to people. When the Federal law was passed, it did not address the issue of how to handle compensating victims for personal injury or economic loss. Instead, a blue ribbon panel of noted legal scholars was established to study the issue—this was the 301(e) study group established under CERCLA.

Last fall the panel issued its report; it recommended that States enact laws establishing strict liability for releases of hazardous substances which cause personal injury or economic loss. I am not sure if you heard from Prof. Frank Grad, but one of the bases for the veto in 1982 was the question of constitutionality. That study, and specifically Professor Grad has found there is no constitutional bar for imposing strict liability retroactively.

The Minnesota law does apply retroactively for cleanup and applies retroactively in a limited way for personal injury and economic loss. In its application to personal injury and economic loss, it does not apply for a substance placed in a facility wholly before January 1 of 1960. In addition, there is an additional defense for substances placed between that date and January 1 of 1973. Strict liability will not apply if the defendant can prove that the activity was not an abnormally dangerous one.

The Federal study group also recommended that joint and several liability be applied. The Minnesota law applies full joint and several liability for cleanup but there is only a limited application for personal injury and economic loss; defendants can be held liable for no more than two times their apportioned share. I yiew this as a part of the bill which needs strengthening. I think that, rather than putting a cap on liability for all responsible parties, it would be preferable to establish some level of de minimis exception. For example, a party who could demonstrate that he was responsible for less than 5 percent of the problem could be held liable for no more than his share of the apportioned damages.

The 301(e) study group also noted that proving causation and related evidentiary problems made it extremely difficult for injured victims to be compensated. In addition, the Minnesota authors looked at the findings of the six studies, the study which was prepared for the Congressional Research Service of the Library of Congress during the 1980 Federal Superfund debate, and again were convinced of the extreme problems resulting from causation issues.

The Minnesota Act does have a causation section. Substantial controversy also attached to this part of the bill. The section which was finally adopted as considerably weaker than that which was originally introduced. As it stands now, the law prohibits a directed verdict if the plaintiff can demonstrate a causal link between the defendant, the hazardous release and plaintiff's injury. There is no shift in the burden proof. That remains with the plaintiff, but it is now somewhat easier for the injured victim to get his case before a jury.

The 301(e) study recommended a federally established reviewing agency which was to refer to scientifically established toxic substance documents which show that injury results from such exposure. I am hopeful that Congress will adopt these proposals. It is clear to many of us in Minnesota that the causation issue remains a problem, at both State and Federal levels.

The study group also pointed to the problems which exist in terms of statutes of limitations. The Minnesota courts had already adopted a discovery rule, and we extended this actually in line with a Supreme Court finding for the statute; 6-year statute is common in Minnesota law. The statute of limitations under our Superfund bill stipulates that the cause of action commences, should be determined when the plaintiff discovered the injury or the loss, whether the personal injury or disease sufficiently manifested itself and when the plaintiff discovered or using due diligence should have discovered a causal connection between the injury, disease or loss.

Whether or not we have done enough in Minnesota law to assist injured victims in obtaining adequate compensation remains to be seen. The law has just gone into effect (July 1, 1983). At this time, the only case of which we are aware for personal injury and economic loss is a request to amend a suit which was brought before this bill was passed, and that is currently under consideration.

Assessment of the effects of the law on adequately compensating victims is to be done in a study which will be presented to the legislature next year. That will look at whether we ought to have a State fund and, if so, what form that would take.

I would like to address one additional aspect of the debate over the Minnesota Superfund. The assertion was made by some representatives of the insurance industry and by some hazardous waste generators that if the bill were passed, insurance would be either unavailable or unaffordable. We investigated the insurance issue in depth and heard from a number of insurance representatives. We heard differing views of the situation. Mr. Leslie Cheek, who I believe has testified before this committee, presented one extreme of the testimony we heard. He opposed virtually any change in the law which would ease the plaintiff's burden in recovering for toxic-related injuries and predicted dire consequences should any such change be made.

A different view came to us through a fairly thorough analysis of the bill done by a vice president of the Fred S. James Co. The study was not requested by us but by responsible parties; that being the Minnesota Waste Association, whose membership includes waste haulers and disposers. I have attached a copy of the study to my testimony and the committee should have it available.

Basically this report found that the Minnesota bill did not create any new liabilities for transporters and disposers other than those they would face either under the common law or under Federal law. A number of changes, however, were made in the bill to address the concerns of the insurance industry. We satisfied a number of representatives, but not all of them. In the final analysis, the legislature was convinced that passage of the bill would not make insurance unavailable or unaffordable to Minnesota companies. Since its passage we have seen no evidence indeed that it has.

Although there will still be questions raised regarding insurance and social and economic concerns, I think it is timely, appropriate and just for Congress to enact a Federal cause of action for third party claims arising out of releases of hazardous substances. Specifically, a clear legal standard of strict, joint and several liability needs to be established.

Also, I think the issue of causation needs to be addressed at the Federal level. I would recommend that the committee look at, as I am sure you already have, the model statute proposed by the Environmental Law Institute.

In regard to causation, I think it is imperative for Congress to fund Government research which can come up with scientifically established toxic substance documents that show the causal link between various injuries or diseases and toxic substances. This is an area which should have received greater attention several years ago. It is true that CERCLA has a provision that calls for monitoring hazardous waste dumps for their health effects on adjacent communities. But EPA has given this such low priority that no useful information has been gathered. It is beyond the means of individual citizens and individual States to do the kind of scientific and medical research that needs to be done—provision must be made in the law for the Federal Government to do it and to do it with all deliberate speed.

Although I know that there are many questions yet to be resolved regarding the establishment of a Federal administrative fund, I tend to support its creation in the context of a two-tiered approach as recommended by the study group. The funding for this certainly is up to Congress to decide. However, since the cleanup needs are so great, I believe it is not sufficient to simply extend the present Superfund tax to 1990 and have that money available for victim compensation. I believe that that money is needed for cleaningup the sites.

In conclusion, I support a two-tiered approach to victim compensation: (1) compensation to be paid from a Federal fund administered by the State and comprised of money generated by an industry tax—and possibly general revenue funds, and (2) redress through the State or Federal tort system with a Federal cause of action for third party claims enacted as part of the Federal Superfund statute.

These actions would go far in addressing the question of who should bear the costs of hazardous waste releases. Congress does not have the option of deciding whether there are going to be high costs for hazardous waste releases or whether someone is going to have to pay them. Regardless of what Congress does, or fails to do, the costs will be there and they will be borne by someone. Congress can move to make sure that the burden does not fall on innocent victims by acting affirmatively on these recommendations.

[The statement of Ms. Long follows:]

TESTIMONY OF DEE LONG, MEMBER, MINNESOTA HOUSE OF REPRESENTATIVES

COMPENSATING PERSONS EXPOSED TO THE RELEASE OF HAZARDOUS SUBSTANCES

Minnesota first considered a state "Superfund" in 1981. The bill introduced at that time was patterned after S. 1480, the federal Superfund bill authored by Senator John Culver which was not adopted by Congress. This bill contained liability provisions not only for cleanup of hazardous waste sites, but for personal injury and economic loss damages as well. The bill was heard in only one committee during that legislative session.

In 1982 it was heard in 12 committees and subcommittees, passed both the House and Senate by wide margins, but was vetoed by the Governor. The bill again passed the Legislature in 1983 and was signed into law by the Governor on May 10.

From the time when the bill was first introduced until it was finally enacted, the bill underwent intensive debate and was the object of significant controversy. What motivated the proponents of the bill to stick with the effort to get it passed and what convinced the Legislature that it was what was needed at this time?

Citizens in Minnesota have become increasingly aware of the serious threats to public health and the environment posed by the discovery of hazardous wastes not only at sites such as Love Canal in New York, but at sites within our own state, such as St. Louis Park, Fridley, New Brighton, and Oakdale. At the time the bill was being considered by the 1982 Legislature, the Minnesota Pollution Control Agency had identified 49 contaminated hazardous waste sites within the state in need of cleanup; that number has now grown to 61. New sites are being added at the rate of one a month. Some are causing serious pollution to groundwater now. Many others will do so in the future if remedial action is not taken.

The Pollution Control Agency estimates it will cost $50 million to clean up these sites. But that is only part of the problem. Injured victims also face substantial costs.

How does the State Superfund Act address these problems? First, it gives the State authority to clean up hazardous waste dumps when there is no one else willing or able to do so. Second, it creates a fund to finance that cleanup. Third, it establishes a clear standard of strict liability for hazardous waste releases in order to make sure that dump sites are cleaned up and victims are compensated for their losses or injuries.

There was usually very little disagreement over the need to clean up or for a fund to finance that cleanup. The controversy centered on how to deal with injured victims. Under existing law, it had been virtually impossible for persons harmed by hazardous substances to recover their loss. This act makes recovery only difficult instead of impossible.

Specifically, the controversy centered primarily over the issue of strict liability. Strict liability means that those parties responsible for producing an disposing of hazardous wastes are held legally liable for any damages that result even if actual negligence cannot be proved.

Risks from hazardous substances can never be completely eliminated. A person engaged in an activity involving hazardous substances may take all precautions that are humanly and technologically possible (i.e., he is not negligent) and yet an innocent person may still be injured as a result of exposure to a release. Those supporting the state Superfund believe that question this raises is one of fundamental fairness—should the loss fall on the innocent victim who is injured or on the person who created the risk? The policy of the act is that the loss should fall on persons who create the risk and who are in the best position to control the causes of injury. Thus, those who profit from the use of the substances will bear the cost of any injuries, not the innocent victims of those injuries.

Industry in Minnesota did not seem to oppose applying strict liability for cleanup. But they did not object to being held strictly liable for the injuries they cause to people. When the federal law was passed, it did not address the issue of how to handle compensating victims for personal injury or economic loss. Instead, a blue ribbon panel of noted legal scholars was established to study the issue—this was the 301(e) Study Group established under CERCLA.

Last fall the panel issued its report; it recommended that states enact laws establishing strict liability for releases of hazardous substances which cause personal injury or economic loss. Our state Superfund does that. The panel did not recommend for or against retroactive application; it left that decision to the states. However, the head of the study found that there is no constitutional bar to imposing strict liability retroactively, but rather that it is essential "because otherwise the victims would be without any effective remedy."

The Minnesota law does apply retroactively for cleanup and it has limited retroactive application for personal injury and economic loss.

The 301(e) Study Group also recommended that "joint and several liability was a useful and necessary rule to allow recovery by a plaintiff who had a valid claim, but who could not pinpoint the precise contribution to his injury by each of several defendants. Joint and several liability and the adoption of an expanded approach to concert of action would allow the allocation of liabilities among a group of defendants by the defendants themselves."

The Minnesota law applies full joint and several liability for cleanup but there is only a limited application for personal injury and economic loss; defendants can be held liable for no more than two times their apportioned share. I view this as a part of the bill which needs strengthening. I think that, rather than putting a cap on liability for all responsible parties, it would be preferable to establish some level of de minimis exception. For example, a party who could demonstrate that he was responsible for less than 5% of the problem could be held liable for no more than his share of the apportioned damages.

The 301(e) Study Group also noted that proving causation and related evidentiary problems made it extremely difficult for injured victims to be compensated. The Group found that "regardless of the cause of action—whether trespass, nuisance, negligence, riparian rights, strict liability or any other—the establishment of the causal connection between the exposure to hazardous wastes and the injury or disease suffered remains a difficult evidentiary problem because of long latency periods and the variety of exposures from one or more disposal sites or other potential causes of injury or disease."

The Study Group recommended that "the several states enhance and develop common law and statutory remedies, and that they remove unreasonable procedural and other barriers to recovery in court action for personal injuries resulting from exposure to hazardous waste, including rules relating to the time of accrual of actions, joinder of parties, apportionment and proof of causation."

In addition to the findings of the 301(e) Study Group, the authors of the Minnesota Superfund also found evidence for the need to address the issue of causation in the Congressional case study of six states on 3,600 incidents of toxic substances-related pollution. (Six case studies of compensation for toxic substances pollution: Study prepared by the Environmental Law Institute for the Congressional Research Service of the Library of Congress, at the behest of the Senate Committee on Environment and Public Works during the 1980 federal Superfund debate over victim compensation.) The summary of the Six Case Studies notes that:

"At least three things are immediately apparent from this study of harm and compensation in six states. First, the legal mechanisms in the states studies are generally inadequate for redressing toxic substances-related harms. Traditional tort law presents substantial legal barriers to recovery. For example, for chronic long-latency diseases associated with exposure to toxic substances it is difficult to develop legal proof that a specific substances was the proximate cause of particular injuries.

"Second, seeking compensation for pollution-related injuries is usually cumbersome, time-consuming, and expensive. Private parties may be unable to establish the complex technical findings for obtaining redress. Thus, injured parties in these six states filed few cases and rarely obtained final judgments. Of over 50 damage suits noted, only one was even partly resolved by judgment; the others were settled.

"Finally, and perhaps most important, the result of these difficulties is that the compensation ultimately provided to the injured parties in these cases is generally inadequate. Although the number of cases studied is small, this study suggests that the available avenues of legal redress and the compensation provided for environmenal pollution in other states may also be inadequate."

The Minnesota act does have a causation section. Substantial controversy also attached to this part of the bill. The section which was finally adopted was considerably weaker than that which was originally introduced. As it stands now, the law prohibits a directed verdict if the plaintiff can demonstrate a causal link between the defendant, the hazardous release and the plaintiff's injury. There is no shift in the burden of proof—that remains with the plaintiff, but it is now somewhat easier for the injured victim to get his case before a jury.

I think the causation section of the Minnesota law does not go far enough in easing the burden of proof on claimants. The 301(e) study proposed that they would merely have to prove an injury and exposure to a toxic substance, not a cause and effect relationship. A federally-established reviewing agency was to refer to scientifically established toxic substance documents which show that injury results from such exposure. I am hopeful that Congress will adopt these proposals. It is clear to many of us in Minnesota that the causation issue remains a problem, at both state and federal levels.

"Proving causation is clearly the most difficult factor in a hazardous waste suit and presents a burden so great that common law actions in this area are *proven losers.*" (W. Rogers, *Environmental Law,* Sec. 61.0 at 694 (1977); see Pfennigstorf, Environment, *Damages and Compensation, Am. B. Foundation Research J.*, 347, 353 (private common law actions not viable alternative to administrative controls). See also Trauberman, "Compensating Victims of Toxic Substances Pollution" An Analysis of Existing Federal Statutes, 5 *Harvard Law Review* 1,3 (1981).)

"For many parties the common law will prove inadequate to deal with the unique problems presented in a hazardous waste case. Because of the scientific uncertainty which surrounds this entire area a plaintiff will likely find it impossible to obtain evidence that will satisfy the traditional standards of proof." (Christine Ver Ploeg, *Suing on Behalf of the Victim of Hazardous Wastes: Minnesota Common Law and Statutory Relief*, (1982), (from a report for William Mitchell College of Law), p. 88.)

The 301(e) Study Group also pointed to problems which existing statutes of limitations could present to injured victims. There was little or no controversy on this issue when the bill was being debated. As adopted I believe the Minnesota law fully addresses the issues raised by the 301(e) Study Group and thus I believe the Minnesota statute could serve as a model for a federal law. It provides:

"No person may recover pursuant to sections 1 to 15 unless the action is commenced within six years from the date when the cause of action accrues. In determining when the cause of action accrues for an action to recover damages for death, personal injury or disease, the court shall consider factors including the following:

"(a) When the plaintiff discovered the injury or loss;

"(b) Whether a personal injury or disease had sufficiently manifested itself; and

"(c) When the plaintiff discovered, or using due diligence should have discovered, a causal connection between the injury, disease, or loss and the release of a hazardous substance."

Whether or not we have done enough in the Minnesota law to assist injured victims in obtaining adequate compensation remains to be seen. The law has just gone into effect (July 1, 1983). At this time, the only case of which we are aware is a request to amend a suit which was brought before this bill was passed. The request is to add a count under the provisions of the Environmental Response and Liability Act (Minnesota Superfund).

An assessment of the effects of the law on adequately compensating victims is to be done in a study which will be presented to the Legislature next year. That same study will also make recommendations on the creation of a victims' compensation fund, including the appropriate scope of compensation, methods of financing compensation, and whether the fund should be established or administered at the federal or state level and the appropriate degree of state and federal cooperation in providing compensation. This study was mandated in Section 30 of the state Superfund law, Chapter 121, *Laws of 1983*.

I would like to address one additional aspect of the debate over the Minnesota Superfund. The assertion was made by some representatives of the insurance industry and by some hazardous waste generators that if the bill were passed, insurance would be either unavailable or unaffordable. We investigated the insurance issue in depth and heard from a number of insurance representatives. We heard differing views of the situation. Mr. Leslie Cheek, who is Vice President for Federal Affairs of the Crum and Forster Insurance Companies and speaking for the American Insurance Association, presented one extreme of the testimony we heard—he opposed virtually any change in the law which would ease the plaintiff's burden in recovering for toxic-related injuries and predicted dire consequences should any such change be made. A different view was presented in a thorough analysis of the bill which was done in a study entitled "Availability of Pollution Liability Insurance Under Proposed State Superfund Legislation." The paper was prepared by Rodney J. Taylor, Vice President, National Services Division, Fred S. James and Company, Chicago, Illinois. The study was not requested by legislative staff, but by potential responsible parties—the Minnesota Waste Association, whose membership includes waste haulers and disposers. Because the study seemed to be balanced and comprehensive, I have attached a copy of it. Basically, Mr. Taylor found that the Minnesota bill did not create any new liabilities for generators, transporters and disposers than what they would likely face under common law. In addition, it seems to me he put the role of insurance in perspective when he stated:

"It is important to recognize that insurance cannot solve all of the problems associated with the disposal of hazardous substances. The current crisis in waste disposal in this country is a complex problem brought about by many factors including decades of improper disposal practices. These problems will only be resolved by decades of attention to correcting past errors and a significant financial commitment to restoring what has already been damaged. Insurance for pollution liability may be a part of the solution to these problems, but it obviously is only a small part."

A number of changes were made in the bill to address the concerns of the insurance industry. We satisfied a number of representatives, but not all of them. In the final analysis, the Legislature was convinced that passage of the bill would not make insurance unavailable or unaffordable to Minnesota companies. This belief seemed to be borne out in a presentation made by William A. Mahoney, Vice President, Marsh and McLennan, Incorporated, New York, at a hazardous waste seminar held in Minnesota on May 23, 1983 (two weeks *after* the bill had passed). Mr. McLennan noted that:

"Capacity, which was a problem originally, is no longer. There are many more companies willing to write the coverage and capacity is steadily increasing. Originally an individual company would consider five million and ten million annual aggregate as its capacity for any one risk. But we are seeing an increase to ten million and twenty million annual aggregate with additional reinsurance capacity coming about in a rather expeditious fashion. Considering that two years ago there were only three underwriters interested in writing the gradual pollution while today there are at least a dozen, it is obvious that there has been substantial growth.

"Hopefully, as more underwriters become interested we will see more competitive pricing, although pricing has not been without fair competition. Premium costs have been set upon the basis of the corporation's sales and credits are granted for self-insurance and deductibles, as well as the underwriter's consideration as to how well the firm is managing its hazardous waste.

"Dealing with hazardous waste is to be involved with the concept of loss prevention and if a firm is able to demonstrate that it is loss prevention conscious, it probably will warrant a premium consideration when the terms of the contract are being negotiated with the underwriter.

"This entire subject of environmental impairment liability insurance is one which is constantly changing. There is no doubt that this mode of constant change will continue for the next several years.

". . . . The policy forms are changing, the excess markets are seriously considering getting involved, and there is every indication that the entire insurance industry—from the primary placement up through the excess layers—will be ready and willing to accept pollution liability insurance risks. Much the same as they accept any other form of insurable risk."

Although there will still be questions raised regarding insurance and social and economic concerns, I think it is timely, appropriate and just for Congress to enact a federal cause of action for third party claims arising out of releases of hazardous substances. Specifically, a clear legal standard of strict, joint and several liability needs to be established.

Without a definite and specific legal standard for hazardous waste cases, each case will require lengthy and expensive court action. Such legal action is beyond the means of the average person. The Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) should be amended to set out legal responsibilities for personal injury and economic loss clearly and with certainty for all parties involved, thus making it more likely that innocent victims will be justly compensated.

As I noted above, the issue of causation needs to be addressed. I recommend that the model statute proposed by the Environmental Law Institute in its study on compensating victims be used as the basis for federal legislation. (See Jeffrey Trauberman, Statutory Reform of Toxic Torts: Relieving Legal, Scientific and Economic Burdens on the Chemical Victim, Environmental Law Institute, Washington, D.C., 1983).

In regard to causation, I think it is imperative for Congress to fund government research which can come up with scientifically established toxic substance documents that show the causal link between various injuries or diseases and toxic substances. This is an area which should have received greater attention several years ago. It is true that CERCLA has a provision that calls for monitoring hazardous waste dumps for their health effects on adjacent communities. But EPA has given this such low priority that no useful information has been gathered. It is beyond the means of individual citizens and individual states to do the kind of scientific and medical research that needs to be done—provision must be made in the law for the federal government to do it and to do it with all deliberate speed.

Although I know that there are many questions yet to be resolved regarding the establishment of a federal administrative fund, I tend to support its creation in the context of a two-tiered approach as recommended by the 301(e) Study Group. The level of compensation suggested by the 301(e) Study Group also would seem to be fair and appropriate; full recovery of medical costs plus two-thirds of lost job income up to a monthly limit of $2,000 which could be adjusted to reflect cost-of-living changes.

The monies for the fund should be generated by a tax on industry—Congress may want to consider whether this tax should be on chemical feedstocks, like the current Superfund tax, or whether it should be a waste end tax along lines established in the Minnesota Superfund and like that set forth in Senator Gary Hart's bill, S. 860. It is possible that a combination of the two would be the best approach. Since cleanup needs are still so great, I believe it is not sufficient to simply extend the present Superfund tax to 1990 and have the money that would be raised used for compensating victims. The present tax does need to be extended—but it needs to be used for cleaning up hazardous waste sites. The tax for compensating victims must be in addition to the cleanup tax. Rather than set this new tax at an unduly burdensome level, it may be reasonable to appropriate some general revenue funds to augment the tax receipts. Senator Hart's bill, S. 860, would authorize $190 million per year in federal funds to be added to the Superfund tax program for each of the next 10 years.

In conclusion, I support a two-tiered approach to victim compensation: (1) Compensation to be paid from a federal fund administered by the state and comprised of monies generated by an industry tax (and possibly general revenue funds) and (2) Redress through the state or federal tort system with a federal cause of action for third party claims enacted as part of the federal Superfund statute.

These actions would go far in addressing the question of who should bear the costs of hazardous waste releases. Congress does not have the option of deciding whether there are going to be high costs for hazardous waste releases or whether someone is going to have to pay them. Regardless of what Congress does, or fails to do, the costs will be there and they will be borne by someone. Congress can move to make sure that the burden does not fall on innocent victims by acting affirmatively on the recommendations I have outlined above.

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November 28, 1983

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Italiano
PUBLIC WORKS AND TRANSPORTATION

Mr. Joe Italiano
Editor
Subcommittee on House Investigations & Oversight
Committee on Public Works & Transportation
2165 Rayburn House Office Building
Washington, D. C. 20515

Re: Subcommittee on Investigations & Oversight
Hearings of November 8 & 9, 1983

Dear Mr. Italiano:

I previously forwarded to you the American Insurance Association's comments in response to the testimony heard by the Subcommittee on November 8 and 9.

With his permission, I am forwarding to you a copy of a letter dated November 23 received from William Mahoney of Marsh & McLennan with reference to the testimony of Representative Dee Long on November 8. If possible, I would hope that you would make this letter part of the record of your Subcommittee hearings on Hazardous Substance Victim's Compensation as I believe this letter accurately places the context for Mr. Mahoney's remarks referred to by Ms. Long.

Very truly yours,

M. Patricia Casey

M. Patricia Casey
Associate Counsel

MPC/dt
Enc.

cc: Mr. Sante Esposito
Mr. Chris Goebel
Mr. William W. Suttle
Mr. James L. Kimble

WILLIAM O. BAILEY, CHAIRMAN   PETER LARDNER, VICE CHAIRMAN   EDWARD H. BUDD, VICE CHAIRMAN   T. LAWRENCE JONES, PRESIDENT

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William A. Mahoney
Vice President

November 23, 1983

Ms. M. Patricia Casey
Secretary
American Insurance Association
85 John Street
New York, New York 10038

Re: Victim Compensation Proposals

Dear Ms. Casey:

I appreciate the concern you expressed to me in our telephone conversation of November 21st regarding certain testimony apparently given to a governmental committee in which I was quoted extensively.

On May 23rd of this year I was invited by the Minnesota Waste Management Board to address the subject of Environmental Pollution Liability Insurance which I did.

Attached is a capsulized version of my my discussion. My comments addressed the EIL markets, the various insurance forms and some of the industry's concerns with respect to the general subject of pollution coverage.

Past history of the coverage, while admittedly brief, was touched upon and I did distribute written material which contained information supporting the development and the changing scene in respect to this "new" insurance coverage beginning approximately in 1980.

I was not asked, nor did I attempt to analyze the Minnesota Statute, which had been enacted only a few weeks prior to this public conference. I positively made no attempt to characterize my remarks as indicative of the insurability that would support "Victim Compensation Legislation". The subject of Victim Compensation is in such an embryonic stage that I could not attempt to relate insurance to a subject which has within it the very important question of "no fault" responsibility.

Marsh &
McLennan

My comments should not ever be construed to have application to such a complex subject which is recognized as such by Sections 29, 30 and 31 which requires studies to be made with respect to sections concerning liability and includes a study of the insurance industry, as well as other affected industries. The first of these reports are not due until July 1, 1984 and some are due in 1985.

I have spoken with Sue Robertson of the Minnesota Legislative Commission on Waste Management, since she participated in the panel discussion. She assured me that the comments attributed to me by the witness were not in answer to any legislative consideration of "Victim Compensation". She has offered to send me the testimony, as you have.

I regret the confusion that appears to be surrounding the question and it is also unfortunate that I was not contacted before the comments were made in a committee environment that could possibly be misinterpreted.

I have consistently underscored in any comment I have made, that this particular market is constantly changing and this observation is never more clearly demonstrated than what is occurring in some of the insurance forms at this very moment and which will result in a restriction of coverage which was available last May.

I would be more than pleased to assist in clearing up any confusion which has been created by someone else using my words, but not necessarily using them in the proper reference.

Sincerely,

William A. Mahoney

WAM:re
Encl.

Long-Term Responsibilities

Legal Liability at Minnesota's Hazardous Waste Disposal Site

Who should bear the legal liability for a potential release of hazaruous wastes from the disposal facility to be sited by the Waste Management Board: hazardous waste generators, the disposal facility operator, the state, or a combination of any of these parties?

This was the question moderator Marcia Gelpe, a professor at William Mitchell College of Law, posed to the panel session entitled "What Long-Term Safeguards Are Needed?"

In May 1983, the Legislature approved a new state Superfund bill designed to provide for speedy clean-up of Minnesota's hazardous waste dump sites and statutorily define the legal liability for property damage and personal injuries caused by past and future releases of hazardous wastes.

Prior to passage of the bill, business and industry groups lobbied heavily against the "strict, joint, and several" legal liability provisions it contained. "Strict" liability enables a victim of a hazardous waste release to sue for damages without having to show negligence on the part of the generator of the wastes. And, generally, "joint and several" liability provides that, if a number of generators deposited wastes at the problem site, each is potentially liable and could be required to share in the damages.

The bill also requires generators to be "strictly" liable for all hazardous wastes disposed since 1973 and makes it easier for victims injured by wastes disposed between 1960 and 1973 to recover damages if they can prove the generator's hazardous waste activities were "abnormally dangerous."

After Governor Rudy Perpich signed the Superfund bill into law, Lewis Lehr, chief executive officer of 3M, Inc., stated in a letter to Perpich that: "3M believes the liability provisions of the present bill will make it impracticable for waste generators to use any designated hazardous waste site in Minnesota."

Panelist G. Robert Johnson, a Minneapolis lawyer and lobbyist for 3M, agreed with Lehr's assertion.

"I don't know how any business could justify the decision to use a site that it didn't locate, didn't design, and doesn't operate and yet. . .bear liability for what happens at that site through no fault of its own," Johnson said.

Rather than requiring businesses that use the disposal facility to share liability for any damages, Johnson believes the State of Minnesota should bear the ultimate liability for the facility it will site and own. "It would seem to me, that for generators to utilize the Waste Management Board process of treating wastes to the fullest extent, you have to hold out some sort of carrot — such as termination of liability. After all, it's going to be very expensive for generators to fully process their wastes," he said.

Solving a complex issue

State Representative Dee Long, who authored the House version of the Superfund bill, disagreed. "I think that is a very simplistic solution to a terribly complex issue," she said.

According to Long, legislators were not willing to put such language into the Superfund law in 1983 because such issues as control and regulation of the facility, availability of insurance, and the financial capability of businesses who use the facility have not been addressed. Instead, she said, the Legislature included a provision in the bill requiring the Waste Management Board to make recommendations to the Legislature by November 1 regarding the allocation of liability among owners, operators, and users of Minnesota's hazardous waste disposal facility.

To Address [illegible]

Long said a "great deal" of damage could have been done by incorporating into the bill a provision terminating the liability of hazardous waste generators once their wastes reach the state-sited facility. Such a provision would have created "innumerable" problems for the Waste Management Board's siting process and residents in the areas the Board is considering as potential disposal sites, she said.

"We're calling for a very thorough study of a complex problem. And what I don't understand is how the industries in this state that have said they will never use a site in Minnesota have chosen to totally disregard the provision for a study of this issue," Long added.

Chuck Riley, a member of the audience from the Michigan Department of Natural Resources, warned Minnesotans to be cautious in determining liability for the disposal facility.

Riley explained that in 1979 the State of Michigan attempted to absolve hazardous waste transporters and generators of liability for their wastes once they were disposed at a state-approved facility. However, Riley said, the EPA objected to this provision and the Michigan attorney general determined that it was not legally possible to absolve generators and transporters of legal liability for their wastes.

A similar effort to absolve disposal facility operators of their liability, in which the State of Michigan would assume ownership of the facility after it was closed, was ruled unconstitutional by the State attorney general, Riley said.

"There are a lot of implications to transfer of liability and I think you ought to think long and hard on it before you put it down in writing and have someone overturn it in court," Riley concluded.

Rep. Dee Long (DFL-Minneapolis)

[illegible]

[illegible]

[illegible]

"You have to hold out to industry some sort of carrot"

Prof. Marcia Gelpe

Robert Johnson

"That's a simplistic solution to a complex issue"

William Mahoney

The view of an underwriter

Panelist William Mahoney, vice president of the New York insurance brokerage firm of Marsh and McLennan, explained that an insurance company might look more favorably on a disposal facility operator that shares legal liability with a state or municipal government. "Anytime a disposal facility operator can show some minuses coming off the total loss picture, the facility operator will get some benefit in containing premium costs," Mahoney said.

Mahoney also said that it would be possible for a generator who deposits waste at a facility to "piggyback" on the operator's insurance, although many operators would be hesitant to do this because it would reduce their limits of coverage.

Mahoney noted that, while the federal Superfund law does not contain a provision for "strict, joint, and several liability" of hazardous waste generators whose wastes cause personal injuries, federal officials have indicated they will enforce the law as though it contained such a provision. Initially, this caused some concern among insurance brokers around the nation.

"For the most part, those concerns have been put aside by the insurance industry. We have found that most underwriters want to insure for legal liability, and if it is strict liability, then that is what they will insure for."

NOT
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Another panelist, Sue Robertson of the Minnesota Legislative Commission on Waste Management, pointed out that because federal officials intend to enforce a strict, joint, and several legal liability standard, Minnesota generators who try to avoid such liability in Minnesota by shipping their wastes to facilities in other states could end up with the same liability anyway.

In essence, Robertson said, Minnesota's liability standard is very similar to the federal standard.

Mahoney also discussed factors underwriters evaluate in determining whether to insure a firm that handles hazardous wastes. "One of the most essential points about this type of insurance is loss prevention," he said. "If a firm deals with hazardous wastes and is sloppy, it is not going to get insurance quite as readily as a 'clean' firm giving its all to loss prevention."

Mahoney stressed that insurance is not the answer to the hazardous waste problem, but that it could assist in restoring lost public confidence in hazardous waste management techniques. "Insurance can be written, but it is not going to be written for every single facility," he said. "However, if a facility is properly located, carefully engineered, and the operators are conscious of loss prevention, it stands a better chance of getting insurance."

Mr. LEVITAS. Thank you very much, Ms. Long. Your closing comments are so very apropos and right on target. The costs of hazardous waste in this country is a fact. How that cost is borne and how enormous it gets to be is still something we can deal with. But the $40 to $50 billion we are now looking at just for cleanup doesn't begin to address the extended impact both on our natural resources, the aquifers, the other water resources, and, as all three of you are addressing today, the individuals who have been exposed to this.

Let me start with you, if I may, Ms. Long. You note on page 7 that the statute enacted by the Minnesota Legislature "fully addresses the issues raised by the 301(e) Study Group." And you also note on the same page that the Minnesota law is still too new to evaluate in terms of its effectiveness or adequacy.

My question is this: In light of your comments, do you feel that at this time there is further need for the Federal Government to take any action either in terms of providing the Federal cause of action or the two-tier approach which you seem to support, or should we for the time being leave it to the respective States to carry out until we have had some more experience under these efforts that are going on around the country?

Ms. LONG. Mr. Chairman, it is my belief that waiting will not accomplish any substantial purpose. It may be years before the effect of State actions is apparent. While States are gradually taking this approach, you have at that point an inequity among States. I support a Federal cause of action enacted as soon as possible. And I support the two-tiered approach.

Mr. LEVITAS. Mr. Connally, would you comment on that?

Mr. CONNALLY. I would agree with Ms. Long. There are really only a handful of States that are legislating in this area seriously at the present time. There are some unique factors which have happened in those States that have brought it to the attention of the local legislators. Your opening comments were right on point in indicating that there are little time bombs all over the country that haven't gone off yet. So it is a function of when those start popping before those States act.

You ought to move now. I think you ought to recognize in moving now that the area is complex enough that there will be initial errors, as indeed California has, and then come back and do the second- and third-year cleanups as we all do on major pieces of legislation. But I would urge you to go forward.

Mr. LEVITAS. Mr. Hackney?

Mr. HACKNEY. I concur, Mr. Chairman. I might say in addition that this may be an area where the States are following the Congress instead of the other way around, as you indicated sometimes happens, because our own particular orphan site legislation is modeled on the Federal cause of action in this area. We hope that bill will pass also in the 1984 short session.

I think, if I may repeat something I mentioned earlier, perhaps some Federal incentive toward State tort law reform might be in order if a Federal cause of action for personal injuries is not created.

Mr. LEVITAS. Before we get away from this point, one of the problems that I have in thinking in terms of a Federal cause of action is the fact that it means another 1,000 or 10,000 suits in the Federal court system, rather than in the State court system. And, it is certainly an easy response when we in Congress look at problems to say let us pass a law. Let us create this cause of action, whether it is on debt collection or on certain other traditional State tort areas.

Do you see the opportunity of perhaps creating a Federal cause of action but one which could be litigated in the State courts rather than in the Federal courts? Any of you?

Ms. **Long**. Mr. Chairman, I would look favorably on that approach.

Mr. **Levitas**. Mr. Hackney?

Mr. **Hackney**. Mr. Chairman, I noted, in light of your comments, with some interest in the Post this morning there are hearings going on elsewhere up here today on the overload of civil cases in the Federal court system, which is, as you all know, a very serious problem. I think it is imperative that there be access to the State courts in these situations. I think that is where torts are traditionally tried except for unusual situations, and I think that is where these ought to be tried as well.

Mr. **Levitas**. Thank you. Let me ask a more difficult and troubling question. I ask it not just to be the devil's advocate but because we have had rather strong testimony before this subcommittee along these lines.

There are those who claim that there is no scientific evidence that links hazardous waste sites, or for that matter even toxic chemical waste, with physical injury, and that what we are doing, these people charge, is finding a solution for a problem that doesn't exist except by way of perception. We have had scientific witnesses come before this subcommittee and say there is no evidence that living near or on top of a hazardous waste site in fact leads to physical injury and that we are simply making a political response to a scientifically unjustified assumption.

My question is did you have that type of argument presented to your respective State legislatures as you were going through this process? And would you care to comment on the assertion in general; are we in fact fashioning a remedy for a problem that doesn't exist? Why don't we start at this end this time, Mr. Hackney?

Mr. **Hackney**. Mr. Chairman, that argument I suppose has some appeal. I don't think we can ever get away from the problems of proof. You are going to have to prove causation in some way or another or you can't recover unless someone reverses the burden of proof. I don't think that is seriously about to happen.

Now, I would point out, however, that there are many, many other smaller injuries which are perfectly susceptible of proof wherein a manufacturer, transporter, or generator ought to be strictly liable. Those include, for example, tanks of chemicals going down the highway. There ought to be incentives so that whether or not the driver of the truck is negligent and if there is spillage there is a strict liability standard. Generators and transporters are responsible for getting the wastes safely to their ultimate destination, and they are responsible for having the wastes in safe enough containers that, if there is an accident, it won't hurt somebody or get in the water supply. Those kinds of things are perfectly susceptible of proof.

To the extent the argument against such legislation depends on the inability of someone to prove current injuries for something that happened 30 years ago, yes, that is a problem. We can't get away from that. But that doesn't mean we don't need to start and go ahead.

Mr. LEVITAS. Mr. Connally, would you comment on that?

Mr. CONNALLY. Yes. We have had substantial testimony from some of the same folks who spoke to your committee, who did not take that line in terms of the presentation to the California Legislature. There was not the dispute that there is a problem, so much as yes there is and how do we go about it. There was substantial evidence presented that there are extreme difficulties in making that causal connection in terms of the costs of the scientific studies. But there was not the assertion by the credible opponents of the two-tiered system that you have heard here that in fact there was not a problem, and therefore you should not be legislating in this area.

Some of the representatives from the chemical industry would acknowledge merit to rebuttable presumptions of some type if you have proof: One, that there is a toxic there, two, that the person was exposed, and three, that there are medical symptons caused by those toxics, if you capped it with some recovery. They acknowledged that type of presumption was not out of line.

Now, this was not all of them, but there were a few that made this point. So on the contrary, it is an appropriate area to look at, an appropriate area to legislate. It is a real problem. The only thing is all we see now is the head and neck of the camel, and it is going to be another 10 or 15 years before we see the rest of it.

Ms. LONG. Mr. Chairman, I would echo those remarks. We certainly heard many objections throughout the struggle with the Minnesota bill that indeed we were addressing a non-existent problem and what was the need for that. Our rejoinder always was, then what are they worried about since even under our fairly liberal legislation it seemed highly unlikely that unjustified victims would be compensated.

I again echo the comments of Mr. Connally, that I think there are a number of potential cases out there. There may be some actual cases not yet ripe. In the State of Minnesota there are not yet a great number of cases. But the Minnesota Pollution Control Agency really just started keeping a log of these hazardous sites about 3 years ago. And they are finding new ones all the time. I think there is a time lag there. The opponents of the legislation who make that argument do not acknowledge the time lag problem.

Mr. LEVITAS. Thank you. I think that I get from all three of you the feeling that there is sufficient evidence that says if we have to err in this matter, we should err on the side of protecting the public and then take a closer look at it as this epidemiological and other evidence is produced over the years.

Tomorrow our witnesses will include some people from the National Institute for Occupational Safety and Health and from the centers for Disease Control. We will get into some of these questions, but I think the experience you have had is extremely helpful to us.

One last question, and then I will recognize the gentleman from Florida. This is to you, Mr. Connally. A number of witnesses—you touched on it in your last answer—who have testified before this committee have argued that the problem of causation is the plaintiff's, or the victim's most difficult obstacle. It is the highest hurdle that they have to overcome, proving the injury results from exposure to hazardous substances.

Some of these witnesses have suggested adopting concepts like a rebuttable presumption where the burden of proof that there was no harm would shift to the generator rather than requiring the victim to show by a preponderance of evidence that the injury was caused by the particular exposure, and that this type of approach would remove the unreasonable barrier that we have heard testimony about.

Now it is my understanding that the California statute does not change the traditional legal approach to establishing causation. Was this because the California Assembly rejected the argument that the burden of causation is too difficult for the plaintiff to prove, or was there some other reason that led to the decision not to shift that burden of proof?

Mr. CONNALLY. This is an issue over which reasonable people can differ. And there is a very powerful and effective lobby in California from the chemical industry, the petroleum industry, the Manufacturers Association in particular, against changes in this area.

The bill, as introduced, did in fact include some rebuttable presumptions very similar to that proposed by the 301(e) study group. Those presumptions were gutted in the text of the hearings on the bill. Under that, if you do that, you negate the very reason for the first tier remedy at all. If you say we are setting forth a first tier administrative claim remedy because it is too expensive to go to the courts and prove that causal connection, and then you take that same causal requirement and put it in the first tier, the only thing you have done is you have shifted the deep pocket from Du Pont Corp. to the fund, and the proof burden is exactly the same. The cost of meeting that burden is exactly the same.

You have done what, unfortunately, California has done, and that is provide no real remedy at all. You have identified one ineffective remedy and set forward a second equally ineffective remedy. So you have to get at this area of presumptions. If you cannot, if you cannot buy off or convince your colleagues to go with the rebuttable presumption and the entirety of it, at least go the rebuttable presumption and then put some type of cap as to the amount of money you can recover for damages you incur in reliance upon that presumption.

For example, if your claim is vested upon a rebuttable presumption only, you can recover 50 percent of the direct damages you experienced. If you move the continuum away from the rebuttable presumption back to the causal connection, you will deny the remedy; you should not legislate in the area; the first tier is a waste of time; it is a bad, bad trick on the public and the citizens of California are just waking up. If you have that rebuttable presumption in there and if you must grade it based on some damage recovery formula, at least there is something there for the victims that is real.

Mr. **Levitas**. Do either of the others of you have any further comments on that point?

If not, let me say this, Mr. Connally: I totally agree with you. At the very least, the tradeoff for a limitation of liability should be this rebuttable presumption, if not absolute liability of some sort. If we are going to set up a first tier mechanism with some administrative remedy, and a workers' compensation type of approach, then quite obviously there has to be a totally different approach to the proof question for that type of system. Otherwise, as you say, you are playing a trick on the public. And, hopefully the public will wake up in California and realize that in this instance the chemical industry got the best of both worlds and did not even suffer the obligation of addressing the question of compensation even in that first tier.

The gentleman from Florida, Mr. Shaw.

Mr. **Shaw**. Thank you, Mr. Chairman.

I would like to pursue that line of questioning of the chairman. I want to talk a little bit about the rebuttable presumption, which we know—and I think it is absolutely clear—that strict liability and preponderance of evidence or rebuttable presumption are certainly compatible under the law.

I seem to have, however, detected a difference of opinion between Mr. Hackney of North Carolina and Mr. Connally from California as to what the burden of proof should be.

Mr. Connally, how would you draft a rebuttable presumption? Would it be a rebuttable presumption such that someone just comes in with injuries which are classic to exposure to a certain substance, and then shows that he was at some time around that substance; then it would become the responsibility of the Government, the State of California, if you may, to rebut the presumption that that is how he got his injuries? How would you draft such a bill?

Mr. **Connally**. Mr. Shaw, initially Mr. Hackney and I were speaking to burdens of proof in two different systems. Mr. Hackney was speaking to the tier 2, as I understand it, general liability tort recovery mechanism, talking about the strict liability process applying there. My discussion was about the rebuttable presumption to tier 1. I think the rebuttable presumption identified in your study group was a consensus recommendation, and that is pretty startling given the broad makeup of that study group where you prove, one, the existence of the substance, two, exposure, and then three, the individual suffered the kind of medical harms that exposure to that particular substance would bring about in laboratory experiments and so forth. That is the type of presumption that I think you ought to move for.

Ms. Long points out in her testimony—and I think there is credence to that, and the study group mentions it also—that in formulating these presumptions based on exposure there has to be some work done, epidemiological work done of a broad character so that that causal connection in general is proved. But once it is proved that exposure to dioxin produces *x* result, and if Mrs. Jones has been exposed to dioxin and then she has *x* result, then it ought to be the burden of the industry at that point to prove that in fact there is some other substance or something else that is causing *x* result.

In the absence of that occurring, there is no real recovery for Mrs. Jones if she is a small claimant because she cannot incur the individual cost to make that connection herself.

Mr. Shaw. I understand that, and I can appreciate that.

I think that in earlier comments by you and by the Chairman, and certainly through my own knowledge, there are people who come in and simply deny the causal relationship. I think that we can also find that people will say that cigarettes are not injurious to your health.

I am a little curious as to how or where would you find the burden of proof shifting? Someone comes in with exposure to something that could be a cause of lung cancer. So at that point it is shown that that particular individual also smokes. Now, has the burden shifted at that point? Where do you stand?

I don't know how you would draw the legislation to come up with those presumptions. That is what is concerning me.

Mr. Connally. What we do in California in the workers' comp field—it has similar kinds of presumptions in a variety of areas—is that if in fact there are multiple causative factors, then the defendant or respondent has to come in and prove and rebut the presumption that indeed this individual has smoked throughout the entirety of their life; they smoke two packages of cigarettes a day. Based on the fact they have had one or two exposures to this toxic substance, which is carcinogenic, on your lungs, it is not reasonable in this instance to assume and to give them the benefit of the presumption that their lung cancer is caused as a result of that one- or two-incident exposure.

But that presumption can be overcome, and it can be overcome based on the submission of the kind of evidence that you have outlined. But the trier of the fact can look at that evidence and make that determination. But the individual initially has the benefit of that assumption. And it makes all the difference in the world as to whether or not the people, particularly those people that have valid claims, are going to recover.

Now, there is one question that you didn't ask that I would think would be a followup, and that is, are errors going to occur? And the answer to that question is yes. Are some people going to receive benefits that probably should not? And the answer to that question is yes. But if the system is structured properly, it is going to be very, very small. And that is an error risk I think we ought to be willing to accept, given the fact that these people were the people who need and are justified in recovery, of no other basis for recovery, in the absence of that mechanism in existence.

Mr. Shaw. I think, as the chairman said, if you are going to err, err on the side of the victim.

In Federal legislation, where would be your election of remedies where you could pursue a claim in either direction?

Mr. Hackney, would you like to comment on that, or any of the other panelists?

Mr. Hackney. I am not sure of your question, sir.

Mr. Shaw. The question is, if you have a remedy under both Federal law and State law, would it be your understanding that the individual would have to elect which law under which he is going to pursue his claim, and then be bound by that decision and not be able to go back and do some shifting at a later date?

Mr. Hackney. I believe, Congressman, the legislation that I have seen at the State level all refers to the Federal legislation and requires that there be no duplication of payments. As to the exclusivity of it, I am not sure that there is in the legislation that I have seen—and my colleagues can correct me—any prohibition against pursuing damages not covered in another forum. Certainly, if you have an administrative remedy set up for one purpose and another set up for a slightly different purpose, I see nothing wrong with pursuing your full damages in both.

Mr. Shaw. Would anyone else care to comment?

Mr. Connally. I think this is an area where I might disagree with Mr. Hackney. At least with regard to the tier 1 administrative remedies, my hope would be the Federal law would be structured in such a way that there would be an incentive for the States to take advantage of it with some type of matching formula. I believe the study groups recommend 80 percent/20 percent. That would effectively preempt the tier 1 or administrative remedies available at the State level.

As to the tier 2, general liability recovery, then I would agree with Mr. Hackney. It may well have been this is what he was speaking to; that the option as to which one to utilize in terms of the Federal or State remedy should be left to the party, as we do now with a variety of tort laws.

Mr. Shaw. You made the statement, Ms. Long, that the Federal fund would be administered by the State. How exactly would that work, and how would it be funded?

Ms. Long. I think there are several proposals before you now which would entail situations similar to that. I believe there is one proposal which has the Social Security Administration, through the State offices, administering the fund. But I think certainly for purposes of convenience, rather than an administration at the top with claims to be filed in Washington, some sort of State administration would make the most sense.

I certainly don't have any specific details. I think there are a number of mechanisms that could apply.

Mr. Shaw. But you are not necessarily advocating any one particular——

Ms. Long. No, sir; I am not.

Mr. Shaw. Mr. Connally, I have one more question I would like to address to you. You spoke of the California fund containing some $2 million, and to date no one has been successful in tapping that particular fund.

How about your tort law? Is it the same situation with regard to tort cases in the State of California?

Mr. Connally. There have been a number of successful recoveries under tort theories, product liability theories, under kind of a modified strict liability theory. The law in California is still emerging in that area. The *Sindel* case in particular, which involves the concept of applying strict liability and then apportioning the damages as opposed to the joint and several liability theory, appears to be applicable to hazardous waste, but that case has not gotten to the State supreme court yet.

So I think the best way to answer your question is, yes, people are recovering. The courts are moving in the direction of the Minnesota law and North Carolina law with regard to strict liability. We are not there yet. It appears, based upon early court cases, that it will be a strict liability with some type of apportionment or di minimis kickout, as opposed to a joint and several liability, as I understand is contained in the North Carolina law.

Mr. SHAW. The litigant would, of course, have the same problem of proof in a tort case, I assume, under California law as he would have in pursuing an action against the fund.

How do you account for the fact that there have been successful tort cases, but not in the instance of the fund?

Mr. CONNALLY. Very easily. In successful tort cases, you have people that have suffered unbelievably horrible damages—mutations at time of birth, death, permanent physical disability. So that the private attorneys representing those parties can make a hard economic decision that it is worth spending the money to make that epidemiological connection and prove that causal link.

The problem is with the smaller person, the person who has suffered damages maybe even requiring hospitalization, but for a very short period of time, that does not justify the expenditure of $50,000, $75,000, even $100,000 in terms of expert work to prove the individual causal link for that comparatively minor damage.

So it can be done. It is just not cost-efficient for the individual claimant.

Mr. SHAW. Under your tort law, I would assume those type of fees would be recoverable as part of the judgment.

Is that also covered under an action against the fund?

Mr. CONNALLY. No. Under California tort law, that is not recoverable as a separate additional charge. That is part of the recovery as a whole. It is not quantifiable in terms of the medical cost recovered. It is expertise. And that comes out of the judgment award.

For example, if you suffer $1 million in damages as a result of a toxic exposure, and it takes $100,000 to prove a causal connection between that damage and the toxic you were exposed to, you will get a $1 million award. You will have to pay the $100,000 in cost. If you suffer $10,000 in medical damages as a result of the toxic exposure, you can prove that, as well, for $100,000; but that would take the $10,000 that you recover plus another $90,000 out of your own pocket. So no one does it.

That, of course, explains why the two-tier concept, the administrative concept, where hopefully with a smaller level of proof using the rebuttable presumptions, will allow an effective recovery for the person who is injured, but not injured to the degree that justifies this inordinate expenditure of expert fee and research money to prove that causal connection.

Mr. SHAW. Do you think that part of the problem in pursuing the fund would be overcome if the California Assembly was to provide for recovery of expert witness fees, things of this nature, in actions against the fund? I assume that would not go as far as rebuttable presumption but would go a long way toward curing some of the problems in pursuing a cause of action against the fund.

Mr. CONNALLY. That is true. But what is the rationale that says if people bring a $2,000 lawsuit and prevail, that they can recover the full cost of the expert witnesses, when as a practical matter the overwhelming evidence is that those expert witnesses' costs are going to be $75,000 or $100,000? As a matter of public policy, at some point that doesn't make sense.

Mr. SHAW. Excuse me. I said I was going to ask the last question about five questions ago. I am somewhat intrigued by this line of questioning.

Wouldn't you, as an attorney representing someone against the fund, still want to go in loaded for bear with regard to what you are going to do with expert witnesses in case they do rebut your presumption?

Mr. CONNALLY. If we had the rebuttable presumptions, if we could prove it existed, if we could prove exposure, if we could take under the 301(e) language the study that shows that the client is suffering the kind of harm that one would anticipate when being exposed to this, we can go with that. If they come back and rebut our presumption, we will know that in a timely enough fashion that we can deal with that on an individual basis.

The practical reality is that most of those assumptions aren't going to be rebutted because the practical reality is that if the person is suffering the medical symptoms expected to be suffered when exposed to a toxic substance and were in fact so exposed, they are in fact suffering as the law anticipates they will from that exposure. So that although that option to rebut the assumption is there, only in a very small minority of cases would it be exercised.

Mr. SHAW. Thank you.

Mr. CONNALLY. I will just tell you, as a private attorney who practiced not in this area but in other areas involving presumptions, you prepare entirely different in terms of the expenditure of funds, expert witnesses, medical examinations and so forth when you have the benefit of the presumption as opposed to when you do not.

Mr. SHAW. I can understand that. But I know, having practiced law myself, that you always have the possibility that you have to be very concerned that someone is going to rebut that presumption. Then you are going to be looking at a Monday morning quarterback telling you how you should have done your case, which opens up a whole new area of tort law which those of us in practice have to be concerned about, also.

I thank you, Mr. Chairman.

Mr. CONNALLY. Thank you, sir.

Mr. ROWLAND [presiding]. Thank you, Mr. Shaw.

Can you tell me what experience you have had in your State thus far with the laws you now have in place with reference to the number of claims that have been filed? Do you have any information on that?

Mr. HACKNEY. Mr. Chairman, we do not. First of all, the legislation I have discussed here this morning has not been enacted. It has passed only one house.

So to back up and answer the underlying question of how we are doing with our existing tort law, my answer would be somewhat the same as Mr. Connally's—not very well. That is, there are lots of small claims that are going uncompensated because of the costs of prosecuting the actions. With or without the benefit of presumptions, that is going to be somewhat true, because the burden of proof with the aid of these presumptions, as I understand them, as explained by Mr. Connally, and the burden of proof that the plaintiff has in getting to the jury in my State, are not all that different. If you have to prove the existence of the injury, exposure, and that harm usually follows, you have a burden of proof—let there be no mistake about that—even if you have to do it in an administrative hearing.

So the only experience in our State which has caught the public imagination and which, I guess, is one of the reasons we are moving in this area, is that a transformer company took a truck out in the middle of the night and spread PCB's—polychlorinated biphenyls, I think that is—along the roadsides of several counties, including one in my district. People were pretty upset about that. That is one of those things where, as one of the other witnesses said, you can't prove any specific injury right now—maybe it was Congressman Young speaking of dioxins.

But that was cleaned up. There was a civil action brought by the State under the Federal legislation in Federal court for the cleanup costs. But that is all that I would report.

Mr. ROWLAND. Let me ask you, in that respect, if the legislation does become law in North Carolina, what sort of initial experience do you anticipate with reference to the number of claims?

Mr. HACKNEY. I don't think there will be very many. I think that industry is by and large doing a much better job in light of the glare of publicity that exists in this area.

I think we have a problem with cleanups. I think we have made a great deal of improvement in the way hazardous wastes are now being handled. We have a way to go, but certainly great improvement has been made. Those hazardous waste occurrences, as they are labeled in our legislation, which would result in personal injuries, would not be of great numbers.

Mr. ROWLAND. Thank you. Let me ask Ms. Long, then you have had your law in place since May 10 of last year; is that correct? You said the Governor signed it?

Ms. LONG. Mr. Chairman, the effective date was July 1 of this year, 1983. So it is a short history. As I have mentioned, in the area of personal injury and economic loss there has been only one suit. That was a suit already underway. And they are requesting that a count be added under the new law. The judge hasn't ruled on it yet.

Mr. ROWLAND. Mr. Connally?

Mr. CONNALLY. Under the tier 2 or general liability theories that exist everywhere, there are a large number of claims that are filed in California, always characterized by substantial major injuries. I can't give you an exact number—I would guess 100 a year. That is very much arbitrary.

With regard to the tier 1 administrative claim procedure, which became effective January 1, 1982, we have had two claims filed. We are receiving literally 30 inquiries a day. We will get 30 inquiries at the Board of Equalization in the State of California today about how to file claims. But as people learn about the proof standard which exists in the present California law, they are deterred and they don't file claims.

I would agree generally with what Mr. Hackney says. I think on the whole the number of claims, particularly after the tail is taken care of, will be much smaller than we fear. It may be in the area of—in a State the size of California we are talking about 500, 600 claims a year; not a very large number.

Mr. ROWLAND. Thank you.

Mr. CONNALLY. That is a guesstimate, Mr. Rowland.

Mr. ROWLAND. If we do enact legislation on a Federal level, special tort liability, you don't then perceive a large number of frivolous claims being filed?

Mr. CONNALLY. I would say no. Whether you have a strict liability theory or not, there is a threshold cost of about $5,000 to go to a courthouse and file a claim. So if you oppose the strict liability theory, individual attorneys are going to impose an evaluation as to the likelihood of success, character of the damages, before they file that claim. And they will, better than anyone else, sift out the credible from the noncredible with rare exception.

Mr. ROWLAND. Are you saying the attorney will absorb the expense of that filing?

Mr. CONNALLY. That is right. The attorney will, at least in California practice, absorb the cost of prosecuting that lawsuit and will not recover unless he or she is successful with that lawsuit. So that there is an initial threshold evaluation made by the attorney whether or not the claim is successful.

The difference between the negligent theory and strict liability theory is that the strict liability will give the attorney a better chance to be successful because he or she will be better able to prove that causal connection. So there will be a larger number of claims. But it is not a "Katie, bar the door; here they come," because there is still that initial threshold cost which is substantial enough to deter the nonserious person; incidentally, also argues for the continued existence of some type of administrative relief procedure for the smaller claimant.

Mr. ROWLAND. Mr. Hackney?

Mr. HACKNEY. I would certainly agree with that. I think one of the reasons we don't have many claims is that the contingent fee system we have requires that a lawyer bringing an action in North Carolina for negligence arising from some sort of toxic tort has a terrible row to hoe. I mean, the burdens of proof are almost insurmountable at this point. And so the cases simply are not being brought, particularly if they are smaller ones. They are going to remain expensive. But with a strict liability standard the risk would be taken in more cases.

Mr. ROWLAND. That is my point, I guess. If the strict liability standard is included, will this open the door for many more claims?

Mr. HACKNEY. For more claims from smaller claimants, is the way I would answer it.

Mr. ROWLAND. You don't perceive frivolous claims being filed then in this event?

Mr. HACKNEY. I would agree with Mr. Connally. I think frivolous claims are by and large filtered out, at least in our State. I can only speak from our experience. But we have not had a problem with that in any area of the law.

Ms. LONG. Mr. Chairman, I would agree. Minnesota basically has the contingency fee situation. Again, I don't think an attorney is likely to take a frivolous case and invest his time at a small possibility of being recompensed.

Mr. ROWLAND. Let me ask you something else: We heard in testimony previously, with reference to dioxin specifically—and we have touched on this some this morning—that we are not even sure what dioxin does. It is still undetermined.

What is your feeling about, for example, a chemical such as dioxin and how it relates to the claims that would be undertaken by attorneys as far as filing suits is concerned?

Have I made my question clear?

Mr. CONNALLY. I think so. And in the area of rebuttable presumption, for example, if there is not general medical evidence supporting the fact that a medical symptom arises from exposure to dioxin, the claimant would not have the benefit of the rebuttable presumption in that particular instance. That speaks, it seems to me, very powerfully to the point Ms. Long made, that the Federal Government, more than the States, more than the individual claimants, have to accumulate and publish a body of knowledge that shows where this causal connection exists on an individual basis by toxic substance, which then can benefit claimants throughout the country.

I would not—and I don't think any court would find or hold the benefit of a rebuttable presumption in the absence of, at least on a general basis, a causal connection between that substance and medical harm.

I am not sure I was responsive to your question.

Mr. ROWLAND. Yes, you were.

Mr. HACKNEY. I would just say that I practice law, as well, Mr. Rowland, and I wouldn't take one. It is just too much of an undertaking. And there is not that body of scientific evidence, it is my understanding, built up to allow an individual attorney, at least one in a small town, to take a case and be successful with it in an economical way.

Mr. ROWLAND. Mr. Wise?

Mr. WISE. No questions.

Mr. ROWLAND. I want to thank the panel for being here this morning and certainly providing us with a lot of information—it has been so helpful—in a very controversial area.

I think at this time the subcommittee will recess until 1:30, and come back at that time. Thank you.

[Whereupon, at 11:47 a.m., the subcommittee recessed, to reconvene at 1:30 p.m., the same day.]

AFTERNOON SESSION—1:30 P.M.

Mr. LEVITAS. The subcommittee will please come to order.

This is a continuation of today's subcommittee's hearings on the subject of victims' compensation. The witness this afternoon is Mr.

J. Paul McGrath, Assistant Attorney General of the Civil Division of the U.S. Department of Justice, who will discuss with us the position of the Justice Department and the Administration with respect to the matter of victims' compensation, toxic tort and the like.

Mr. McGrath, we welcome you. We are most interested in your testimony today. You may proceed as you so desire.

TESTIMONY OF J. PAUL McGRATH, ASSISTANT ATTORNEY GENERAL, CIVIL DIVISION, U.S. DEPARTMENT OF JUSTICE

Mr. McGRATH. Thank you very much, Mr. Chairman.

Mr. Chairman, I have submitted a written statement. I hope that that can be put in the record.

Mr. LEVITAS. Without objection, your entire statement will be made a part of the record.

[The statement of Mr. McGrath follows:]

STATEMENT OF J. PAUL McGRATH, ASSISTANT ATTORNEY GENERAL, CIVIL DIVISION, DEPARTMENT OF JUSTICE

I appreciate having the opportunity to testify on compensation for people harmed by exposure to toxic substances. The issue raised at this hearing is an extremely important one. For the last several decades we have placed a great strain on our tort system to resolve health-related claims, and now questions about the future of this system are being raised on all sides. Many have asked whether our current litigation and claims processes are working efficiently and cost-effectively. They have also asked whether sensible alternative schemes to litigation can be developed, and if they can be developed, whether they can be financed. And social scientists and other thinkers are asking where all this fits into our economic and social priorities.

We realize that the forces at work raise very difficult questions about whether the current system can survive without substantial change. The following are a few of the current trends:

1. The last decade has brought an explosion of knowledge about the causes or supposed causes of cancer and other serious diseases. Today it is possible to point a scientific, or at least a quasi-scientific, finger of suspicion at various substances for literally millions of cases of disease, although difficult questions of causation remain in the bulk of individual cases.

2. The last decade has brought an explosion of litigation, with more and better-financed attorneys and support personnel pointing toward an even-larger flood of litigation in the future.

3. The courts have greatly expanded the rights of claimants, liberalizing traditional rules concerning fault, knowledge, causation and injury.

These and other factors have created unprecedented numbers of claims against companies in many industries, seeking enormous dollar recoveries. In some cases the potential claims greatly exceed the assets of the defendant companies. In addition, it is no secret that the federal government also is faced with enormous potential liabilities in litigation.

All this raises the question whether our current tort and compensation systems can indeed cope. There is concern that these systems will not adequately compensate significantly injured victims of toxic torts. There is concern that defendant companies in some industries may face bankruptcy in unprecedented numbers. There is concern that the public Treasury may be subjected to enormous liability, perhaps diverting resources from other social programs. There is concern that the combined political pressure from all this may result in premature and inappropriate legislative solutions that we and our children may live to regret.

All this has presented this Administration with a host of difficult and unanswered questions which have led us to establish an interagency coordinating mechanism in an effort to do the best job we can to analyze and develop solutions in the area of toxic and environmental torts. This Toxic Torts Working Group is under the co-chairmanship of Michael Horowitz, Counsel to the Director of OMB, and myself, and it includes representatives from many agencies in the Executive Branch. Compensation issues involve many different agencies within the government: HHS because of its experience with administering income maintenance systems and medical care programs; EPA because of its environmental expertise; Labor because of its knowledge of workers compensation systems and occupational diseases; OMB because of its expertise in fiscal and budgetary matters; Justice because of the need for extensive legal analysis and to assess implications posed for the legal system as a whole; Treasury and the Council of Economic Advisors because of the economic considerations and the Office of Science and Technology Policy because of the broad scientific issues involved. The mandate of this group is very broad but relatively specific. We are not concerned with specific regulatory standards or controls in the management of toxic substances. Rather, our principal goal is to coordinate the government's analysis of proposed legislative means of alleviating toxic and environmental tort problems. Our mandate, put most simply, is to pull together the most complete information possible about the problems, to undertake the best analyses of different possible legislative solutions and to seek the best results for the country as a whole.

To date, our work is still at a preliminary stage. Different subcommittees of our group have been doing studies on various areas of significance, including analysis of legislation already proposed; studies of the environmental and toxic problems involved; a focus on the litigative realities and the ability of our torts system to handle them; an inquiry into what history teaches us, including the troubling lessons of the black lung program; and a compilation of information on what existing compensation programs are accomplishing. A number of people are involved in this work, and it is proceeding quickly. Even though we are still at a relatively early stage, a few observations can be made which suggest the complexity of this problem.

First, one of the things we recognized at the outset is that this is a broad problem which must be considered, analyzed and addressed in a comprehensive manner. In the past, various areas of toxic and environmental torts or injury have been dealt with on a piecemeal basis. There have been proposals to amend the hazardous waste statutes to deal with injuries from toxic waste sites. There have been separate proposals concerning asbestos-related illnesses, radiation-caused cancers, Agent Orange contamination and uranium mining diseases, to name a few. Some of the proposals do not appropriately recognize the difference between workplace and nonworkplace exposure. For the most part, these proposals have been considered in relative isolation from each other. We have come to recognize that this kind of disjointed consideration of what are economically, socially and politically related topics makes no sense.

The fact is each day there is a growing recognition of an expanding range of environmental problems which may have potentially injurious effects on individuals and which result in pressure for legislative solutions. Yet adopting fragmented solutions raises problems of fairness to claimants not covered or those denied other benefits because of the resources devoted to particular compensation schemes, as well as issues of society's priorities and the scientific uncertainty regarding the causation of a particular disease. If we were to adopt a governmental solution for some of these problems but not others, disfavored claimants could properly charge that they had been treated unfairly. On the other hand, when we look at the whole range of problems as an amalgam, we recognize that financially and administratively an overall solution raises very extreme resource allocation issues.

A second general point is a further development of the fairness issue I raised earlier. The current push is for special solutions for those individuals suffering from cancer or other maladies where we have isolated or think we may have isolated a cause of the disease. Those victims of diseases, however, are only a fraction of people similarly afflicted. No doubt a high percentage of others suffer from similar diseases, although we may only discover the cause of their disease in the future or we may never discover it. One might question whether it is fair to have a special compensation scheme for individuals who happen to suffer cancer from an identifiable cause, or one thought to be identifiable, but not for those suffering the identical disease where we have not yet identified the cause. Under the torts system such a result arguably makes equitable sense because the "wrongdoer" will pay. Under many of the schemes proposed as legislative solutions however, there would be no specific or known "wrongdoer" initially footing the bill; rather, society would be funding the relief more broadly through taxation, fees, general revenues or increased product costs. We might also ask whether a government program might not remove any incentive for the party responsible for the exposure to correct conditions.

Third, any answer to these interrelated problems will require us to analyze, question and perhaps make alterations in our fundamental legal institutions and procedures. For example, for many decades the torts system has provided a fairly effective mechanism for compensating victims of environmental hazards, at least in a great number of cases. Recently, however, some have asserted that the torts system is an inefficient and ineffective means of compensating victims or of transferring funds. In support of this assertion, statistics have been compiled which show that a high percentage of the dollars expended in the asbestos cases have been paid not to victims but to lawyers and paralegals and others who are simply part of the compensation system. The statistics referred to may well be accurate, but before reaching too much of a firm conclusion on the basis of them, at least two additional questions should be considered. First, do the high costs of these cases simply reflect the fact that they are at a relatively early stage? It is a well-established fact that such litigation entails large upfront costs for computerizing documents and other background materials and otherwise preparing to defend massive litigation. Second, are the alternatives that have been proposed substantial improvements over the current situation, when all their costs and benefits are considered? This may well be a promising area for alternate schemes for private dispute resoluton. Indeed, I understand some private groups are investigating alternate dispute resolutions for some aspects of these cases.

A fourth point relates to the very difficult issue of causation. The standard of causation will, of course, determine whether the program compensates those who truly deserve compensation. A standard that is too narrow may leave many who deserve compensation uncompensated. A standard that is too broad or too vague will undoubtedly benefit someone, but may not compensate those whom a program is intended to compensate, may benefit many more than is justifiable and may undercompensate a core class of intended beneficiaries. Furthermore, any causation standard that is developed as part of a toxic substances compensation program will significantly affect the standard of causation used in other areas particularly in tort litigation and workers compensation. In addition, we need to ask how we can develop causation standards which do not compromise the integrity of the scientific and medical decisionmaking process—that will not force it into premature and speculative conclusion in a search for false certainty.

There are many other more specific questions that have to be considered in analyzing this issue. For example: what type of compensation should be awarded—only medical costs and lost earnings or should a program offer the full panoply of tort damages, including compensation for pain and suffering? Should a compensation program be structured as a regulatory tool or should it be used primarily for remedial purposes? How should a compensation program be funded? Should a program be administered on the federal or state level? How should changes in the compensation laws be related to other existing remedies? Should any compensation scheme be built on the tort system or should it be an administrative system with nonadverserial proceedings?

New legislation should be measured against remedies now available. The main bills now before Congress relating to compensation for exposure to toxic substances contain a number of possible alternatives to the current system. They vary in coverage, from bills that would cover workers and non-workers to those that would cover only non-workers. They vary greatly in administrative procedures and remedies, standards for liability, funding mechanisms, exclusivity of other remedies, court jurisdictions and review, and standards of causaton. In examining these schemes, we need to answer very specific questions about their relative benefits, their other costs over time, the extent to which they may duplicate presently available remedies, the extent to which they fairly allocate transfer payments in a manner consistent with acceptable societal goals and the extent to which any legislative program can be kept within sensible bounds over the furture.

The history of the last several decades teaches us two troubling lessons: income distribution schemes have proven very difficult to control, and many times the total costs of such programs have been vastly understated at the time of passage. The terrible object lesson in the toxic and environmental hazard area is the black lung program. The billions of dollars it has cost the taxpayers were not even hinted at by its proponents; indeed, if its true costs had been known from the outset, it is difficult to believe that it would ever have been enacted into law. In addition, the black lung program demonstrates the tendency to add beneficiaries to any income distribution program who seem similarly situated to the original beneficiaries, thus multiplying the program's costs. The cold, hard reality is that whatever genius is used to fashion new legislation, we know there is a dire risk that the original sensible scheme will be turned into an economic monster. That risk creates large budgetary and fiscal dangers. Perhaps even more important however, in an era of limited resources, it also threatens to reduce our future flexibility to deal with the other problems of our society.

This concern with constraints on our budget makes the creation of a federal cause of action for toxic torts attractive to some as the means for insuring compensation to victims. We should understand that it is not a "free" compensation system; by establishing a legal mechanism for the transfer of resources from one societal group to another, it operates no differently than a tax or regulatory scheme. Indeed, on the whole it would operate less efficiently than a tax system because of the large transaction costs arising out of litigation. In addition, we should recognize that these cases would further burden an already crowded federal court system. It may satisfy a desire for "fairness", but establishing a private cause of action is not a "free" good.

The Administration is firmly committed to responsible policymaking in this area. Given the extraordinary potential costs and the fundamental changes to our legal system that could be generated by compensation programs, it is imperative that policy not be driven by anecdotal information or vague and unproved assertions of need. The Toxic Tort Working Group is intended to help achieve this goal.

Mr. McGrath. I would simply like to touch on some of the points that seem to me of most significance.

First, obviously I am grateful for the committee's invitation to appear here this afternoon to share with you information about efforts of the executive branch to analyze the problems that this committee has spent so much time and effort in examining itself, and I hope that this is the beginning of a common effort among us to try to deal with some of the very complex but critical problems which this committee has been dealing with that concern, the well-being of so many Americans.

I would like to discuss two main subjects. First, I would like to describe the Cabinet council group that has been established, the toxic torts working group. I would like to discuss the purpose of this group, its structure, and what we are currently doing. Second, I would like to discuss some of the conclusions, albeit in some instances tentative conclusions, that we have reached to date.

First, the toxic torts working group. As the committee is well aware, a variety of forces are at work which raise substantial questions about whether our current institutions can adequately deal with injuries caused by manmade toxic and harmful environmental conditions. Some of these factors include an expansion of scientific knowledge which has given us much more information as to the causation of many diseases, a veritable explosion of litigation in this area; and a broadening of claimants' rights, which at the same time is accompanied by increasing concern that some claimants are not really able to establish their rights. In addition, serious concerns have been raised concerning the increased costs that all this entails, and difficulties it creates for the operation of our judicial and our claims systems.

But, in addition, from the executive branch's point of view, we were also very concerned about the increased potential costs of these problems to the Government. The Government is defendant, or a potential defendant, in thousands of lawsuits alleging billions of dollars of damages, and obviously that is of concern.

Second, a number of the proposed legislative solutions do appear to raise very important fiscal concerns that ought to be looked at, both from that point of view and also from the point of view of our overall priorities. Thus, with that background the toxic torts working group was established as a working group under the Cabinet Council on Legal Affairs, and it was given the job of analyzing possible legislative solutions to the problems that this committee has been discussing and the job of perhaps developing alternative legislative proposals as appropriate.

I am one of the cochairmen of that working group. The other one is Michael Horowitz, who is the counsel to the Director of the Office of Management and Budget. On the working group are representatives of all the relevant agencies, such as Labor, and HHS, and EPA, and a number of other ones.

I want to emphasize that our goal is to coordinate the approach of the executive branch to legislation, or to legislative-type solutions. We are not dealing with or passing on regulatory controls for toxic substances. That is not our mandate. Our mandate is in the legislative policy arena.

We are now really at the first stage. We are trying to pull together relevant information on this area. One of the things, of course, that we have found is not that there is a paucity of information, but there is a deluge of information.

The first thing we are trying to do is to sort out what is helpful from what is not helpful, and to do specific studies in areas where we have found that insufficient data exist. In particular, we are undertaking to analyze the legislative proposals that have been advanced to date. We are doing a study of the tort system and its adequacies or inadequacies in this field. We are undertaking a study of the extent to which victims of toxic or hazardous substances are actually being compensated under current programs. We are doing a study of the Black Lung Program and its experience. We are analyzing the 301(e) study, which of course was the subject of extensive testimony previously before this subcommittee.

We are doing other studies like that in an attempt to pull together, as it were, in an organized fashion, the kinds of background information we feel are necessary to make a contribution in this area. Obviously, our next step is to react to proposed legislative solutions, to develop alternatives, and to work with Congress as best we can in a cooperative fashion to see what we can accomplish in this area.

I would like to mention several preliminary conclusions that we have arrived at in the several months that we have been discussing some of these subjects. One is that we believe you really cannot consider the problems of compensating victims of toxic torts on a piecemeal basis. The general history in this area is for bills relating to different kinds of injury to be considered totally separately, frequently by quite different groups in the executive branch, different groups on the Hill. For instance, there would be a wholly separate set of hearings on asbestos and a wholly separate set of hearings on Superfund and so on.

But it is our view that whether or not there are separate considerations as to these individual problems—and, obviously, there are—nevertheless the area as a whole really needs to be considered as a whole. A reason for this is that there is an expanding recognition scientifically of what problems are caused from certain kinds of toxic substances and hazardous conditions. The kinds of considerations that go into what kinds of compensation schemes might be appropriate are essentially similar. The issues tend to be the same. Overall, when you consider the costs, the administrative concerns about different kinds of programs, the kinds of overall priorities that have to be considered are the same.

A related conclusion is that you come to a matter of fundamental fairness fairly early on in considering these problems. The legislative proposals that we have been dealing with by and large concern victims or people who may be victims of certain kinds of conditions, certain kinds of waste sites, certain kinds of industrial pollutants and the like. They concern victims or people who we believe may be victims where we think we know the cause. The problem, of course, is that those people are only a small fraction of the people who suffer from essentially the same disease, be it cancer or some other disease.

We undoubtedly will learn over the coming years of other groups who are similarly afflicted. But the problem is, we, through many of these compensation schemes, would only be compensating a small fraction of the people who have the same disease, who are suffering from the same ailments, and who perhaps may have indeed been inflicted by causes that are the same or essentially the same as those who would be compensated. Yet, we simply don't have enough scientific information to be sure that that is the case.

Now, singling out certain groups for compensation makes a good deal of sense if you are operating under a tort system in court, or before some kind of compensation board, because then the wrongdoer in the tort sense would compensate the victim. However, to the extent that you set up a compensation scheme where the wrongdoer in the tort sense is not providing the compensation, then what you are really doing is you are singling out certain people for special consideration. And there may be issues of fairness in doing that. Obviously, whether those conditions arise depends on the kind of scheme that is developed.

Third, in looking at our current compensation systems, and particularly our court system, our tort system, there are very difficult issues in analyzing how well the courts are doing. We have all read, with some horror, I think, some of the statistics that have come out of the asbestos litigation, for instance, which have dealt with what percentage of recoveries are going to preparation of litigation, lawyers' fees, and so on, as opposed to victims.

A problem with those statistics is that they may not really reflect the actual situation because those cases are at an early stage, and in that kind of litigation the expense of just getting geared up for the defense tends to be very heavily loaded at the front.

Now, I don't mention this as a critical factual issue. I mention it as the kind of difficult factual issue which we run into when we try to make a judgment as to how well the current system is running. I think we have got to look very carefully at the current system, what kind of a job it is doing, and make a reasoned comparison with the current system and any alternatives that are suggested.

The issue of causation leads me to a fourth general conclusion. I am sure I am not sharing any wisdom with the committee when I say that we have found just from the studies we have done to date that that issue is a very difficult one. The glib statements by some people that there is simply no causation relationship between toxic waste sites and toxic substances goes much too far, obviously. The assertions by others that there are all sorts of causation relationships that we have established really goes too far the other way.

We are advancing in our scientific knowledge all the time. But the issue of causation is very important because to the extent we accept very broad causation, that obviously expands almost geometrically the cost of any program, both in terms of compensation provided and in terms of the management of the program. And it also raises, again, issues of fundamental fairness, because whenever you are going to compensate certain groups of people, a preliminary issue is: Who is in that group? And we, I think, in any kind of a compensation scheme we are talking about here, cannot get away from the fact that the question of causation and the conclusions we reach as to causation will probably be very important in determining who is in whatever group will be compensated.

Finally, the last observation I have is that we are concerned that any legislative solution, however good a job we do in coming up with a legislative solution to any one of these problems, over time, will meet with changes that perhaps can so distort the program physically or in any other way that we may end up with something that we really didn't want at the beginning.

There is obviously no way that we, sitting here in 1983, can protect ourselves for all times. But it does seem to me that we have to be at least cognizant of the fact that whatever scheme we set up—for instance, however we define causation or whatever presumptions we may put into it, can be shifted ever so slightly so as to create a much more expensive or cumbersome scheme.

In my prepared remarks I did refer to the Black Lung Program as an example of a program where that happened. I don't mean really now to say that Black Lung Program was good, bad, or indifferent. It is simply that it was a program that became vastly more expensive than was originally assumed, and which changed over time through slight modifications in the presumptions as to causation which vastly expanded the class coverage.

I would like to say in closing that the hearings this committee has had to date have been very helpful to us. I have read what I believe to be all of the testimony, at least all the testimony I was given, that has been presented before the committee. It has been tremendously helpful to me personally. And I know a number of the others of us who have been working in this group have been reading it, because we are struggling with the same problems. We do very much appreciate the opportunity to participate in the process.

I again repeat, I hope this is only a beginning.

Mr. **Levitas**. Thank you very much, Mr. McGrath.

I think I can certainly agree with you that it is a problem that is so important and so complex that it will require the cooperative effort of everyone involved. I hope our hearings, as they continue, will shed some additional light on that.

I am only going to ask maybe one and one-half questions before I recognize the gentlewoman from Connecticut, and then get back to you later. Let me tell you what my main concern is with the Administration's position. Not that you have failed to identify the problems; I think you have.

What keeps coming back to my mind is: What are you going to do about it? When I say "you," I mean our Government. Right now, it is this administration. In a few years, it may be another administration. This problem is so tremendously important. It affects the lives of so many millions of people today, and in years and indeed generations to come, will continue to affect the lives of people.

My sense is that we cannot simply say—as a Government responsible for the well-being of this Nation—that the problem is so bad and difficult that we can't do anything about it except say how bad and difficult it is. I think we have a responsibility—we in Congress and whoever is in charge of the administration—to say this is a terribly difficult problem. We have got to do something about it, and that is not what I am hearing.

I have a copy of a memorandum dated May 19, 1983, entitled "Compensation for Toxic Substances Exposure," which I understand comes from OMB. Under Roman Numeral V, Superfund Compensation Commission Report, which I take it is the 301(e) Commission study report, the memo says, and I quote, "As part of the 1980 compromise, a commission was established to study compensation. The Commission's recommendations adopted the worst of both worlds, an administrative compensation system with liberal evidentiary standards and generous benefits as well as continued availability of traditional tort law."

The next comment is entitled "Open-Ended Fiscal Threat . . ." "Although its advocates," and I am quoting now, "price Superfund compensation proposals at only a few billion dollars,"—they did put "only" in quotation marks, as you would expect, "The amounts at stake are literally open-ended and potentially enormous. Given the ambiguity of Superfund, compensation could be provided for virtually any environmental exposure and not merely exposures directly related to waste disposal sites. Under the proposed compensation mechanisms, substantial amounts will go into lawyers' fees creating a strong incentive for increased litigation. For example, one-half to two-thirds of asbestos industry payouts go to lawyers' fees and litigation expenses."

Now, that is the end of what they said. Part of that is quite true. However, I think the problem and impact are more significant. If you read just what those few witnesses from Times Beach had to say, I don't think you could help but be impressed that we, sitting here in Washington, don't really appreciate the enormity of what happens to a person when his dreams are abolished and wiped out, along with his health, as well.

What are we going to do about it? How do you respond to the concerns expressed by the Office of Management and Budget? And, where do we go after having heard their fiscal comments?

Mr. McGRATH. I really don't want to respond directly to the conclusions of the 301(e) study today, because we still are in the process, and we are quite a ways along in the process, I might add, at the Justice Department of coming up with our view on that. But let me make this point. The 301(e) study proposals, in effect, would involve setting up a system which might, for instance, do one thing. It might provide for a compensation scheme which would reverse presumptions, and reverse the burden of proof.

The kind of thing that it can do is, for instance, if you have an area of the country, and it is alleged that a given toxic site increased the incidence of a given disease there, for example, some kind of cancer, it may result in a situation where everyone in that area who had that cancer could effectively recover from this compensation system. That could mean, for instance, that let us say the increase of that particular cancer in that area was 10 percent. Yet under this scheme 100 percent of the people who had cancer in that area might recover. Now, to be sure it is a beneficial thing in a sense that all those victims of cancer are compensated. It would be beneficial in many senses if all victims of cancer could be compensated. The problem is that under that kind of a scheme, you end up compensating certain people on the happenstance that they are in an area where you can show some incidence of increased cancer, whereas other people who are similarly situated, who probably got cancer from the same kinds of causes, are not compensated at all.

The additional problem is that to the extent you expand recovery to a vast number of people, the physical consequences can be so large that you have a real priority problem in this sense. Once it gets large enough it puts pressure on other programs, for instance, more pressure on programs that compensate the needy. And then we come up with a direct conflict between the ability to compensate, for instance, those victims of cancer who cannot afford housing, who cannot afford to pay for their education of their children and so on; you come up with a conflict between compensating them on the one hand, and on the other hand compensating people who happen to fit within certain classes.

One of the things that we are seriously considering is to what extent victims of toxic torts are compensated under current systems, for example, disability systems and so on. What are the loopholes in that system? What are the groups of victims who fall between the cracks and end up not only with disabling diseases but with a situation where they cannot cope financially with their problem? And one could argue that those are the people we ought to be primarily concerned about and not some group who are similarly afflicted with a disease but as to many of whom there may be suitable financial wherewithal to do the things that people need to do. Now that is a slightly different way of looking at this problem.

It does, however, turn on your priorities. If we had an unlimited amount of money to spend, that question would not get raised. But we don't.

Mr. LEVITAS. I agree with that. But let me say this. This is the other half of my question—I said one and a half—and then I'll recognize Mrs. Johnson. I have never handled a toxic tort lawsuit. I am not sure they had toxic torts when I was still practicing law. Whatever. The point that you make about overinclusion I suggest exists under the tort system as well. Because it would seem to me at least one of the ways a plaintiff's attorney would go about making the case that there is a causal effect between that hazardous waste site and the plaintiff's injury, physical condition, the cancer, is precisely what you have just described.

He would say all across the Nation the rate of cancer is 3 per 100, or 3 per 1,000. In this community it is 5 or 10, or whatever the increment is. And that, if accepted under the rules of evidence in that particular jurisdiction, might be the basis upon which a jury could conclude that this plaintiff or that class of plaintiffs are therefore all entitled to recovery. So even though that is a very intriguing logical argument, I don't think it argues against whether or not we need a two-tier system of the sort that the 301(e) Commission has proposed on the theory that we might be overincluding, because I suggest to you that that overinclusion, because of that type of epidemiological evidence, could occur even if we had the traditional tort system.

Mr. **McGrath**. It could, Mr. Chairman, but at least it is my view it should not. For example, in the Allen litigation in the district court in Utah, the downwind low-level radiation case, the legal issue you have just posed is probably the most important legal issue before the court at this time. And obviously, the argument we are making in the litigation is essentially the argument that I just advanced in connection with this legislation. And under traditional tort law, and under the tort law as we think it should evolve, I think the argument should come out the same way in the litigation.

I do want to add, what I am doing in the legislative arena is divorced from the litigating arena. It happens that I have responsibilities for the litigation. But if it seems right to come out some different way in the legislative thinking, then I will and we will. I just add that as a footnote. I am not here advancing our litigating interest.

Mr. **Levitas**. I understand that, and I certainly would not want you as an attorney to do anything that would jeopardize your position in some ongoing litigation. I only make that point to suggest to you that if that evidence gets to a jury, and if the jury in this case are the Members of Congress, I think, although I don't want to prejudge anybody's position on that, that is going to be considered too much of a nice lawyer's argument.

It is a good argument, but I think the question is, to the individual who suffered from cancer, why should I have to decide whether I would have gotten cancer any way if in fact I fall within that group that is at risk, because you are never going to find out. If the alternative is to compensate nobody, or to overcompensate, my guess is that whichever jury you are talking to, whether it is this jury here or that jury out there, it is going to say if we err we err on the side of the victim.

Mr. **McGrath**. I think I agree with you 100 percent, that the issue is very, very close to the same issue in either context. The countervailing consideration is that once we overinclude at a vast level, and I am not talking about just a few percentage points, I am talking about the situation that I referred to before, we may exhaust our resources. It is not a free good. In other words, once you do that you place a big financial burden on somebody.

It has been suggested that we place it on the industries in question. Obviously we can do that. But we have to recognize that if we do it in certain ways you will place such a big burden on them that perhaps the industry will not be viable anymore. For example, one of the things that I believe was referred to in that paper you quoted from before, is a proposal involving asbestos where the tax on the industry would have doubled or tripled the price of asbestos, obviously making it a product that simply couldn't be sold.

Now, all I am saying is that you have to look at the consequence. And I am sure you recognize this.

Mr. LEVITAS. I recognize that, Mr. McGrath. Although we sometimes lose sight of it, and I want to repeat it as often as necessary, I am convinced that we live in a society in which the quality of life has been vastly improved because of the utilization of chemicals. However, we now know of certain risks and hazards associated with them, and we are paying a price for that.

I also recognize the fact that we are going to continue to live in a society that produces hazardous waste. Our next agenda is going to be how do we eliminate or minimize the risks from it. But again, while I accept the fact that we have benefited from it, there is a cost and it cannot be loaded in such a way that we throw out the baby with the bath water.

Mrs. Johnson?

Mrs. JOHNSON. Thank you very much, Mr. Chairman.

Thank you, Mr. McGrath, for being with us today. First, just a couple of very specific and narrow questions. You referred to in your testimony to some preliminary conclusions. Could you give us some sense of timetable? For example, your analysis of the current programs and to what extent victims of toxic torts have been compensated under current programs. When do you expect that study to be completed?

Mr. MCGRATH. My hope is that at least the early stages of all these studies, or the initial studies, will be done before the end of this calendar year because our hope is that we really will have together a high percentage of what we want to have finished before the next session of Congress.

Now, it wasn't as if we set this up with that timing in mind. It is rather that the committee got set up this summer, and that is the way the work has been going. But I say preliminary studies because obviously the job of analyzing legislation will go on. When we get preliminary information on the extent to which current schemes are compensating people, presumably there will be more work that will be done.

But I really hope and our plan is that we would have the bulk, at least, of the early work we are doing done before the end of this calendar year. And I hope enough before that so that some of us really have a chance to sit down and think about where it leads us, what other things we ought to look at, and so on.

Mrs. JOHNSON. And hoping to be in a position to propose legislative remedies, if they are appropriate, by the beginning of the next session?

Mr. MCGRATH. By the next year, yes.

Mrs. JOHNSON. A second question. What is the role of EPA in your work?

Mr. MCGRATH. Well, EPA is represented on the working group. The General Counsel of EPA, Jim Barnes, is personally involved in our work. They are involved in helping us pull together some of the facts. We have split the working group's responsibility for each of these studies up so that the agencies who have particular knowledge are helping on particular areas. For instance, I believe EPA is one of the agencies that is assisting in the analysis of the legislative proposals that have been made, because obviously some of these proposals they have been directly involved in, but also they tend to have people who are more knowledgeable than in other areas.

There is no magic to the way we have split up the work. It has been an effort to have the agencies that have the expertise in a given area be involved in a given study.

Mrs. JOHNSON. If there is more than one subcommittee that affects very deeply EPA, does EPA then have somebody on each of the subcommittees?

Mr. MCGRATH. Yes.

Mrs. JOHNSON. To follow up on their work, yes?

Mr. MCGRATH. Yes.

Mrs. JOHNSON. In spite of the limit on their number on your overall council?

Mr. MCGRATH. That would be correct, yes. There are some agencies that are involved in a number of the studies, and other agencies that are involved in perhaps only one. For example, one of the things that we are doing is trying to come up with estimates on the costs of different programs. We have some representatives from the President's Council of Economic Advisers and the Treasury Department in the group. That obviously is the area that those people are involved in. They are not analyzing legislation or studying the Black Lung Program, or something like that. They are, hopefully, people who can help us come up with as good information as we can come up with on the costs of doing different things.

So that would be an example of some agencies that have a relatively narrower role. I don't want to say a minor role, because obviously that is an important consideration, whereas an agency like EPA or HHS or the Justice Department would be involved in several different areas that we are concerned about.

Mrs. JOHNSON. Thank you. Now, earlier in your statement you talked about the fact that causation is very difficult to determine. That it is critical to determining the eligible group, the compensatorily eligible group.

Mr. MCGRATH. Yes.

Mrs. JOHNSON. It seems to me, sitting on the Veterans Committee and on this committee, that indeed causation is very difficult to judge. And at this point, in terms of the amount of scientific information we have available to us, our ability to judge causation does vary dramatically from area to area. I find that conclusion from my experience in contradiction to your apparent assumption that you should be looking at this problem as one single great problem.

You mentioned earlier in your testimony that the issues are always the same. But where you don't have similar scientific data, are the issues the same, or are we even in a position to know if the issues are the same? And yet the availability of that data may make it possible still to deal, at least to some degree, with the compensation issue.

Recently we did, for instance, go part way in dealing with the agent orange issue—that portion of the problem that does seem to be fairly well documented. There were those who wanted to go beyond that.

These issues are very difficult. Causation is difficult. And it seems to me that that reality is in contrast to your approach, that the issues are fundamentally the same, and that they are all aspects of a single problem.

Mr. McGRATH. Well, I would not say that all the issues as to each of the individual substances are the same. For instance, obviously the issue as to what diseases agent orange or other forms of dioxin cause and in what circumstances they may cause them are quite different from the issues relating to low-level radiation or asbestos or benzene or other things. But for instance, there are overall policy issues for the Congress, for instance, which really are quite the some for each one of these. One of them is the issue that the chairman and I were discussing before. And another is the extent to which you require what I will call loosely "good" proof of causation before someone would be put into a class, and what is "good" proof of causation. Because I don't think that it is good policy to treat, let us say, victims of asbestos totally different from victims of benzene or hazardous waste sites.

It seems if we are talking about either using public funds or taxing industry groups, which in economic theory is really not much different, it is not good public policy to single out one group and say, okay, we will deal with agent orange victims or dioxin victims but we will forget about all these other people who for all intents and purposes are similarly situated.

Mrs. JOHNSON. But see that is the question. Are they similarly situated? To what extent? In other words, aren't we capable of actually dealing with all those groups where we do have enough data to be able to define causation? I think you are overdrawing the equity problem.

Mr. McGRATH. The problem is if we do that, let us say to take an extreme, in one of these areas we decide the thing to do is to set up a Federal compenstion scheme. I just mention this as the extreme example. We say all right, this year we have a pretty good lead on what victims of x substance, which people are victims of x substance. Well, if we say we will compensate them and then every other group that comes along where we have good causation proof we will simply expand the scheme to them, the chances are that over the next few years we will end up with an enormous program, for two reasons. One is that we have to assume that over the next few years we will learn more and more about this causation issue. Certainly we all hope we will. Second, by the nature of these programs there is at least a substantial risk that once one is set up the presumptions that will be made, the inclusions of people will be vastly expanded, the same way that they were in the Black Lung Program until just recently.

So that you have to really be very careful about saying, well, we will just deal with this part of the problem, because as a matter of political reality, fairness, whatever you want to call it, we are not likely to leave it there. And we may well create something that would be enormously expensive over time.

And if, indeed, over time we did not expand it to all the people who were similarly situated, you would have a very fundamental fairness issue. And that really, I think, gets to the heart of the concern that I have had. We all recognize that victims of toxic waste sites and harmful substances should be compensated if at all possible. But before we embark down a road where we are going to pick one way of doing that as opposed to another way of doing it, let us think very carefully about what is the goal gain that we are moving toward.

Mrs. JOHNSON. I think I generally agree with your concept. Though I don't agree with the way that you have phrased it earlier in your testimony. I think that as we do know more, we have the ability to create compensation programs that are appropriate and reflective of our knowledge. And there would only be an equity issue if, in the future, people about whom we learn similar amounts were not included.

Mr. McGRATH. But you would not even have an equity issue, for instance, if it were approached from certain other directions. For example, there are ways to foster effective use of private insurance, both to permit the, say the industry in question or the toxic site in question, to actually provide the compensation where they have caused the damage, and also to permit people in industries to have insurance to cover disability.

There are ways of making sure that our existing disability and other programs cover people who may become disabled so that the basic expenses of life can be dealt with. There are various ways to compensate people. And when I said that we all agree that there should be compensation available for the kinds of victims we are talking about, I don't necessarily assume that that scheme need be a Government-funded or run compensation scheme or that it ought to involve the creation of new presumptions or other rules of Federal tort law.

There are other ways to go at the problem, and that it seems to me ought to be a focus. Let us not just assume that we are going to create another entitlement program. Let us look at the whole range of ways that we can deal with this, because this is a problem that is going to be with us for a long, long time. It is going to become an expanding problem. And let us at least try to do the best we can at the beginning, not to create something we will regret.

Mrs. JOHNSON. I would certainly agree that compensation is the issue. And publicly financed compensation is not the only approach to that issue. And I think you recognize that by looking at the current programs and current systems to see how indeed compensation for disability is now being achieved or granted.

That leads me to my last question, and that is, your use of the word entitlement. In my estimation entitlement and compensation are two really very different things. And the word entitlement right now, at least in our society, really implies a much broader eligibility. You are entitled to Social Security simply by virtue of having contributed, whereas compensation implies much more specific—an assumption of injury. I think it is unwise to confuse those two concepts in a program like this.

What we are really talking about here, in my estimation, is compensation, not entitlement.

Mr. McGRATH. I agree with that. An entitlement program is one sort of a compensation program, but only one sort. In other words, an entitlement program could be set up here. Congress could pass an authorization bill which authorized, and indeed required, the payment of money to any individual who could establish a certain disability that he or she lived in a certain area, whatever. That would be an entitlement program. I am not suggesting that one should be created. Indeed I am suggesting we ought to think long and hard before we create one.

Compensation, however, covers a whole range of things from there all the way over to programs which encourage people to have private disability insurance, or corporate group disability insurance.

Mrs. Johnson. Thank you.

Mr. Chairman, that concludes my questions at this point.

Mr. Levitas. Thank you, Mrs. Johnson.

I thought the last point that Congresswoman Johnson made is extremely important, and I am glad it came out the way it did. In response to her observation, you said that you do understand and appreciate the difference between compensation and entitlement. My question to you is, based on your dealings with OMB, does OMB understand the difference?

Mr. McGrath. I believe so. Certainly in the sense that I have described the difference and in the sense that I believe this committee is considering the two. Just from having read as much as I could of the proceedings here, I have understood you to be really considering the whole range of options and issues and so on. So I assume the answer to your question is yes.

Mr. Levitas. Well——

Mr. McGrath. At least speaking for the people we are really focusing on.

Mr. Levitas. The reason I say that is because we are considering the whole range. We are not prejudging or eliminating in advance any option that is available to us. But there is clearly a difference between our compensatory system, like workmen's compensation, for example, on the one hand and entitlement systems, disability under Social Security, on the other. If you put those together you are going to make some wrong judgments about where you are going to come out.

The reason I mention that is as I read this infamous OMB memo of May of this year I don't come away with a great deal of comfort that somebody is really looking for a solution within the context of the leadership which the administration should be providing. I get a very negative feeling about it. I get the idea that they think there are a lot of unrepresentative horror stories upon which Congress is about to respond, and create some unaffordable system without the positive feeling that I would like to come away with and say, yes, we have got a problem.

We have got to find a solution. We have got to look at a lot of different alternatives, but the bottom line is we have got to find a solution to that problem and not simply to say the people who suffer: "suffer."

Mr. McGrath. All I can say is that I do—my approach is the latter and a lot of what you see and what you have read is in part our effort to get the executive branch thinking about this whole thing. As you have seen from that document and some other ones that have been published, the main goal of this working group was to try to get the different parts of the executive branch that have knowledge, that have ability, working together on this thing. And the only way you get that done is to really do something that gets everybody's attention. And about the only thing that really gets that attention sufficiently is to talk about costs and talk about overall fiscal concerns.

I hope we can go from there to be constructive, to work constructively. I believe we can. I don't really have any doubt that we can.

Mr. LEVITAS. Let me ask you this: is the Federal Government a defendant in any pending hazardous waste litigation now?

Mr. McGRATH. I don't know the answer to that, Mr. Chairman.

Mr. LEVITAS. Would you find out and supply it for the record?

Mr. McGRATH. I will supply that for the record.

[The information received from Mr. McGrath follows:]

U.S. Department of Justice
Land and Natural Resources Division

Office of the Assistant Attorney General
Washington, D.C. 20

DOC # ______
ADDE ______ REC # ______
DATE ______
PARA ______

December 16, 1983 DEC -7 1983

COMMENTS ______

Honorable Elliott H. Levitas
Chairman
Subcommittee on Investigations & Oversight
Committee on Public Works & Transportation
Rm. B-376, Rayburn HOB, S.W.
Washington, D.C.

Re: Request for Information Concerning Hazardous Waste Litigation, Hearings of November 8, 1983

Dear Mr. Chairman:

It is my understanding that, during the testimony of Mr. J. Paul McGrath, Assistant Attorney General for the Civil Division, before the Subcommittee on November 8, 1983, a request was made that the Subcommittee be provided with information concerning the number of pending actions in which an agency of the United States is allegedly liable for cleanup costs or damages relating to a hazardous waste site or facility.

To assist you in your deliberations, I have informally requested an inventory of actions under the Resource Conservation and Recovery Act (RCRA) and the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) in which federal agencies have been alleged to be liable in some way for hazardous wastes. A list of those actions and a short description of the case, its status, and the nature of the allegations against the United States is attached as Appendix I. Most of these suits were originally initiated by the United States and involve counterclaims commonly alleging relatively small contribution to the problem by a federal agency. The list does not include suits under the Clean Water Act or other environmental statutes which may involve hazardous wastes but which are not "hazardous waste suits" as that phrase is commonly used.

We are particularly pleased with the progress being made to resolve City and County of Denver v. Ruckelshaus, et al., D. Colo., Civ. 83-1043, where a consent order has been negotiated and submitted to the governing body of Denver for its approval, by which Denver would initiate a response at the subject site,

Shirley-File pls.
Jack

while reserving any rights it may have to seek participation by or compensation from others. A settlement has not been fully negotiated, but progress to date reflects our ability to deal with special circumstances presented by hazardous sites which may involve federal agencies. It continues to be this Administration's firm policy that federal facilities have a strong obligation to set an example in meeting the standards which both RCRA and CERCLA impose upon private facilities and the Administration will continue to make every effort to see that federal facilities set such an example. This Department and EPA have been quite successful in receiving contributions from federal agencies toward the cleanup of several hazardous waste sites where multiple generators were involved.

Thus far, the United States has not taken a formal, written position regarding its legal status as a potential defendant in hazardous waste suits.

Please let me know if you desire other information concerning the listed cases or other cases. Within the limitations imposed by the conduct of litigation, I will be pleased to assist you in any way possible.

Sincerely,

F. Henry Habicht, II
Assistant Attorney General
Land and Natural Resources Division

cc: J. Paul McGrath
Assistant Attorney General
Civil Division

Attachments

APPENDIX I */

City and County of Denver v. Ruckelshaus, et al., D. Colo., Civ. 83-1043:

This action was filed June 10, 1983 against numerous federal and state agencies and officials and private corporations, and involves the "Lowry landfill," which is owned and operated by Denver. The action is in response to a CERCLA Section 106 administrative order issued against Denver on June 8, 1983, ordering it to take certain response actions at the site. The suit seeks declaratory and injunctive relief to prevent enforcement of the order, asserting that the United States and others are also legally responsible for conditions and should be compelled to share in costs associated with compliance. The United States has moved to dismiss the complaint and is conducting negotiations with Denver and others, with the objective of settlement. A consent order has been negotiated and presented to the Denver governing body for approval, by which Denver would begin a response, while reserving any rights it may have to seek the participation of or compensation from others. The action is pending.

Florida v. Silvex, et al., M.D. Fla. Civ. 83-926-CIV-J-14:

An action was filed against the United States in state court, under state hazardous waste laws similar to CERCLA. The action has been removed to federal court on a motion by the United States. The United States has moved for dismissal of the suit on grounds of sovereign immunity. The Navy is alleged to have sent wastes to the site which were released and have damaged natural resources. Florida is seeking natural resource damages and a response. The action is pending.

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*/ Several actions which may involve allegations against the United States were filed around the nation between December 9th and 12th, 1983, due to a CERCLA statute of limitation. To the best of our knowledge this list is accurate as of close of business December 12, 1983.

United States v. Chem-Dyne Corp., et al., S.D. Ohio, Civ. C-1-82-840

This action was initiated by the United States under various provisions of RCRA and CERCLA to obtain abatement of an endangerment to health and the environment and for other relief. Some defendants have counterclaimed against the United States, alleging that it used the site and encouraged others to use the site. The United States has admitted use of the site, but declined to respond to allegations of liability, which are legal and not factual in nature. The action is pending.

United States v. Columbia Steel and Metal, et al., D.S.C., Civ. 83-1487-0:

This action was initiated by the United States under various provisions of RCRA and CERCLA to obtain abatement of an endangerment to health and the environment and for other relief. Some defendants have counterclaimed, alleging that the United States generated wastes which were disposed of at the site. The United States has acknowledged that 12 of 550 drums of waste located at the site are marked "EPA", but has declined to respond to allegations of liability, which are legal and not factual in nature. The action is pending.

United States v. South Carolina Recycling and Disposal, Inc., et al., D. S.C., Civ. 80-1274-6

This action was initiated by the United States under various provisions of RCRA, and amended to include allegations under CERCLA, to obtain abatement of an endangerment to health and the environment and for other relief. Some defendants have counterclaimed, alleging that the United States generated some wastes which reached the site and exercised regulatory control over the site. The United States had admitted that some of its wastes went to the site, but has declined to respond to allegations of liability, which are legal and not factual in nature.

United States v. J. B. Stringfellow, Jr., et al., C.D. Cal., Civ. 83-2501(MML)(Mcx):

This action was initiated by the United States under various provisions of RCRA and CERCLA to obtain abatement of an endangerment to health and the environment and for other relief. Three defendants have counterclaimed against the United States, alleging its responsibility as a generator of certain wastes. The action is pending.

United States v. Wade, et al., E.D. Pa., Civ. 79-1426:

This action was initiated by the United States under various provisions of RCRA, and amended to include allegations under CERCLA, to obtain abatement of an endangerment to health and the environment and for other relief. Some defendants counterclaimed, alleging that wastes of the United States were disposed of at the site. The United States has answered, denying that any of its wastes went to the site. The action is pending.

State of New York v. U.S. et al., E.D.N.Y., Civ. No. 82-2228:

This action was filed in the summer of 1982 against the United States. It involves groundwater contamination by jet fuel in Suffolk County, Long Island at Westhampton Air Force Base. In its initial complaint, the State of New York sought injunctive relief and damages on a variety of theories under the Clean Water Act, Federal Tort Claims Act and common law doctrines. We have learned that they are now moving to amend their complaint to include a CERCLA Section 107 claim for natural resources damages for an amount which is presently unknown but probably will be $50 million, estimated response cost. The action is pending.

U.S. v. Shell Oil Company, D. Colo., Civ. No. :

This action was initiated by the United States for recovery of response costs and damages to natural resources at the Rocky Mountain Arsenal, Denver, Colorado under Sections 104 and 107 of CERCLA and for damages and injunctive relief for breach of contract, restitution, and tort, and for a declaratory judgment on December 9, 1983. The State of Colorado served the Department of the Army with a claim under Section 107 and 112 of CERCLA for natural resources damages at the same site on December 2, 1983 and filed a complaint in the Colorado District Court on December 9, 1983. The action is pending.

New York v. U.S. (Solvent Savers), N.D.N.Y., Civ. No. :

This case was filed on December 9, 1983. The State of New York is suing the Air Force as a generator under CERCLA, for response costs and natural resource damage claims arising out of activities at Griffiss Air Force Base in Syracuse. The action is pending.

Cadillac Fairview/California v. Shell Oil Co., Ruckelshaus et al., C.D. Calif., No. :

This action was filed on December 9, 1983 against Shell Oil Company, Dow and the United States. Cadillac Fairview is seeking a declaration that GSA, as successor in interest to two defunct federal agencies, is liable under Section 107 of CERCLA for response costs for removal of hazardous wastes, and an injunction requiring EPA to undertake further remedial action at the site. The action is pending.

United States v. Saul Levy, et al., W.D. Wash., Civ. No. :

The United States initiated this suit seeking hazardous waste cleanup and other relief. The defendant, site operator has counterclaimed alleging that the United States sold surplus substances to him. The action is pending.

Mr. LEVITAS. I see, obviously, the potential of that occurring. Let's take a certain DOD site in a southwestern State which has been recently found, and contrary to earlier hopes that it was not penetrating through an impenetrable soil, we now find out that it is. In addition, lets assume that right underneath that particular waste site is an aquifer serving a very large urban center.

Now, if that aquifer gets contaminated—if it is not already—and if individuals who have come in contact with that site or water begin to suffer some damages, obviously every lawyer in the neighborhood is going to start suing everybody they can find, including the United States.

I am concerned that, if we don't find some solution, since DOD is probably the largest single new provider in this country, we may end up paying a great deal more as taxpayers, unless we devise a system that properly compensates, and, if appropriate, places limits on the amount of recovery.

Mr. MCGRATH. Well, that is a concern that I share with you completely. And that is one of the reasons why I am the co-chairman of this working group. I will be happy to supply that information.

Mr. LEVITAS. I would appreciate that. Also you might, when you do that, if you say this is normally handled by the Lands Division, you might ascertain what sort of memoranda exists, if any, with respect to the Government's liability in matters of the sort I just identified.

Mr. MCGRATH. Suits against the Government for toxic waste sites?

Mr. **Levitas**. That is right. I have no further questions at this time.

Mrs. Johnson?

Mrs. **Johnson**. Thank you very much, Mr. Chairman.

I have a couple of other additional questions that occurred to me. First of all, you have referred in your testimony, to the Black Lung Program as an example of what we ought not to do. And I think in view of our conversation about compensation versus entitlement, could you spell out a little more clearly so we have it on the record, and more extensively than you did in your written testimony what it is in the Black Lung Program that you think we should avoid?

Mr. **McGrath**. Two things. One is, at least as I understand it, that the estimates that people made as to the cost of that program at the outset turned out, for whatever reason, to be grossly inadequate. The program simply cost a lot more than it was estimated. My caution there is that whatever program is put together let's make sure we know what the costs are.

I don't mean just the direct costs out of the Treasury; all the costs. Second, over time, by changing the presumptions, the inclusion of people in the program was vastly increased to a level where it has involved several billion dollars a year of costs.

Now, as I said earlier on, I am not involved day to day on the Black Lung Program. But I simply use it as an example of a program that, once set up, was expanded a good deal simply by fiddling a little bit with the presumptions. And what I am saying is that any solution that we come up with, we should look at it to see what little changes in presumptions will create what additional costs, what additional coverage and so on.

Mrs. **Johnson**. I agree with you that certainly a rational process of legislating would require that we have as good an estimate as possible of costs and that we understand what changes could be made in the program and what the effects would be, both fiscally and in human terms. But the implications of what you say tend to be that if it is going to cost a lot, we ought not to do it. And that expansion of a program is necessarily unwarranted.

I think we can expect that, whatever program we start with now, if we do it properly—and in my estimation that would be pretty narrowly drawn because that is where the information is now—that we would expect that the program will be expanded as we know more in a greater variety of areas.

Consequently, that while it is useful and important to be aware that cost estimates that we make now are only relative to the narrow definition of the program that we are drawing now, it is unrealistic to think that either we shouldn't act because we don't know what the future holds, or that we can act, and not expose ourselves to the need in the future to expand the program to achieve equity.

Mr. **McGrath**. Well, my concern is really this. Certain of the kinds of programs that have been proposed would create more risks of the type of problem found in the Black Lung Program than other ones, because, for instance, once you start saying here is a whole group of people we are going to compensate, it is very easy to, that, as time goes on, throw people who look pretty similarly situated, into the hopper. History shows that this frequently happens.

Now that means we could well start out with a program that might cost a billion or two billion a year early on, and be tens and tens of billions as the natural momentum got going. And our thought is that we really ought to think long and hard about whether we ought to do that, because what we would be creating would be something that 5 or 10 or 15 years from now might be an enormous program. And that, of course, among other things, would restrict other things we might want to do with the public treasury at that point.

And that is something we just, I believe, should not ignore. And I know that the committee isn't ignoring it. I am not suggesting that because I know what you are looking at. But it is a concern that I underline for that reason.

Mrs. JOHNSON. I appreciate your concern. It simply doesn't relieve us from acting. And so we are still stuck with the problem of definition.

Mr. McGRATH. That is right.

Mrs. JOHNSON. And eligibility at this point. I have only one other brief question.

Mr. LEVITAS. You may proceed.

Mrs. JOHNSON. More as a matter of calling it to your attention. Your working group will be making recommendations. Considering the diversity of your working group, aren't you concerned that the recommendations by the time they achieve consensus would be so general as to be not terribly useful? And if there is a difference of opinion, will agencies like EPA carry more weight than some of the other agencies in making those final determinations?

Mr. McGRATH. Well, I can only make a prediction based on the way we are organizationally set up now. Certainly, overall, the people who are involved in either are or have direct access to the highest level decisionmakers. I don't think if there is a right way to do something and those people are convinced that is the way it has to be done, those people——

Mrs. JOHNSON. It isn't an answerable question, but I think it is something we ought to be aware of because I think it may not be so easy for just the reasons we were talking about earlier.

Mr. LEVITAS. I think my colleague is making a gentle suggestion.

Mr. McGRATH. Yes; I did get the drift of the suggestion.

Mr. LEVITAS. Let me thank you, Mr. McGrath, for your attendance today and your contribution. I rather suspect we will be talking about this with each other in the weeks and months ahead as all of us address this terribly important problem that affects millions of people today, and millions in the future.

Tomorrow morning, the subcommittee will convene at 10 a.m. in room 2253 of the Rayburn Building, at which time we will hear from officials from the Centers for Disease Control, the National Institute for Occupational Safety and Health, and from others in the research community as well as the chemical industry. I expect that the hearings tomorrow will provide us with additional information on this important subject.

Accordingly, the subcommittee will stand in recess until 10 a.m. tomorrow morning.

[Whereupon, at 2:40 p.m., the subcommittee was recessed, to reconvene at 10 a.m., Wednesday, Nov. 9, 1983.]

HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(Compensation of Victims Exposed to Hazardous Wastes)

WEDNESDAY, NOVEMBER 9, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 10 a.m., in room 2253, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Mr. Levitas. The subcommittee will please come to order.

This morning the Subcommittee on Investigations and Oversight of the House Committee on Public Works and Transportation is continuing its investigation now into its second year on the entire subject of contamination of the Nation's water resources and the impact it has on public health and on the economy of this country.

The hearings that we are in the midst of are focusing on one important aspect of that question, which is the matter of compensation for victims who have been exposed to toxic chemicals at abandoned hazardous waste sites. This will be the 5th day of hearings on this particular aspect of the subject, and while there is still some question in the minds of some of the witnesses we have heard from concerning the linkage between hazardous waste sites and health, there are others who feel either directly, or by coincidence, that they have been physically affected by this problem.

The present Federal laws on the subject omit compensation mechanisms for victims. We are in a situation where our laws seemingly provide for compensation for damaged property but not to individuals. We pay compensation, if you will, for the trees, but not for the people inside the houses. And what we are focusing on today and in the other hearings that we have held are both the scientific, as well as the procedural basis for dealing with the question of compensation for victims.

Do we have enough scientific evidence to legislate? Are there other questions which must be answered? What are the best approaches to take? Are the solutions Federal or State? Should we simply leave the law as it is? Should we make modifications in the tort law by way of presumptions, or should we create a new form of administrative compensation, similar to workers' compensation, for people exposed to hazardous waste? These are some of the extremely complex and difficult questions to which we are seeking answers. Today, we are going to hear from two important panels that I am satisfied will contribute greatly to our understanding of this subject and will enable us better to make those legislative decisions that will be made in the relatively near future as we approach the subjects.

The first panel will consist of Dr. Vernon Houk, who is Director of the Center for Environmental Health, Centers for Disease Control, in Atlanta. I understand that Dr. Donald Millar will not be here today due to illness in his family. I regret to have that sad news, but I certainly understand.

However, I understand that Dr. Fingerhut will be here. She is also with the National Institute for Occupational Safety and Health, and Dr. Sheila Jasanoff, who is with the Program on Science, Technology and Society, Cornell University, and a consultant for the National Science Foundation.

If the members of this panel will please come to the witness table. For those of you who are not familiar with the environment of this particular place, that clock behind you and the lights on it from time to time make noise, and different lights will go on, indicating occurrences on the floor of the House of Representatives. Two lights, which are now on display, indicate that a vote is in process. So I think what I will do, as we are also waiting for Congresswoman Johnson to join us, is go over at this time, vote, come right back and get directly into your testimony. Then we will make an announcement later in the morning as to whether we will go straight through or take a break around noon and then come back about 1:30. We will have to see what the circumstances are on the floor.

Accordingly, the subcommittee will stand in recess for 10 minutes.

[Recess.]

Mr. LEVITAS. The subcommittee will please come to order.

I have already introduced the first panel we will hear from today. Does any member of the subcommittee have an opening statement to make? If so, please feel free to do so.

Mrs. JOHNSON. Thank you, Mr. Chairman. Very briefly at this time, I do want to acknowledge and credit the participation of Arco and Union Carbide representatives here today. I understand and can appreciate to a certain extent that many of the so-called generator industries are reluctant to publicly address the victim compensation issue. Even though their appearances may not guarantee that the subcommittee gets answers to all of its questions, I want the witnesses to know that we do appreciate their contributions.

And I just want to make as a side comment the fact that the Union Carbide plant in my district has the statewide record for the longest number of days, consecutive days, when there has been no lost time due to an accident. So your record in terms of safety and employee concerns is really a very fine one in my State, and I hope nationally as well.

The other point I would like to make is that the 3-M Co. has also expressed an interest in helping the subcommittee examine this issue. Although 3-M representatives couldn't be here today I hope we will provide them with the opportunity to submit their views for the record.

Mr. LEVITAS. Thank you, Mrs. Johnson.

Does any other member desire to be heard at this time? If not, Dr. Houk, why don't we begin with you? If you would for the record identify yourself, your title and give us a brief description of what it is you do; then you may proceed.

TESTIMONY OF VERNON N. HOUK, M.D., DIRECTOR, CENTER FOR ENVIRONMENTAL HEALTH, CENTERS FOR DISEASE CONTROL, ATLANTA, GA; MARILYN A. FINGERHUT, PH.D., EPIDEMIOLOGIST, NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, CINCINNATI, OH; SHEILA S. JASANOFF, PROGRAM ON SCIENCE, TECHNOLOGY AND SOCIETY, CORNELL UNIVERSITY, CONSULTANT, NATIONAL SCIENCE FOUNDATION, ITHACA, NY

Dr. HOUK. Thank you, Mr. Chairman. I am Dr. Vernon N. Houk, Director, Center for Environmental Health, Centers for Disease Control and Assistant Administrator of the Agency for Toxic Substances and Disease Registries of the Public Health Service [PHS], Department of Health and Human Services. I am pleased to be here this morning to discuss with you some of the issues that relate to our activities under Superfund.

You asked for testimony on the issue of compensation for persons exposed to toxic chemical waste. Because I have no official responsibilities related to compensation policy, my testimony will be limited to those questions contained in your letter which address my duties in managing specific studies of the human health effects of exposure to hazardous substances. I direct a major effort within the Public Health Service to study the human health effects of community exposures to hazardous substances. From these studies, scientists on my staff and I frequently formulate recommendations to limit or prevent future and further exposure. I do not, however, recommend or set policy related to the compensation of persons inadvertently exposed and/or injured.

As you know, a Cabinet-level work group has been formed to address issues related to toxic compensation. The work of this group may be useful to you and your colleagues in this matter, and I understand you had testimony relating to this from Justice yesterday.

I would like to illustrate our Superfund activities under section 104i(1) related to the establishment and maintenance of registries, and section 104i(5) related to determining the relationships between exposure to toxic substances and illness. These two sections of the Superfund law present us with some of the most complex and perplexing scientific problems faced by public health officials today.

At Times Beach, MO and other dioxin sites, we have been attempting to measure the human health effects, if any, of exposure to 2,3,7,8 tetrachlorodibenzo-p-dioxin, the most toxic form of dioxin. Although the dioxin contamination incident in Missouri is not necessarily typical of the overall problem addressed by Superfund, it is useful for me to illustrate our work by describing both the immediate and the long-term program we have implemented in cooperation with the State of Missouri in response to the dioxin contamination.

In the early 1970's waste oil which had been mixed with dioxin-contaminated byproducts from the manufacture of various chemicals was spread widely in the town of Times Beach and in many other areas of Missouri to control dust. Just before Christmas last year, the Environmental Protection Agency informed us of the positive results of their dioxin testing program in Times Beach. EPA asked us to adivse them, the Federal Emergency Management Agency, and State officials regarding the potential human health effects which could result from contact with dioxin-contaminated soil.

Unfortunately, there are no easy answers to this question. At the time, and even today, very little is known about the human health effects of exposure to very low levels of dioxin. Almost all of what is known has been learned from animal studies, that is by exposing various species of animals to various levels of dioxin and observing the outcomes, then extrapolating from that information to arrive at assumptions about kinds of health effects which might be expected in humans. There have also been studies of workers who were inadvertently exposed to higher levels of dioxin while on the job, but the method of exposure in an occupational setting is quite different from the situation in Times Beach.

Although no adequate human health studies have been done, we knew based on the studies described above that dioxin could be potentially very highly toxic.

Between the time the soil samples had been collected by EPA and the analysis had been carried out, the town of Times Beach was flooded. When we received the results of the testing program the citizens of Times Beach had temporarily moved away from their flooded homes. Because of our deep concern about the potential for harmful health effects from dioxin, and the many uncertainties about this particular exposure incident and our fear that the flooding may have moved contaminated soil into the houses, we recommended that the citizens of Times Beach not return to their homes. Using our best estimate of exposure and health effects, we assessed the risk of dioxin exposure under the conditions present at Times Beach to be excessive if continued exposure were permitted to levels of dioxin above 1 part per billion.

We recognized then, and we recognize now, that this estimate of risk was based on a number of assumptions that required testing. We convened a panel of scientists from within and outside Government, from academia, industry, and environmental groups to review the process, assumptions, and calculations we used to arrive at the 1 ppb value. This group gave us valuable suggestions for improving the process, but did not take issue with our conclusion that long-term exposure to dioxin in soil at levels above 1 ppb in a residential setting should be avoided.

The State of Missouri, the St. Louis University School of Medicine, and St. Joseph Hospital participated in a pilot study. The pilot study consisted of a questionnaire survey of current and past health status, a physical examination for clinical signs and symptoms of potential health effects from dioxin, and a battery of specialized laboratory tests. On October 16, 1983, we reported the findings of our human health effects study. I would like to submit the Executive Summary of our report to you for the record.

[The following was received from Dr. Houk:]

MISSOURI DIOXIN HEALTH STUDIES PROGRESS REPORT, OCTOBER 16, 1983

EXECUTIVE SUMMARY

This interim progress report consists of 2 parts: (a) An overview of the public health response activities to the 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) environmental contaminations in Missouri, prepared by the Center for Environmental Health, Centers for Disease Control (CEH/CDC) and the Missouri Division of Health (MDH); and (b) a report on the pilot epidemiologic study of health effects due to TCDD contaminations in Missouri. The report of the pilot study was prepared by the above two agencies and the participating co-workers from the Departments of Medicine at St. Louis University School of Medicine, St. Louis, and St. Joseph Hospital, Kirkwood, Missouri.

In 1971, waste oil containing TCDD was sprayed for dust control on residential, recreational, and work areas in Missouri. For many of these areas, the presence, magnitude, and extent of environmental contamination was not documented until late 1982 and new information is being developed daily. The first document in this progress includes an overview of the environmental TCDD contamination in Missouri, a summary of the risk assessment process used to recommend what concentration of TCDD in soil in residential areas should raise a level of concern for human health, and an outline of the initial activities undertaken by MDH and CEH/CDC. The public health Agency (EPA) and the Missouri Department of Natural Resources (MDNR) on review and evaluation of environmental data, the development of health advisories to be used by EPA in considering remedial or preventive actions at specific contaminated sites, a helath education effort for the medical community and general public, a dermatologic screening clinic, establishment of a central roster of potentially exposed individuals, administration of a questionnaire survey, and a pilot medical-epidemiologic study of a group of individuals 1983.

The purpose of the pilot medical-epidemiologic (i.g., symptoms on medical history questionnaire, findings on physical examination, and results of laboratory tests) present a group of individuals with high risk of environmental TCDD exposure. The prelinary information on possible health effects from these exposures provided by the pilot study would enable medical epidemiologists to develop more refined and specific epidemiologic protocols to be used in further evaluations. Pilot studies by their nature are an initial probe to obtain an early impression of the types of problems being seen and thereby bring a problem into better focus and help future activities. Pilot studies are not by their nature meant to resolve all, or even the majority of, questions pertaining to a specific problem.

In this instance, the pilot medical-epidemiologic study was undertaken because many uncertainties were initially apparent in the Missouri dioxin situation. These uncertainties mitigated against a quick, simple design of a definitive health study. These uncertainties included: (a) very limited data on the types of health effects to be expected, with many potential effects to be evaluated, but very few known effects which are unique to dioxin exposure; (b) the limited, but rapidly emerging, environmental data at the time of status for large study groups was precluded; (c) the unavailability of a direct measure of exposure (such as a TCDD blood levels) for comparison with health findings in a study; and (d) the distribution of most highly exposed individuals among a number of different sites and settings.

Within the constraints and limits noted above, and with a resulting sense of caution, the following summary of the pilot survey is provided. With regard to the previously reported effects must closely associated with dioxin exposure, no cases of chloracne or porphyria cutanea tarda (or precursor stages of the latter) were documented. With regard to the large number of clinical tests for the organ systems suggested to be most sensitive to TCDD exposure (e.g., the liver), as well as the large number of tests of other systems and functions in the study, the results are largely negative, i.e., the majority of test results did not show an association with TCDD exposure status. The overall impression of negative results is based on three lines of reasoning: (a) There were very few statistically significant differences in these clinical tests between the high and low risk groups, (b) the group as a whole showed few clinical abnormalities and there was not a pattern of findings suggesting consistent trends of increased abnormalities in the high risk group, and (c) for specific areas (e.g., tests of liver function) multiple tests were consistent in not demonstrating abnormalities.

While these overall, first observations are encouraging and are not indicative of significant, dioxin-related effects in this pilot study population, several points should be kept in mind: (a) Inconclusive results should be evaluated in further studies; (b) Subtle health effects could possibly have been missed; (c) certain areas, such as psychological or behavioral function, were not evaluated in this pilot study; and (d) some degree of exposure misclassification of individuals could have occurred.

Consistent with our original purpose, however, MDH and CEH/CDC are developing protocols for further evaluation of groups thought to be exposed to TCDD in an attempt to provide more definitive conclusions. Efforts are also underway to develop the laboratory techniques to provide a direct measure of TCDD exposure (e.g., serum TCDD analyses) which would be helpful in providing accurate classification of exposure status.

It would be unwise to extrapolate from these preliminary findings to other possibly TCDD exposed groups in or outside of Missouri. Such extrapolation may be possible after more definitive studies of both health effects and dioxin exposure status are available.

Finally, public health policy in situations such as this environmental contamination with TCDD must continue to be focused on the prevention of any potential health effects even if such effects were not demonstrated in a small pilot study. Health effects could occur with a latency period exceeding the time from initial exposure to examination in this study. For this reason, all appropriate efforts need to be made to prevent human exposure.

Dr. HOUK. In summary, we could not definitively identify any significant health problems associated with exposure to the levels of dioxin present for this study group. While these overall first observations are encouraging and do not indicate significant immediate dioxin-related effects in this pilot study population, several points must be kept in mind: Inconclusive results on a number of tests will be evaluated in further studies, subtle health effects could possibly have been missed in this pilot phase, certain areas such as psychological or behavioral function were not evaluated in this pilot study, and some degree of exposure misclassification of individuals in the study may have occurred.

It would be unwise to extrapolate from these preliminary findings of our pilot study in Missouri to other dioxin-exposure incidents; either in the occupational setting, in other community settings, or among veterans of the Vietnam war. We intend to follow up with additional studies to delineate better the health effects in persons accidentally exposed to this potentially toxic compound.

Thus far, I have concentrated on our efforts to evaluate the immediate acute or subclinical effects of dioxin in people. You also asked about the efforts to identify the chronic, long-term effects which may result from exposure to low levels of toxic substances in the environment. We are attempting to answer the question: What long-term effects, if any, are there to persons exposed to dioxin and other hazardous substances? One of the most commonly recommended tools for studying the chronic or long-term effects of exposure is to construct a registry of persons exposed. The persons in the registry can then be tested periodically to determine their health status and eventually to determine the cause of death.

The use of registries to evaluate the long-term human health effects of toxic chemical exposures began prior to the passage of Superfund. In 1975, for example, we, in cooperation with the State of Michigan, set up a registry of 4,000 persons exposed to polybrominated biphenyls after the occurrence of an accidental mixture of that chemical into feed grains. As you will hear later, CDC's National Institute for Occupational Safety and Health has been studying the health effects of chemical exposures among workers through the establishment of registries.

Registries cannot be used to give quick conclusive answers to the many questions that people ask about the effects of toxic chemical exposure on their health or the health of their children. Even the most active and aggressively pursued studies using registries require decades to produce meaningful results. It is also true that even when the results of long-term studies are made available, the findings are likely to be hotly debated by scientists who honestly represent different points of view. I point out these inherent limitations in registries to call to your attention the fact that expectations for registries being used as tools to set public policy in the near future may be unrealistically high.

Even with these limitations, the Public Health Service has a commitment to the use of registries, where appropriate, to attempt to clarify the link between toxic exposures and adverse health outcomes. In 1980, the Public Health Service convened a group of experts to address this issue. The findings of this committee were presented to the Assistant Secretary for Health in June 1982 and with your permission, I would like to provide them for the record.

[The following was received from Dr. Houk:]

REPORT OF HHS/CCERP SUBCOMMITTEE ON USE OF LONG-TERM REGISTRIES FOR HEALTH FOLLOWUP OF POPULATIONS EXPOSED TO TOXIC HAZARDS

HEALTH FOLLOWUP REGISTRIES

I. Introduction

It is commonly suggested that health followup registries[1] be established as a means for assessing potential chronic or subacute health consequences of toxic chemical or radiation exposures in human populations. Although the concept of registry followup is intuitively simple, many practical difficulties exist. The issue is of growing importance, however, since opportunities for toxic exposure are increasing in industrial societies and since the low-dose nature of most such exposures focuses attention more on delayed than on acute health problems.

This report summarizes the principal considerations that underlie the establishment and operation of health followup registries. Its purpose is to assist policy development in the Public Health Service regarding promotion and support of such registries. Impetus for the report comes from a) recent toxic exposure episodes in which registry development has received close attention (Love Canal, Three Mile Island, Michigan polybrominated biphenyl (PBB) exposure), b) increasing concern for health followup registries of workers exposed to chemical toxins (for example, PCB, dioxin), and c) present emphasis on control of toxic wastes through the newly enacted "Superfund" legislation (Public Law 96-50: Comprehensive Environmental Response, Compensation, and Liability Act of 1980).

II. Establishment of registries

A health followup registry may be established for various scientific or medical reasons, ranging from assessment of health effects to early diagnosis and treatment of illness. It may also be established in a variety of exposure settings: known exposure to known hazard, potential exposure to known hazard, known exposure to potential hazard, or potential exposure to potential hazard. Different settings evoke different levels of scientific justification for registry followup or may result in different levels of followup intensity. Although, in practice, decisions regarding establishment of registries are often influenced by public concern, the decision process as far as possible should be dispassionate and objective. Considerations that deserve attention in this process can be described under three headings: scientific value and feasibility, resource availability, and ethical issues.

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[1] The term "registry" can connote both disease registries in which groups of persons with a particular disease are available for studies of disease etiology and exposure registries in which persons with particular exposure histories are followed in hopes of defining health consequences. This report concerns exposure registries exclusively.

A. Scientific value and feasibility

Scientific considerations include 1) the relative importance of hypotheses that may be tested through any particular registry, 2) accessibility and size of the population to be registered and followed, 3) the adequacy of exposure information available for the population, and 4) the adequacy and availability of comparison information regarding unexposed populations.

1. Testable Hypotheses.—Before establishing a registry followup study, it is important to articulate as precisely as possible the scientific hypotheses that may eventually be tested by such a study. Possible settings range from ones where the nature of toxins and exposures is clearcut to ones where few data are available. Precise hypotheses will be more difficult to frame in the latter setting. It is also conceivable that, in situations where greater information is at hand, particular toxins may be of relatively little concern with respect to delayed health effects or may already be adequately understood. Although absolute answers cannot be expected in assessing the relative strength of proposed hypotheses, it is important that judgments be made as a first step before proceeding.

2. Population Size.—Aside from the intrinsic value of proposed hypotheses, various practical aspects of the population setting dictate whether or not testing such hypotheses is feasible. One is the size and accessibility of the population to be registered and followed. Most hypotheses regarding delayed toxic effects concern diseases of low frequency such as cancer which require a large population sample or a large accumulation of person-years for valid epidemiologic assessment. Small populations, particularly if dose levels are estimated to be low, will be less informative. This problem may be partly surmountable if registries of several small populations exposed to the same or similar toxins can be pooled for eventual analysis, although such pooling may be complicated by extraneous group differences. In any case, the need for large populations is itself a practical deterrent in view of resources needed to establish and maintain registries. Also of practical concern is the accessibility of populations for registration and followup. Registry development is easier in relatively immobile populations or where well-developed mechanisms already exist for tracing individuals. In the United States neither condition is common.

3. Exposure Information.—Of crucial importance is the state of exposure information available not just for the population as a whole but for each number of the population. The most attractive setting is one where a precise objective measure of each individual's exposure is available, either by direct biologic measurement or by some reliable reconstruction of individual dose, and—where information exists on other important variables related to health outcomes. Analysis can then relate individual health outcomes to individual exposures with some hope for objective dose-response assessment of the population. Situations where such precise individual information is available are rare. This is particularly true since toxic exposures may be transient, involving materials which are quickly cleared from the body and hence not available for dose assessment when plans for health studies are being marshalled some time after the exposure episode (persistent chemicals such as PCB and PBB are notable exceptions). Useful studies can be constructed, of course, using indirect exposure assessments either for individuals or for groups of individuals of individuals (work histories, records of residence near toxic waste dumps, etc.). However, the most interpretable situation is one in which direct information is retrievable.

4. Comparison Populations.—Perhaps the most critical scientific consideration is the availability of adequate population data against which to compare health outcome findings in exposed populations. In practice, this is often a relative matter whereby a relatively highly exposed population is compared to one with relatively light exposure (since for most toxic materials today it is not so much a matter of whether or not one is exposed as how much one is exposed). In the ideal followup registry such high-low comparisons can be made internally. This implies, of course, that precise individual exposure data, or sufficiently detailed group data, are at hand and that population size is adequate, the combination permitting calculation of dose-response relationships. Even under ideal circumstances, however, there may be need for comparison with outside populations, as is the case with more usual situations where exposure information is less precise and population size is limited. Whatever the setting, the important issue is comparability of populations. Information must be available to assess whether the exposed and unexposed populations being compared are in fact comparable in most respects other than the toxic exposure of interest. Such information may involve a fairly extensive array of variables ranging from standard demographic information to data concerning competing biologic influences, whether comparison populations are generated de novo in connection with a particular registry or are derived from existing national data sources (see below). In the absence of such mechanisms for making comparisons among populations, with adequate allowance for comparability considerations, registry followup studies are ill-advised.

B. Resource availability

The long-term, continuing nature of health followup registries dictates two special resource considerations: the need for sustained funding support to be committed for time periods measured in terms of decades and the need for sustained institutional commitments to maintain and utilize registries once started. Both of these considerations are self-evident, yet they may be the most difficult to fulfill given short-term shifts in government funding policies and the finite professional lifespan of individual investigators. Yet without consistent policy guaranteeing steady long-term support for followup registries, initiation of registry systems may be non-productive.

Institutional commitment at the federal level may call for the creation of some enduring focus for registry maintenance, complete with funding responsibilities. The existence of such a federal focus, perhaps within the PHS in cooperation with EPA, need not mean, of course, that all such registries would be centralized there, although bureaucratic pressure might inevitably develop in that direction. Instead, individual registry maintenance might better be a local concern through appropriate institutional commitments by state health departments and university centers, using the federal focus as a coordinating resource.

C. Ethical considerations

The third general area which needs consideration concerns the ethical impact of long-term followup registries on the individual persons being followed. No registry should be undertaken without informing each member of the population to be studied about the purposes of the registry and how it will or will not affect her or him (and if necessary her or his offspring), and without seeking each individual's consent for participation. Such informed consent is an essential part of any study; however, in the case of long-term population followup, it involves a greater ethical problem, not widely recognized, particularly for registries which will actively follow populations through repeated questionnaires or home visits. The problem centers on the sustained awareness by each individual being followed that he or she was exposed to a particular toxin and that it may yet affect his or her health. The difficulty that therefore must be resolved during the initial process of deciding to establish registry followup is whether such awareness is justified by the potential benefit for society as a whole from eventual registry findings. Potentially, this central ethical concern may favor the preferential development of registries using passive or non-obtrusive followup techniques (including record linkage) as opposed to registries relying on active population contacts. Such a potential trade-off would be of particular scientific concern in registry followup settings where interest centers on morbidity health parameters not easily assessed from existing record linkage systems.

III. Operation of registries

Once it is decided to establish a long-term health followup registry in a particular toxic exposure situation, attention shifts to how the registry will operate. As suggested already, many options exist, depending on the purposes the registry will serve and the resources available. Four areas of operation deserve comment: (1) the nature of baseline data to be collected at the start, (2) procedures to be used in population followup once baseline data are collected, (3) methods for analyzing eventual registry results, and (4) questions regarding where registry operations should be located and who should have access to registry data.

A. Baseline data

Three kinds of information are necessary as baseline data for long-term health followup of individuals. First are identification data, essential for tracing individuals over time and for linking registry information to independent data sets which can define different aspects of health outcome (for example, vital records, hospital records). In this category are name (including parents' names and women's maiden names), day, month, and year of birth, social security number, full address, phone number, addresses and phone numbers of close neighbors or relatives, name and address of employer, and available registration information for such things as insurance policies, union memberships, and veteran's benefits. All of these items can be invaluable for eventually tracing the whereabouts and vital status of persons participating in registry followup. The first 3 items (names, date of birth, social security number) are of special importance in that they represent the core requirement for potential followup through the National Death Index or other administrative record systems.

The second information category concerns documentation of individual exposure to the toxin or toxins of interest. Depending on the registry setting, such data may take many forms. Ideally it would center on some objective and precise biologic measurement of toxin levels in individuals. Lacking that (or in addition), indirect indicators of exposure should also be recorded: presumed mode of exposure (water, occupational contact, etc.), pertinent levels of toxin measured in the person's immediate environment, and duration of residence or work in relation to toxin exposure.

The third category of information comprises health covariates, facts about an individual's existing health, habits, and occupation that may influence eventual health outcomes (other than the particular toxic exposure of concern). Lists of information in this area can become formidable, and it is hard to define precisely what an option record should contain. Under existing health status one might confirm major health problems (for example, cancer, diabetes, genetic abnormalities), the relative importance of which would be strongly determined by the particular hypothesis being pursued by the registry followup study. Personal habits must include information about cigarette smoking and alcohol use but could also include information on many other items, particularly use of drugs or medications. Again, selection of items to record will depend on registry objectives. Occupational history is essential, although the detail of such history in terms of nature of employment, potential toxic exposures on the job, and changing occupations over time or throughout a lifetime can be most difficult to resolve.

The questionnaire administered to subjects enrolled in registry followup must be of manageable size, and hence not all items of potential interest can be included. In preparing questions to be asked of enrollees, particular attention must be given both to the precise format in which the questions are asked (so that comparison or linkage with similar data sets may later be possible) and to issues of information reliability: do the questionnaire responses provide hard objective data, and if not will they be verifiable from independent sources (hospital records, for instance)? If baseline data will require independent confirmation from other sources, operational plans for the registry must allow for such work to be done.

B. Followup procedures

Like the items of information recorded, procedures for subject followup should be fashioned to suit the nature and purpose of the particular registry. Where health endpoints of interest consist of disorders that are reasonably completely accounted for in mortality statistics (for example, many types of cancer), followup may be relatively passive. After initial registration, no further subject contact may be necessary, the registry relying on linkage of identifying data with death certificates via the National Death Index for deaths in 1979 and later and via Social Security records for earlier deaths, and subsequent tracing of available clinical records. Where morbidity information is desired, however, particularly if morbidity assessment of the registry population is needed at intervals throughout the total followup period, varying degrees of personal contact with registry subjects will be necessary. This can be done via phone, mailed questionnaire or interview visits to obtain desired information which subsequently may require confirmation through review of pertinent medical records or physician contacts. If biologic measurements of toxin levels in subjects are feasible and are needed to define changes in levels over time, collection of pertinent biologic specimens can be combined with the process of subject followup. Extent and frequency of followup, of course, is heavily dependent on available resources, since frequent personal contacts with subjects in any registry of reasonable size rapidly becomes a major ongoing activity for the staff involved. Coordination with providers of medical care to the population is necessary, since they often must receive information generated by the registry and serve as a source of data sought by the registry.

C. Analysis of data

Decisions to establish and maintain health followup registries entail commitment to perform full and timely analyses and interpretation of the data as they are assembled. Although this commitment may seem to go without saying, it deserves specific emphasis because of the extraordinarily long time periods involved in registry followup. It is easy if not likely for investigators or institutions initiating registry followup to lose interest or motivation to complete final analysis of data 20 or 30 years later. The need for analytic commitment must therefore be heavily stressed.

The process of data analysis and interpretation itself must also not be underestimated. The central need here is for adequate comparison populations. As indicated earlier, these can involve either populations internal to the registry, if the registry cohort itself is large enough and contains precise enough exposure data to permit dose-response exploration, or populations or population data drawn from the general community at large. The most likely scenario for analysis will actually involve combinations of the two, both internal and external comparisons, the latter often having the statistical advantage of large sample size.

General community data which may be feasible for use range upward in complexity from death certificates to various periodic population samples performed in this country both for developing health statistics and for other purposes. Prominent among these survey sources which might be useful in comparative analysis of registry data are the HANES and HIS programs operated by NCHS and the CPS (Current Population Survey) as well as the decennial census. Unfortunately the usefullness of such population samples is presently limited because they provide neither adequate record linkage information (e.g., social security number) nor certain items of information essential or important for health analysis studies. Particularly useful would be the eventual inclusion of a simple smoking history as part of the CPS or of the census itself. Efforts are underway, however, to meet this need at least partially through creation of data files for use in research regarding illness etiology (e.g., the Linked Administrative Statistical Sample project).

A final point with respect to data analysis is the importance of independent peer review both in the design phase of registry development and during the process of data analysis. The wide and multilateral public interests which focus on environmental exposures and on their potential long-term health implications dictate that broad scientific concensus underlie and sustain registry work once established. To establish and maintain an effective peer oversight system may be a sizable task but one which might appropriately be conducted through the central federal focus proposed earlier.

D. Registry location and access

It seems important, for optimal registry operation and data analysis, that the whole scope of work related to particular followup registries be focused in appropriate local health agencies or universities. The alternative that registry data collection be handled by one group, perhaps a local institution, and that data analysis later be handled elsewhere, perhaps at a central national focus, lacks the professional continuity and motivation necessary to fullfill registry objectives. The guiding principle, therefore, should be to encourage that registries be operated entirely in local settings where local community responsibility is felt and hopefully where local professional interest and competence can be brought to bear. There still remains, the need for a national focus for sustained registry support and policy guidance.

Data contained in registries should be available, as a basic rule, to any investigator who has appropriate scientific credentials and can demonstrate legitimate scientific need for use of the data. Guidelines whereby such access is guaranteed yet controlled will need to be developed. It is not desirable that particular individuals or institutions establishing registries consider that the data so generated remains their particular possession until their particular research purposes are fulfilled. The inherently public nature of data contained within registries of this sort must therefore be reaffirmed.

At the same time, the privacy of individual registry subjects must be scrupulously preserved. Such assurance must be stated in clear and unequivocal terms during the initial process of informed consent and must be safeguarded consistently thereafter. Again, the importance of this fundamental principle is underscored by the long-term nature of potential data usage in the context of long-term registry followup.

IV. Summary and recommendations

This brief review of long-term health followup registries stresses their complex nature scientifically, logistically, and ethically. At the same time, such registries are becoming of increasing importance for assessing chronic health consequences of environmental toxic exposures. As suggestions for PHS action in this area at the present time, this subcommittee would offer the following 2 particular recommendations:

(1) While particular registries are best managed on a continuing basis at local institutional levels, the long term commitments involved and the need for scientific consistency and comparability among registries call for development of a national focus, appropriately within PHS in cooperation with EPA, or promoting policy guidelines and peer review and for assuring sustained support for registry operations once initiated. The PHS should act to develop such a focus.

(2) Eventual analysis of registry followup data can be greatly enhanced if particular national data resources can be improved with respect to accessibility and utility

for health followup studies. The PHS should actively promote such improvements, both within its own agencies and within other parts of the federal establishment.

Dr. HOUK. This report on registries is not totally sufficient to establish a policy for nonoccupational registries within the PHS. There are too many unanswered questions:

For example, what measures of exposure should be used to set guidelines for eligibility for inclusion in a registry?

To what extent are people willing to participate over the course of a lifetime in active followup registries—a constant, stressful reminder of the fact that they were exposed to a toxic substance?

How can we assure that a commitment made today to establish and maintain a registry will be carried through over a period of 10, 20, 30 or 40 years? I might add, Mr. Chairman, that the resources for this are not inconsequential.

What level of followup is necessary, scientifically, to provide estimates of risk which can then be used to make sound decisions to manage the risk?

We are seeking to answer these questions. On December 21, 1983, the PHS is convening a meeting of scientists who have managed major long-term followup registries to seek their advice, based on their practical experience.

In carrying out our responsibilities under Superfund, we will cooperate with States, and with other Federal and local officials to establish and maintain registries of persons exposed to toxic substances as a result of an environmental exposure, whenever their inclusion in such registries would be scientifically appropriate or valuable for long-term followup or specific scientific studies. We are committed to defining the appropriate circumstances for such registries to be reviewed by scientists, and others, both within and outside Government by the end of May 1984.

In the meantime, both CDC and the agency for toxic substances and disease registry will continue to respond to specific incidents of human exposure to toxic or hazardous substances. We will also continue our efforts to measure both the immediate and long-term health effects, and to make sound recommendations to EPA for the attenuation of the risks.

Although the results of our studies may not provide the conclusive answers about health risks which are so necessary, we have hope that we can demonstrate strong associations, where they exist, between exposure and adverse health outcomes so that, very importantly, sir, reasonable people can make reasonable judgments.

This concludes my testimony.

Mr. LEVITAS. Thank you, Dr. Houk.

[The statement of Dr. Houk follows:]

STATEMENT OF VERNON N. HOUK, M.D., DIRECTOR, CENTER FOR ENVIRONMENTAL HEALTH, CENTERS FOR DISEASE CONTROL AND ASSISTANT ADMINISTRATOR, AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY, PUBLIC HEALTH AND HUMAN SERVICES

I am Dr. Vernon N. Houk, Director, Center for Environmental Health, Centers for Disease Control (CDC) and Assistant Administrator of the Agency for Toxic Substances and Disease Registries (ATSDR) of the Public Health Service (PHS), Department of Health and Human Services. I am pleased to be here this morning to discuss with you some of our activities in implementing the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, commonly known as Superfund.

In your letter of invitation requesting that I appear, you asked that my testimony concentrate on the issue of compensation for persons exposed to toxic chemical wastes. Because I have no offical responsibilities related to compensation policy, my testimony will be limited to those questions contained in your letter which address my duties in managing specific studies of the human health effects of exposure to hazardous substances. I direct a major effort within the Public Health Service to study the human effects of community exposures to hazardous substances. From these studies, scientists on my staff and I frequently formulate recommendations to limit or prevent further exposure. I do not, however, recommend or set policy related to the compensation of persons inadvertently exposed and/or injured.

As you know, a Cabinet-level work group has been formed to address issues related to toxic victims' compensation. The work of this group may be useful to you and your colleagues in this matter.

I would like to illustrate our Superfund activities under section 104i(1) related to the establishment that maintenance of registries, and section 104(5) related to determining the relationships beween exposure to toxic substances and illness. These two sections of the Superfund law present us with some of the most complex and perplexing scientific problems faced by public health officials today.

In your letter of invitation asking for me to serve as a witness before this committee, you asked that I describe to you our work at Times Beach, MO, where we have been attempting to measure the human health effects, if any, of exposure to 2,3,7,8 tetrachlorodibenzo-p-dioxin, the most toxic form of dioxin. Although the dioxin contamination incident in Missouri is not necessarily typical of the overall problem addressed by Superfund it is useful for me to illustrate our work by describing both the immediate and the long-term program we have implemented in cooperation with the State of Missouri in response to the dioxin contamination. In the early 1970's waste oil which had been mixed with dioxin contaminated byproducts from the manufactuer of various chemicals was spread widely in the town of Times Beach and in many other areas of Missouri to control dust. Just before Christmas last year, the Environmental Protection Agency informed us of the positive results of their doxin testing program in Times Beach. EPA asked CDC to advise them, the Federal Emergency Management Agency, and State officials regarding the potential human health effects which could result from contact with dioxin-contaminated soil.

Unfortunately, there are no easy answers to this question. At the time, and even today, very little is known about the human effects of exposure to very low levels of dioxin. Almost all of what is known has been learned from animal studies, that is by exposing various species of animals to various levels of dioxin and observing the outcome, then extrapolating from that information to arrive at assumptions about kind of health effects which might be expected in humans. There have also been studies of workers who where inadvertently exposed to higher levels of dioxin while in the job, but the method of exposure in an occupational setting is quite different from the situation in Times Beach.

From these studies we knew that dioxin could be potentially very highly toxic. Based on the non-human animal data, we assume it could contribute to the development of certain kinds of cancerous tumors and could adversely affect the unborn children of women who were exposed to the substance while pregnant. We also knew that dioxin accumulates in fat and in other human organs, particularly the liver, and is not easily excreted from the body. There is no known cure for dioxin poisoning. There was also great uncertainty about the extent to which dioxin in soil could be absorbed by humans who either worked in their yards, gardened, or otherwise were exposed to the contaminated dirt.

Between the time the soil samples had been collected by EPA and the analysis had been carried out, the town of Times Beach was flooded when heavy rains caused the Merrimac River to overflow it banks. When we received the results of the testing program the citizens of Times Beach had temporarily moved away from their flooded homes. Because of our deep concern about the potential for harmful health effects from dioxin, and the many uncertainties about this particular exposure incident and our fear that the flooding may have moved contaminated soil into the houses, we recommended that the citizens of Times Beach not return to their homes. Using our best estimate of exposure and health effects, we assessed the risk of dioxin exposure under the conditions present at Times Beach to be excessive if continued exposure were permitted to levels of dioxin above 1 part per billion (ppb).

We recognized then, and we recognize now, that this estimate of risk was based on a number of assumptions that required testing. The National Toxicology Program, within the Public Health Service, began conducting tests to determine whether, in the laboratory, animals exposed to the dioxin-contaminated soil from Times Beach would actually absorb dioxin into their systems. We also convened a panel of scientists from within and outside Government, from academia, industry and environmental groups to review the process, assumptions, and calculations we used to arrive at the 1 ppb value. This group gave us valuable suggestions for improving the process, but did not take issue with our conclusion that long-term exposure to dioxin in soil at levels above 1 ppb in Times Beach should be avoided.

The State of Missouri, the St. Louis University School of Medicine, and St. Joseph Hospital participated with scientists from the CDC in a pilot epidemiologic study of residents of several eastern Missouri dioxin Superfund sites, whom we judged to have experienced a spectrum of exposure to dioxin ranging from least heavily exposed to potentially most heavily exposed. The pilot study consisted of a questionnaire survey of current and past health status, a physical examination for clinical signs and symptoms of potential health effects from dioxin, and a battery of specialized laboratory tests. On October 16, 1983, we reported the findings of our Human Health Effects Study.

I would like to submit the Executive Summary of our report to you for the record. In summary, we could not definitively identify any significant health problems associated with exposure to the levels of dioxin present for this study group. While these overall first observations are encouraging and do not indicate significant immediate dioxin-related effects in this pilot study population, several points should be kept in mind: (1) inconclusive results on a number of tests will be evaluated in further studies; (2) subtle health effects could possibly have been missed; (3) certain areas, such as psychological or behavioral function were not evaluated in this pilot study; and (4) some degree of exposure misclassification of individuals in the study may have occurred.

It would be unwise to extrapolate from these preliminary findings of our pilot study in Missouri to other dioxin-exposure incidents; either in the occupational setting, in other community settings, or among veterans of the Vietnam War. We intend to follow up with additional studies to delineate better the health effects in persons accidentally exposed to this potentially toxic compound.

Thus far, I have concentrated on our efforts to evaluate the immediate acute or sub-clinical effects of dioxin in people. You also asked about our efforts to identify the chronic, long-term effects which may result from exposure to low levels of toxic substances in the environment. We are attempting to answer the question: What long-term health effects, if any, are there to persons exposed to dioxin and other hazardous substances? One of the most commonly recommended tools for studying the chronic or long-term effects of exposure is to construct a registry of persons exposed. The persons in the registry can be tested periodically to determine their health status and eventually to determine the cause of their death.

The use of registries to evaluate the long-term human health effects of toxic chemical exposures began prior to the passage of Superfund. In 1975, for example, CDC, in cooperation with the State of Michigan, set up a registry of 4000 persons exposed to polybrominated biphenyls after the occurrence of an accidental mixture of that chemical into feed grains. Also, CDC's National Institute for Occupational Safety and Health (NIOSH) has been studing the health effects of chemical exposures among workers through the establishment of registries. Among these is the worker registry of employees of industries which manufacture or manufactured products which may have been contaminated with dioxin. The intention is to assess the health status of workers who are listed in these registries and possibly to identify illness which may be related to a particular toxic exposure in the work place.

Registries cannot be used to give quick conclusive answers to the many questions that people ask about the effects of toxic chemical exposure on their health or the health of their children. Even the most active and aggressively pursued studies using registries require decades to produce meaningful results. It is also true that even when the results of long-term studies are made available, the findings are likely to be hotly debated by scientists who honestly represent different points of view. I point out these inherent limitations in registries to call to your attention the fact that expectations for registries being used as tools to set public policy in the near future may be unrealistically high.

Even with these limitations, the Public Health Service has a commitment to the use of registries, where appropriate, to attempt to clarify the link between toxic exposures and adverse health outcomes. In 1980, the Public Health Service convened a group of experts under the leadership of a CDC scientist to review the scientific and programmatic utility of registries and to make recommendations for a PHS policy. The findings of this committee were presented to the Assistant Secretary for Health in June of 1982 and with your permission, I would like to provide them for the record.

This report on registries is not totally sufficient to establish a policy for non-occupational registries within PHS. There are too many unanswered questions:

What measures of exposure should be used to set guidelines for eligibility for inclusion in a registry?

To what extent are people willing to participate over the course of a lifetime in active followup registries—a constant, stressful reminder of the fact that they were exposed to a toxic substance?

How can we assure that a commitment made today to establish and maintain a registry will be carried through over a period of 10, 20, 30, or 40 years?

What level of followup is necessary, scientifically, to provide estimates of risk which can then be used to make sound decisions to manage the risk?

We are seeking to answer these questions. On December 21, 1983, the PHS is convening a meeting of scientists who have managed major long-term followup registries to seek their advice, based on their practical experience.

In carrying out our responsibilities under Superfund, we will cooperate with States, and with other Federal and local officials to establish and maintain registries of persons exposed to toxic substances as a result of an environmental exposure, whenever their inclusion in such registries would be scientifically appropriate or valuable for long-term followup or specific scientific studies. We are committed to defining the appropriate circumstances for such registries to be reviewed by scientists, and others, both within and outside government by the end of May 1984.

In the meantime, both CDC and the Agency for Toxic Substances and Disease Registry will continue to respond to specific incidents of human exposure to toxic or hazardous substances. We will also continue our efforts to measure both the immediate and long-term health effects, and to make sound recommendations to EPA for the attenuation of the risks.

Although the results of our studies may not provide the conclusive answers about health risks which are so necessary, we have hope that we can demonstrate strong associations, where they exist, between exposure and adverse health outcomes so that reasonable people can take reasonable actions to protect public health and the environment.

This concludes my testimony. I would be pleased to answer your questions.

Mr. LEVITAS. Now, we will hear from Dr. Fingerhut.

Dr. HOUK. Mr. Chairman, with your permission Dr. Millar asked that we submit his statement for the record. I will summarize it. Then Dr. Fingerhut.

Mr. LEVITAS. Will Dr. Fingerhut be in a position to respond to questions?

Dr. HOUK. Yes, sir.

Mr. LEVITAS. First, the entire statements of all the witnesses will be made part of the record without objection.

Dr. Houk, if you would summarize for us Dr. Millar's statement.

Dr. HOUK. Thank you, Mr. Chairman. Dr. Millar is disappointed that he could not be here today. He particularly did want to come but, as you mentioned, he could not. He did ask me to summarize his statement.

I think this reflects the very close and necessary working relationship within the Centers for Disease Control between the occupational component and the nonoccupational component, and why we must work together and that we have worked together.

Dr. Millar is Director of the National Institute for Occupational Safety and Health of the Centers for Disease Control, Public Health Service, of the Department of Human Services. With me today is Dr. Marilyn Fingerhut, an epidemiologist in NIOSH's Division of Surveillance, Hazard Evaluations and Field Studies.

As you know, NIOSH was established by the Occupational Safety and Health Act of 1970, and is charged with studying the human health effects of occupational exposures to hazardous substances.

From the results of these studies and other available data, we make recommendations to prevent disease and injury among workers from such exposures. NIOSH, however, neither establishes nor recommends policy related to the compensation of persons exposed and injured.

It is in the performance of the Institute's research mission that several studies on dioxin are being conducted by NIOSH. I will first describe the "Dioxin Registry and Mortality Study" and then summarize other research exploring the relationship between soft-tissue sarcoma and exposure to dioxin.

The NIOSH dioxin registry has identified a cohort of workers who are known, by records of employment, to have worked in chemical processes which generated dioxin as a contaminant. Hence, these are workers who are known to have been potentially exposed to dioxin. Upon completion, the registry will contain a listing of the names, vital status, demographic information, and work histories for all U.S. workers potentially exposed to dioxin during the production of products such as trichlorophenol or pentachlorophenol. Enrollment of workers into the registry will be completed by the end of December 1983; 5,000 such workers at 10 U.S. facilities have already been identified. We are currently evaluating data systems at four additional sites, and, if the records are adequate, approximately 1,000 more workers will be added to the registry.

This group constitutes a unique population of persons with documentation of potential exposure to dioxin among whom meaningful epidemiological observations can be made. Many of the workers already identified have been exposed to dioxin-contaminated materials for 20 years or more. The registry will provide the data base necessary to conduct epidemiological research, the results of which will be a valuable source of information regarding potential human health effects from exposure to dioxin. In addition, the International Agency for Research on Cancer of the World Health Organization, with funding provided by the National Institute of Environmental Health Sciences, National Institutes of Health, is developing an international dioxin registry for countries other than the United States.

The mortality study, which is already underway, tests the hypothesis that soft-tissue sarcoma and other cancers may be associated with exposure to dioxin. Additionally, because information that can be obtained from a mortality study is limited to those causes of death which are coded on death certificates, we will seek more detailed health information through a questionnaire survey and a medical examination of several hundred living members of the cohort of exposed workers. In the first or pilot stage of this effort, a questionnaire and medical study will be administered to about 500 exposed workers and a comparable number of persons who have not been exposed. This study is designed to evaluate the presence of effects on the skin, liver, neurological, and immunological systems that have occurred among workers after exposure.

We also have extensively reviewed medical literature concerning the link between exposure to dioxin and soft-tissue sarcoma. In 1977, several clinical cases of soft-tissue sarcoma were observed among Swedish lumberjacks who had previously been exposed to dioxin-contaminated products. This observation led researchers in

Sweden to conduct two separate epidemiologic studies of the relationship of dioxin to soft-tissue sarcoma. Both studies found that workers with exposure to dioxin had a fivefold to sixfold greater risk of developing a soft-tissue sarcoma than those not exposed to dioxin.

Subsequently, three studies were conducted in the United States of workers exposed to the herbicide or its chemical precursor, trichlorophenol, products whose manufacture produces dioxin as a contaminant. No excess in total mortality or in deaths from malignant neoplasms was found in these studies. However, the number of workers in each study was small and the expectation of rare causes of death in such small groups was very low. Consequently, the failure to find excesses was of little significance. In order to have a meaningful sample to evaluate, we combined the data from the three studies and added results from a fourth unpublished study.

In the merged cohorts there were three deaths—2.9 percent of the total 105 deaths—reported by the researchers to be due to soft-tissue sarcoma; only 0.07 percent of total deaths, or less than 1 death, due to soft-tissue sarcoma was expected. A fourth individual in one of the cohorts died recently; the death also was reported as due to soft-tissue sarcoma. Three additional persons who worked at 2,4,5-T production facilities were reported by their physicians to have had soft-tissue sarcomas. Thus a total of seven cases were initially reported as due to soft-tissue sarcoma among workers exposed to dioxin.

In July, before the House Committee on Science and Technology, we discussed this issue. At that time, we knew of seven cases reported as soft-tissue sarcoma in U.S. workers. We indicated then that we were actively pursuing clarification of these reported cases.

In continuing these investigations, we have sought to confirm both the diagnosis and the history of exposure in each of these seven cases. We have obtained detailed work histories for each person and sent available pathological tissue specimens from each case for review by the Armed Forces Institute of Pathology and by an independent expert on the pathology of soft-tissue sarcomas.

Based on the expert review of specimens, we have recently stated that of the four cases reported to be soft-tissue sarcoma by the authors of the four U.S. studies, two of these cases are confirmed by expert pathologists as such. Both had documented exposure to dioxin-contaminated products. Of the three cases reported by physicians all were found to have soft-tissue sarcoma based on the review of their specimens by the experts; however, exposure of these employees to 2,3,7,8-tetrachlorodibenzodioxin-contaminated products could not be verified in a review of their work histories. We cannot determine whether these three workers had exposure to this contaminant.

As a result of a rigorous scientific process, we have now determined the following:

Four individuals had work histories documenting their employment in the manufacturing process of dioxin-contaminated products. Two of these have histologically verified soft-tissue sarcoma and two do not.

The three additional individuals who have histologically verified soft-tissue sarcoma have work records that do not provide documentation of their employment in the manufacturing process of the contaminant in question.

In sum, of the seven cases which were initially reported only two have histologically verified soft-tissue sarcoma for which we have documentation of their employment and the manufacturing process of the contaminant in question.

The two verified cases, while a small number, still represent an excess over the less than one that would be expected statistically. While our observations to date do not confirm a causal link between dioxin and soft-tissue sarcoma, they give us reason for continuing our investigations.

Mr. Chairman, this concludes Dr. Millar's summary. We would be happy to answer any questions you and members of the subcommittee may have.

[The statement of Dr. Millar follows:]

Statement by J. Donald Millar, M.D., Assistant Surgeon General, Director, National Institute for Occupational Safety and Health, Centers for Disease Control, Public Health Service, Department of Health and Human Services

Mr. Chairman and Members of the Committee: I am Dr. J. Donald Millar, Director of the National Institute for Occupational Safety and Health (NIOSH) of the Centers for Disease Control, Public Health Service, of the Department of Health and Human Services. Thank you for inviting me to testify on studies conducted by NIOSH which relate to long term health consequences among workers exposed to dioxin-contaminated products. With me today from the Institute is Dr. Marilyn Figerhut, an epidemiologist in our Division of Surveillance, Hazard Evaluations and Field Studies.

As you know, NIOSH was established by the Occupational Safety and Health Act of 1970, and is charged with studying the human health effects of occupational exposures to hazardous substances. From the results of these studies and other available data, we make recommendations to prevent disease and injury among workers from such exposures. NIOSH, however, neither establishes nor recommends policy related to the compensation of persons exposed and injured. As you know, a Cabinet-level work group has been formed to address a broad range of issues related to the compensation of the victims for exposure to toxic substances and the results of work by that group, when they become available, should prove useful to you in your deliberations on this issue. Because I have no responsibilities related to compensation policy, my testimony will be limited to those questions contained in your letter which address my duties in managing specific studies of human health effects of exposure to hazardous substances.

It is in the performance of the Institute's research mission that several studies on dioxin are being conducted by NIOSH. I will first describe the Dioxin Registry and Mortality Study and then summarize other research exploring the relationship between soft tissue sarcoma and exposure to dioxin.

The NIOSH Dioxin Registry had identified cohort of workers who are known, by records of employment, to have worked in chemical processes which generated dioxin as a contaminant. Hence, these are workers who are known to have been potentially exposed to dioxin. Upon completion, the Registry will contain a listing of the names, vital status, demographic information, and work histories for all U.S. workers potentially exposed to dioxin during the production of products such as trichlorophenil, 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), or pentachlorophenol. Enrollment of workers into the Registry will be completed by the end of December 1983; 5,000 such workers at ten U.S. facilities have already been identified. We are currently evaluating data systems at four additional sites, and, if the records are adequate, approximately 1,000 more workers will be added to the Registry.

This group constitutes a unique population of persons with documentation of potential exposure to dioxin among whom meaningful epidemiological observations can be made. Many of the workers already identified have been exposed to dioxin-contaminated materials for twenty years or more. The Registry will provide the data base necessary to conduct epidemiological research, the results of which will be a valuable sorce of information regarding potential human health effects from exposure to dioxin. In addition, the International Agency for Research on Cancer of the

World Health Organization, with funding provided by the National Institute of Environmental Health Sciences, National Institutes of Health, is developing as international dioxin registry for countries other than the United States.

The mortality study, which is already under way, tests the hypothesis that soft tissue sarcoma and other cancers may be associated with exposure to dioxin. Additionally, because information that can be obtained from a mortality study is limited to those causes of death which are coded on death certificates, we will seek more detailed health information through a questionnaire survey and a medical examination of several hundred living members of the cohort of exposed workers. In the first or pilot stage of this effort, a questionnaire and medical study will be administered to about 500 exposed workers and a comparable number of persons who have not been exposed. This study is designed to evaluate the presence of effects on the skin, liver, neurological and immunological systems that have occurred among workers after exposure.

We also have extensively reviewed medical literature concerning the link between exposure to dioxin and soft tissue sarcoma. In 1977, several clinical cases of soft tissue sarcoma were observed among Swedish lumberjacks who had previously been exposed to dioxin-contaminated products. This observation led researchers in Sweden to conduct two separate epidemiologic studies of the relationship of dioxin to soft tissue sarcoma. Both studies found that workers with exposure to dioxin had a five to six-fold greater risk of developing a soft tissue sarcoma than those not exposed to dioxin.

Subsequently, three studies were conducted in the U.S. of workers exposed to the herbicide 2,4,5-trichlorophenoxy acetic acid (2,4,5-T) or its chemical precursor, trichlorophenol, products whose manufacture produces dioxin as a contaminant. No excess in total mortality or in deaths from malignant neoplasms was found in these studies. However, the number of workers in each study was small and the expectation of rare causes of death in such small groups was very low. Consequently, the failure to find excesses was of little statistical significance. In order to have a meaningful sample to evaluate, we combined the data from the three studies and added results from a fourth unpublished study. In the merged cohorts there were three deaths (2.9% of the total 105 deaths in the merged cohorts) reported by the researchers to be due to soft tissue sarcoma; only 0.07% of total deaths, or less than one death, due to soft tissue sarcoma was expected. A fourth individual in one of the cohorts died recently; the death also was reported as due to soft tissue sarcoma. Three additional persons who worked at 2,4,5-T production facilities were reported by their physicians to have had soft tissue sarcomas. Thus a total of seven cases were initially reported as due to soft tissue sarcoma among workers exposed to dioxin. In July of this year, we reported these preliminary observations to the House Committee on Science and Technology.

In continuing these investigations, we have sought to confirm both the diagnosis and the history of exposure in each of these seven cases. We have obtained detailed work histories for each person and sent available pathological tissue specimens from each case for review by the Armed Forces Institute of Pathology and by an independent expert on the pathology of soft tissue sarcomas. Based on the expert review of specimens, we have recently stated that of the four cases reported to be soft tissue sarcoma by the authors of the four U.S. studies, two of these cases are confirmed by expert pathologists as such. Both had documented exposure to dioxin-contaminated products. Of the three cases reported by physicians all were found to have soft tissue sarcoma based on the review of their specimens by the experts; however, exposure of these employees to dioxin-contaminated products could not be verified in a review of their work histories. We cannot determine whether these three workers had exposure to dioxin.

In July, before the House Committee on Science and Technology, we discussed this issue. At that time, we knew of seven cases *reported* as soft tissue sarcoma in U.S. workers. We indicated then that we were actively pursuing clarification of these reported cases. As a result of a rigorous scientific process, we have now determined the following:

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The two verified cases, while a small number, still represent an excess over the less than one that would be expected statistically. While our observations to date do not confirm a causal link between dioxin and soft tissue sarcoma, they give us reason for continuing our investigations.

Mr. Chairman, this concludes my summary of the NIOSH dioxin studies. We would be happy to answer any questions you or other members of the subcommittee may have.

Mr. **Levitas.** Thank you, Doctor.

Why don't we hear from Dr. Jasanoff before we get into questions?

Ms. **Jasanoff.** Thank you, Mr. Chairman.

Mr. **Levitas.** Again, Dr. Jasanoff, we welcome you. If you would, give us your full name, employment, and general description of what you do.

Ms. **Jasanoff.** I am Sheila S. Jasanoff, a senior research associate at the Program on Science, Technology and Society at Cornell University. I am pleased to be here this morning to talk about my participation in the study carried out by the National Science Foundation's Division of Policy Research and Analysis about 2 years ago on the issue of victim compensation. I have here with me this morning a copy of the report that was prepared in consequence of that study if the subcommittee should wish to look at it.

The objective of the victim compensation study was to look at the question of policy responses in a more concrete context than it had previously been examined in. So instead of having academic analyses of the alternative approaches, the National Science Foundation took a case study approach which would subject the same facts to different types of compensation models in an effort to yield some conclusions about the comparability of these models and the relative advantages and disadvantages they offer.

One principal thrust of the study at all times was to see how the linkage between uncertainties on the scientific side and procedural uncertainties created by the different compensation approaches fit together to affect the probability of compensation in different cases. And the case studies were chosen to represent a spectrum of substances about which different degrees of scientific information were available, ranging from a relatively well-known health hazard, scientifically well documented, namely, asbestos, though unfortunately that case study was never completed, and three others that went from relatively certain to quite uncertain in the range of scientific information then available.

The study that I personally did was on 2,4,5-T and one focus of interest was the fact that 2,4,5-T, although it fell into the quite uncertain health hazard category, was contaminated by dioxin which was known at that time to be a highly toxic compound. The study was coordinated from the start in ways that would ensure the comparability that we were seeking. The three case study performers met together to decide on which model compensation processes would be looked at, and also what kinds of criteria of evaluation could be applied to the case studies. The objective then was the development of factual situations which we constructed on the basis of information available about exposure conditions, and a fitting of the different fact situations to the approaches taken in the different compensation processes, and finally, an evaluation section in each case study that used the same criteria.

Briefly, the outcome of the study was to show that there were two main ways in which victims of toxic substance pollution can encounter difficulty in trying to obtain some remedy for their injuries. The obstacles fell either into a legal and procedural subgroup or into a science-related subgroup. Obviously there are overlaps between these. But just to exemplify what I mean, in the first subgroup there are things like statutes of limitations which did not take account of the fact that many injuries associated with toxic substances may not become apparent to the sufferer until a certain amount of time has elapsed between the exposure and the injury itself, and these statutes of limitations in some cases completely bar recovery and make it very difficult in others.

In the second category, namely, limitations imposed by science, were the standards of proof that victims of toxic substance pollution would have to meet under virtually all the compensation systems that we looked at. A total of six systems were examined in the course of the study. Only one required no showing of causation, and that was the Social Security disability insurance scheme. The others, even including workers' compensation, which was originally set up to be a relatively noncausal no-fault approach to compensation, did entail a certain showing of a causal relationship between exposure and injury.

In the course of my case study on 2,4,5-T, I concluded that with respect to the causal showing it would be very difficult for the kinds of victims and persons exposed to 2,4,5-T, with the injuries that might be caused by 2,4,5-T to meet the standard of proof, even in systems that were specifically constructed to ease the burden of proof for victims. Thus, one of the main approaches that we were considering at the time was the Brodhead bill. That bill required as a minimum threshold showing that victims would have to establish that their exposure resulted in the etiology of the disease or injury for which they were claiming compensation. Looking at the scientific evidence then available on 2,4,5-T and dioxin, it appeared almost certain that most of the kinds of victims we would expect to see would not be able to meet that showing.

The NSF report did not so much recommend a particular policy approach as summarize the kinds of pros and cons associated with the different policy approaches. My own reaction to the systems we studied was that whatever policy approach is undertaken in this area, if a comprehensive policy is developed for toxic substance victim compensation it will have to combine theoretical approaches that reflect a number of different philosophies of compensation.

Obviously one extreme is to ignore the causal question completely and to say that we want to compensate victims for the injuries they suffer. In this case it makes no sense to have a policy that relies primarily on a producing industry for the funds required for compensation. At the other extreme, one could keep the tort system with no changes. But I think the study amply demonstrated that that system is quite inefficient in a number of regards, and also inequitable in its selection of certain people to compensate and others not. Some sort of middle road is exemplified in the study we did by a proposed collective liability system which would have set up two different approaches to compensation. One would be for victims who could not uniquely identify a manufacturer or producer of the substance that possibly injured them. Those people would be entitled to recovery from a fund, a concept familiar from a number of Federal statutes. The other approach resembled tort law more in that it required victims to go against specified manufacturers and producers, and correspondingly, that approach entitled victims to a high level of compensation because it included punitive damages as well.

This concludes my prepared statement. I would be happy to answer questions.

[The statement of Ms. Jasanoff follows:]

Statement of Sheila Jasanoff, Program on Science, Technology and Society, Cornell University

Good morning, Mr. Chairman and members of the Subcommittee. I an Sheila Jasanoff, Senior Research Associate in the Program on Science, Technology and Society at Cornell University. I an pleased to be here this morning to report on a study of toxic substance victim compensation carried out by the National Science Foundation (NSF) in 1981. The purpose of the study, in which I participated as a consultant, was to evaluate the adequacy of several existing and proposed victim compensation mechanisms, taking into account the impact of incomplete scientific knowledge on the settlement of claims.

In my remarks I will briefly describe the objectives and methodology of the study and the results of my own case study analysis of the herbicide 2,4,5-T. I will also summarize the major conclusions and policy options that emerged from the study as a whole.

STUDY OBJECTIVES AND APPROACH

To provide a concrete factual context for the evaluation of compensation systems, NSF selected a case study approach centering on four substances, each of which presented different gaps in scientific information and different problems of proof for injured parties. The four substances were asbestos (a well-known and scientifically recognized health hazard), nitrilotriacetic acid (as substance of uncertain hazard to health), 2,4,5-T (an uncertain health hazard, but contaminated by a well-known toxic compound, TCDD or dioxion), and diethylstilbesterol (a substance recognized as safe in certain uses but presenting a health hazard to an identifiable subgroup of the population). Upon reviewing work plans submitted by outside consultants, NSF commissioned three case studies focusing on the substances whose effects on human health were most inconclusive and controversial: NTA, 2,4,5-T and DES. (A fourth study on asbestos was commissioned but never completed.)

The performance of the case studies was carefully coordinated from the outset to ensure that the findings would be genuinely comparable. The case study performers met once at the start of the project to select the compensation systems that would be analyzed and a to design a set of common criteria for evaluating them. Each performer agreed to examine the tort liability system and the Brodhead bill (H.R. 5074, 96th Congress, 1st Session), one of the leading legislative proposals for establishing an administrative compensation scheme at the time of the study. Further, each performer was required to evaluate at least one other compensation framework more closely tailored to the facts of the individual case studies. In all, six compensation systems were examined in the three cases studies: the tort system, the Brodhead bill, the LaFalce bill (H.R. 5291, 96th Congress, 1st Session), workers' compensation, social security disability insurance (SSDI), and a proposed collective liability system.

Each case study involved three discrete tasks. First, the scientific information available on each substance was reviewed and the uncertainties relevant to the disposition for compensation claims were identified. Second, each performer constructed a set of hypothetical fact situations to illustrate the kinds of exposure and injuries that might be associated with each substance. Third, the selected compensation mechanisms were applied to these fact situations and the effectiveness of each was measured in terms of the previously agreed upon criteria. Ten of the fifteen evaluation criteria related to the institutional and procedural structure of the compensation systems: choice of forum and factifinder, identification of plaintiffs and defendants, evidentiary showing, financing of awards. The remaining five addressed the equity and efficiency aspects of different compensation processes, including the time to recovery, the cost of pursuing claims to completion, the deterrent effect of awards, and the utility of the process for generating new information about toxic substance-related injuries.

Preliminary drafts of the case studies were circulated among the performers for comment and criticism. In this way a close coordination of the separate efforts was maintained throughout the project. The completed case studies were edited and published by NSF as a research report entitled "Compensation for Victims of Toxic Substance Pollution—Assessing the Scientific Knowledge Base" (March, 1983).

THE 2,4,5-T CASE STUDY

I would like now to turn to my own study of 2,4,5-T and the conclusions I drew from that analysis. At the time of the NSF project, 2,4,5-T, a component of Agent Orange, was already implicated in lawsuits growing out of the use of that defoliant during the Vietnam war. Much of the controversy focused on the effects of dioxin, a contaminant unavoidably produced in the manufacture of 2,4,5-T and present in commercial preparations of the herbicide in at least trace amounts. To assess the availability of compensation for health injuries caused by 2,4,5-T, I constructed a number of fact situations covering the range of possible exposures: those environmentally exposed to aerial application of the herbicide, and those occupationally exposed both during the manufacturing process and in the course of application. The class of potential victims also included children born with birth defects possibly as a result of parental exposure to the substance.

Both 2,4,5-T and dioxin are the subjects of a voluminous scientific literature comprising a wide variety of animal and human studies and examinations of the substances' mutagenic potential. However, a review of this literature (carried out with the assistance of a researcher familiar with 2,4,5-T) revealed surprisingly few areas of consensus. It is well known, for example, that exposure to dioxin and to 2,4,5-T can result in such toxic effects as chloracne and other dermatological injuries, metabolic disorders, neurological symptoms and psychological alterations. But it was unclear in many cases whether the reported effects were induced by 2,4,5-T itself, dioxin, a combination of the two, or some other contaminant. The genotoxic effects of both compounds were even more uncertain. While dioxin was identified as a teratogen at high doses as early as 1969, later studies using 2,4,5-T with known low concentrations of TCDD yielded conflicting results on the question of teratogenicity. Reports on the carcinogenicity of these compounds were inconclusive as well, although there was evidence that chronic administration of TCDD could produce excess neoplasms in test animals.

Given the pervasive gaps and conflicts in the scientific literature, I concluded that it would be extremely difficult for any of my hypothetical victims to recover for their injuries under the three cause-based compensation processes I evaluated (tort law, the Brodhead bill and workers' compensation). Social security disability insurance offered the most certain prospects for recovery, since awards in that framework do not depend on a showing of causation. However, since SSDI covers only persons who have been gainfully employed, this mechanism would offer nothing to unemployed victims, such as environmentally exposed children or children born with birth defects occasioned by pre-natal exposure to 2,4,5-T.

STUDY CONCLUSIONS

All three case studies illustrated certain systematic difficulties confronting persons attempting to obtain compensation for injuries caused by toxic substances. One set of problems relates to the proof of causation, which is the greatest obstacle to recovery for most victims. The inadequacy of current scientific knowledge contributes enormously to the claimant's evidentiary burden. The case studies revealed a persistent dearth of evidence about the low-dose effects of environmental pollutants. Both epidemiological and animal data present inconclusive evidence about the human health effects of toxic substances. In all cases, the difficulty of establishing a scientifically plausible causal showing is compounded by such factors as the cost of expert testimony and the time lag between exposure and the detection of adverse health effects.

A second set of problems relates to administrative and procedural rules that create characteristic barriers to recovery in each compensation system. Tort law, responding only slowly to the specific issue of toxic substances, still presents the greatest number of such impediments, for example, restrictive statutes of limitations, legal obstacles against suing multiple defendants, rigid evidentiary rules excluding speculative scientific information, and limitations on class actions. These features of the tort system have been widely criticized in a number of more recent studies as well, including, prominently, the report to Congress prepared by the Superfund § 301(e) Study Group (September, 1982).

Administrative compensation schemes, such as the Brodhead bill, can ease the path to compensation by specifically addressing both sets of problems encountered in existing recovery processes. Proposals directed explicitly at the issue of toxic substance injuries generally lower the claimant's burden of proof through use of carefully structured rebuttable presumptions. Such proposals can also eliminate artificial impediments, such as overly rigid statutes of limitations, reduce the cost of preparing and processing claims, and facilitate the production and consolidation of relevant scientific information. However, my analysis of the 2,4,5-T case suggested that even the reduced burden of proof associated with administrative compensation processes may be difficult to meet for many toxic substance victims. The case for recovery would remain highly problematic for those exposed to very low doses of environmental pollutants and those whose symptoms are not uniquely related to a particular toxic exposure. In an important sense, then, all cause-based compensation systems are likely to prove under-inclusive, i.e., they will only compensate a fraction of those actually hurt by exposure to toxic substances.

POLICY OPTIONS

The range of mechanisms examined in the NSF study represent two fundamentally divergent pholosphies of compensation. One, exemplified by the SSDI system, forcuses exclusively on the need to make the victim whole and to do so with reasonable efficiency. This approach is unconcerned about the source of the victim's injuries. Although claimants may have to meet minimal threshold requirements, as for receiving SSDI benefits, these have nothing to do with establishing causation. As a result, this approach offers toxic substance victims the surest route to recovery. By eliminating the need to prove causation, it also does away with the burden of grappling with the voluminous, complex and frequently inconclusive scientific record that currently forms the basis for most toxic substance-related claims.

A very different philosophy, one geared to making the "polluter pay," characterizes all cause-based compensation systems. The assumptions underlying this approach are both moral and economic. They include the judgment that the cost of chemically induced injuries should fall on those who caused them, and the belief that such costs can be borne most efficiently by manufacturers and producers, who may, in turn, pass them on to the larger consuming public.

Most of the compensation processes under serious consideration today fall into the second category. However, even under this heading one can discern a number of distinct policy alternatives. One course is simply to let the existing mechanisms evolve gradually to meet the demands created by pollution-related claims. Cases like *Sindell* v. *Abbott Laboratories, et al.*, in which the California Supreme Court adopted the theory of "market share liability," indicate a willingness on the part of the courts to evolve creative doctrines in the face of new legal challenges. Another is to intervene legislatively only to the extent of removing glaring inequities in existing processes, for example, certain precedural barriers to recovery in both the tort and worker compensation systems.

But these are patchwork solutions. They will not substantially reduce the delays, frustrations and anxieties experienced by most toxic substance victims. They will not significantly mitigate the costs of pursuing compensation claims. Nor will they limit the impact of chance factors which result in adequate compensation for a victim in one jurisdiction while denying all recovery to a similarly situated claimant in another. A continuation of the present arbitrary and uncertain state of affairs, moreover, hurts not only those who are injured, but also those potentially subject to liability. It discourages industry from systematically dealing with compensation issues, since the success rate for victims is, on average, quite low. At the same time, it leaves open the possibility of disastrously high damage awards in an unpredictable minority of cases.

A mechanism explicity designed to deal with the issue of victim compensation appears the only viable response to these problems. In my view, the most promising option considered in the NSF study is the hypothetical collective liability system. It rests on a philosophy reflected in a number of existing federal laws, including the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980. It is also similar in conception to compensation schemes adopted in a number of other countries, for example, West Germany's framework for handling drug-related injuries.

The collective liability proposal presents two attractive features. First, it makes available a fairly low-cost way for victims to claim compensation even when they cannot uniquely identify the manufacturer or producer responsible for their injuries. Through creation of a fund, it forces manufacturers of toxic substances to set aside a regular fraction of profits for purposes of compensation. In these respects, the collective liability approach approximates some of the efficiencies associated with no-fault liability systems, without, however, completely dispensing with the need to prove causation. For victims capable of meeting a higher standard of proof, the proposal also offers a modified version of a fault-based recovery process. In particular, this avenue allows the recovery of punitive damages against identified defendants, thus deterring reckless conduct, but places ceilings on such awards to keep them within reasonable bounds. One may well disagree with the details of the collective liability proposal outlined in the NSF study. It appears to me, however, that a satisfactory solution to the victim compensation problem will have to blend elements of several different approaches, as this proposal seeks to do. This evaluation represents my personal opinion and not that of the National Science Foundation.

This concludes my prepared statement. I would be happy to respond to questions.

Mr. LEVITAS. Thank you very much, Dr. Jasanoff. Most interesting. At least we can take comfort in the fact that others have been plowing this ground before us. We are not the first explorers of these uncharted waters.

Let me start first by trying to clarify something in Dr. Millar's statement. Toward the end of the statement, on page 5, you talk about these cases that you have studied that were originally discussed back in July. Were there any additional cases looked at since July?

Dr. FINGERHUT. No.

Mr. LEVITAS. Of those seven cases that you went into greater detail, only two have histologically verified soft tissue sarcoma for which we have documentation of their employment and the manufacturing processes of the contaminant in question. Now, that does not say to me that those other five might not have also fallen in that same category; is that correct?

Dr. FINGERHUT. The definition that we have used for exposure is a restrictive one. We looked for a record that the person was assigned to making 2,4,5-T or to making trichlorophenol. In general for production workers, one can find their regular assignments. This person worked regularly in the 2,4,5-T department. One would not find temporary assignments. Six of these seven people were production workers. And so we have a record of their regular assignments throughout their career. The seventh person was a maintenance worker.

Many maintenance workers in chemical facilities never have a record of assignment to a particular department, because they work throughout the entire complex. Therefore, the maintenance worker may well have been in the 2,4,5-T area, but he would not have a record of documentation. So we have used a restrictive——

Mr. LEVITAS. So you have a very restrictive universe——

Dr. FINGERHUT. That is right.

Mr. LEVITAS [continuing]. You are really looking at, even though the others might well have had similar exposures. You just don't know.

Dr. FINGERHUT. That is right. There is no documentation of it. We don't know. That is right.

Mr. LEVITAS. Now, even with that restrictive definition, you then say the two verified, and you underline verified, I guess anticipating the question I just asked you, while a small number still represent an excess over the less than one which would be expected statistically. As I understand your testimony, it represents that even that restricted verified number represents almost three times what you would normally anticipate.

Dr. **Fingerhut**. Let me back up just to one point. The verified refers to pathologic confirmation by soft tissue sarcoma experts that they had their pathology of soft tissue sarcoma. We reviewed the work history records and used documentation as I explained earlier. The first four cases, one to four, were originally from small company studies in the United States. Those workers were reported by the companies in studies they had done of their own workers who made 2,4,5-T or trichlorophenol. Of those four we found, yes, they did work in those departments, by looking at their records. And the two independent reviewers of their pathology found that two had soft tissue sarcoma, two did not. So two of those four is still above what we would have——

Mr. **Levitas**. What is, statistically, the magnitude of the excess?

Dr. **Fingerhut**. The NIOSH researchers wrote a letter in 1981 to *Lancet*, a medical journal, reviewing the then existing literature. At that time three of these four individuals were deceased and had been reported as deceased. In the 4 studies put together, there were 105 deaths. The 3 deaths due to soft tissue sarcoma out of 105 was 2.9 percent.

The researchers used national statistics to get an estimate of what percent of deaths you would suspect in the male population, ages 20 to 85, and found that one would expect 0.07 percent of deaths to be due to soft tissue sarcoma.

Mr. **Levitas**. That would, therefore, be about three times what you would expect; that is, what you found is three times what you would expect. If you would expect 0.07 percent and you found 2.9 percent of the total of 105 deaths, that, if my arithmetic is correct, is about 3 times, 4 times.

Dr. **Fingerhut**. It is about 40 times.

Mr. **Levitas**. Forty times.

Dr. **Fingerhut**. Yes, sir.

Dr. **Houk**. Mr. Chairman, could I——

Mr. **Levitas**. I see. OK.

I had the zero in the wrong place.

Dr. **Houk**. Could I respond, Mr. Chairman.

When one is looking for rare events and statistically you suspect 0.1 death or one-half a death or one-third you obviously——

Mr. **Levitas**. What is one-half a death?

Dr. **Houk**. You obviously can't find one-half a death, you can't find one-third of a death, so if you find no deaths, it doesn't make it a negative study, and if you find one, it doesn't make it a positive study. But if you find two, you become a little more concerned. If you find three, heightened concern; if you find four, more heightened concern.

These epidemiologic data do not prove, do not ever demonstrate conclusively cause and effect. We live in a society fortunately that we cannot do those studies. We can't purposely expose people to toxic chemicals to get cause and effect.

I come back to my statement, sir, that what we are trying to do is demonstrate association if it is there; to remove as many confounders as possible, other things that could influence this—so that reasonable policymakers can make reasonable decisions.

Mr. **Levitas**. Right. I accept that. I don't think we expect that the epidemiological data will be able to conclusively establish cause and effect.

But it can provide information and direction, whether it is in this area or other areas. I'm thinking now of the ongoing Framingham, MA study on coronary heart disease, other environmental factors. I don't think anybody would claim what we know from those studies have given us causal relationship, but there has been a lot of behavior modified as a result of those findings.

That is what I am simply trying to get at, the magnitude of the information that we have at this moment.

Dr. **Houk**. I made my statement, Mr. Chairman, because you frequently hear that because we don't have and cannot have cause and effect, we can't make decisions.

Mr. **Levitas**. We hear that frequently. We hear it about acid rain and chloroforocarbons and a whole bunch of other things, but we do have to make decisions based upon the best available information.

Could you discuss just for a moment, Dr. Houk, and possibly Dr. Jasanoff who I think may want to contribute for the record, about the long-term effect of exposure to certain chemicals that don't manifest themselves immediately—you swallow it and get sick type of things—but you are exposed over a period of time, and then you manifest certain symptoms that are commonly associated with long-term exposure to that particular substance.

I think this is an area that a lot of people don't understand. I remember when I was in the State legislature and we were debating mirex. A legislator from one of the rural areas of our State who wanted to kill some more fire ants swallowed some mirex to prove conclusively that it wasn't bad for you, whatever the point was, he was trying to make.

As I understand it, there are certain chronic effects that occur after a long period of time, and not necessarily tomorrow morning.

Dr. **Houk**. There are a few diseases, and I believe you can count them on one or two hands, where a specific chemical causes a specific disease, either acutely or sometime down the pike, vis-a-vis—mesotheliomee-asbestos; angio-sarcoma of the liver-polyvinyl chloride, et cetera. But those are very few. It is my view that the risk of our chemical world and risks of the hazardous materials around us increases the risk of the more common diseases we have, increases risk of hypertension, increases the risk of birth defects, increases the risk of cancer, increases the risk of certain liver diseases by exposure to these various kinds of chemicals. And to ferret that out, how much of an increase on top of something that is relatively common, is going to be very difficult.

With the issue of the long-term effects, of being able to measure this, we really have two problems. One is what is exposure? For most of these chemicals, we do not have laboratory tests that can determine if a person has been exposed or not exposed. We must use records and use those very tightly as best that we can.

In addition to that, in this country we do not have a very good system of following people over the long term. Hence, we are establishing some registries to keep track of those individuals. Then if something occurs with a frequency of, say, 2 in 100, for example, and exposure to a material increases that risk by 1, then you have to have a large number of people followed for a large period of time to be able to demonstrate that increased risk, whether it occurred or did not occur.

And I believe for that reason that science is not going to be able to produce the conclusive answers. We are going to have to make our best judgments. We have, as you say, in the Framingham study, changed a lot of behaviors. We have regarding the lead in our environment, we know we have millions, that is an exaggeration, but at least hundreds of thousands of papers on lead. Lead is bad for you. Lead makes our children a little less intellectually capable than they should be. The major source of lead in this country is gasoline lead, from automobile emissions. And the Government has taken action to limit the emissions of lead from gasoline.

I think also that because of the complex problems that we have, one of our real goals in this issue should be to prevent the exposure in the first place, to really address the issue of chemicals. Should they be added? Is there real reason to add this one? Is there enough economic necessity?

If we have to, if a contaminant is produced in the manufacturing process, can we clean that up at best? Or if chemicals such as polychlorinated biphenyls when they burn through accidents, inadvertent events, produce dioxins. Can we prevent that accident? If we can't prevent that accident, maybe we shouldn't allow it in the first place.

Mr. LEVITAS. Dr. Jasanoff, could you just address the same question regarding the long-term effects and so forth, in the context of your statement, and why it is that persons exposed to 2,4,5-T, victims as I would call them, cannot meet, as I understood you to say, even the modified proof requirements of some of the mechanisms that have been put in place to provide compensation for such victims?

Ms. JASANOFF. Obviously, the time gap between exposure and the appearance of a disease like cancer is not the only thing that makes it difficult for this category of victims to meet the standard of proof. But the way that the time lag affects the case is that during the intervening period where victims don't know that they may 20 years down the road end up with this disease, they have no reason to maintain their own medical records. And they have no reason, particularly, to keep track of other exposure circumstances that could eventually in a court of law become intervening causes, and these intervening causes could undermine the case for tying the effect that shows up many years down the line with an exposure that occurred 15 or 20 years ago.

So both for the recordkeeping reason and for the reason that other causes, particularly when we are talking about ordinary diseases of life, may intervene in that period, it becomes very difficult for claimants to make their case.

Mr. LEVITAS. Many complexities. Obviously, someone who has been exposed to a particular substance and may, as a result, manifest a certain illness later on, can get run over by a car and, therefore, never manifest that illness.

I have just one last question in this round, Dr. Houk, and then I will get back to you after the other members have had a chance. In your testimony you note that based upon inquiries from EPA and FEMA concerning the Times Beach situation, under the circumstances there, CDC made the recommendation that the residents not go back to their homes, not go back to Times Beach.

It would be helpful, I know to me, if you could explain just what is the role that CDC plays in matters of this sort. I am constantly hearing that when a situation develops, whether it is at Love Canal or Stringfellow or Seymour, that everybody says, "We will ask CDC to tell us whether the situation is such that we need to have some action response," whatever the action may be.

What is CDC's role in all of this? What do you do? As I understand, what CDC does in this area is of an epidemiological nature, and not the sort of activity that I think you were called on to play in Times Beach. But I may be wrong.

Dr. Houk. That is the CDC of old. And it would be very, very pleasant if that is what our role was. We have a role under CERCLA, a legislative role, relating very specifically to health studies, and to advise EPA on health issues and to determine if there are health issues or if there are not health issues. It is our role under the CERCLA legislation to do the risk assessment. And it is EPA's role to take that and to do the risk management.

I point out that the classic risk assessment is somewhat less precise than 5-year weather forecasting. When we have to use animal data and try to extrapolate it to human, it doesn't mean it can't be used, but it does mean we have to recognize what it is.

We were very concerned, as in the Times Beach issue, as Mr. Young knows very well, that the dioxin in Times Beach had moved because of the flood and people would go back into their houses. Before that could be measured, we made the recommendations that they stay out.

Based upon animal data and assumptions that being exposed to a level of one part per billion dioxin in a residential setting for a lifetime, from children to adult, results in an excess cancer death of about 1 in 100,000, which is a standard risk assessment thing that we use, we do not think that that is an acceptable risk. If it can be prevented, it should be prevented so that you don't have to be faced with that risk.

Other activities, other authorities that we have to respond to are in the Public Health Service Act, in various situations. In the original Love Canal we were directed by the Congress to become involved. I think that one has to use the epidemiologic process and try to balance the risks as best that one can and arrive at some sort of decision of what is acceptable, and what is not acceptable risk by a definition that people could agree upon, and then try to devise ways to prevent exposure to those particular compounds that are already in our environment from happening so that we do not have to have that risk.

Mr. Levitas. But in the case of Times Beach, as I understand your testimony, CDC made a specific risk management decision, or recommendation; that is, don't move people back into Times Beach. There is a site which you may be aware of that has just been identified in Metropolitan Atlanta, the ABC Chemical Compound Co.

After analysis of the chemicals that were discovered there, EPA determined to make an emergency response and action is commencing this morning.

Does CDC get asked the question, "What should we do, CDC? Should we evacuate the people from the neighborhood? Should we dig wells to find the migration, if any, of the chemicals of the site?" What is CDC's role in that regard?

Dr. HOUK. Our role in this, Mr. Levitas, in the emergency responses, I think it is very clear where those decisions are made. For some of the remedial sites that you are talking about we would have a role in helping design the environment monitoring system, the testing system, to ensure that it adequately represents the areas. We would also have an advisory role, to ensure the adequacy of the information, the quality control of the information, to look at the human health effects, the avenues of exposure, the population around it, whether there are potential toxicities to these exposures; and then to make recommendations to EPA.

The Times Beach decision was early in the process of Superfund. And we would probably not word the Times Beach advisory in the same way of saying tell the people not to move back. We would have worded it and said, "Do not allow the people to be exposed to the potentially contaminated soil." There was only one available solution in our view at that time, since the flood, and that was not to allow them to move back.

Mr. LEVITAS. Mr. Sundquist.

Mr. SUNDQUIST. Thank you, Mr. Chairman.

Mr. LEVITAS. Let me say before you begin. Mr. Sundquist wasn't able to be with us yesterday. But on the previous day he chaired a very impressive conference, the Mid-South Conference, on hazardous waste in Memphis. And, based on the people I have talked to who had the opportunity to participate in that, it was extremely important. I want to compliment you on that.

Mr. SUNDQUIST. Thank you, Mr. Chairman, and thank you for your presence at that conference.

Dr. Houk, let me go back a second. Could you give me the numbers again? You quoted figures of 1 out of 100,000 relating to Times Beach.

Dr. HOUK. That was the risk assessment that we used, and that is a fairly common one that is used on asbestos.

Mr. SUNDQUIST. Tell me what that means is what I am asking. Translate that into layman's terms.

Dr. HOUK. It is very difficult, Mr. Sundquist, to put it in layman's terms. I have tried to do this in public meetings and lectures for course 101A in biostatistics, as you can imagine. What this is, is looking at the avenues of exposure, looking at the total lifetime of someone who would take in a particular compound; then looking at the mechanism and action of carcinogenesis, mutagenesis, or whatever, in the animal studies, making the calculations that this does or will produce this many excess cancer deaths in a population.

It is a technique that is used for making decisions, but it is a very difficult technique to explain to people because over and over, I don't care about all your crazy numbers, but what if I am the 1 in 100,000? It means we do not have adequate human data, this is all we have to go on.

Mr. SUNDQUIST. Are you saying if 100,000 people lived in Times Beach, 1 would come down from—with exposure, 1 more than what normally would occur?

Dr. HOUK. We would expect one extra individual to die of carcinoma, which that was based upon, with a lifetime exposure.

Mr. SUNDQUIST. So we moved everybody out of Times Beach on your recommendation or your risk assessment, or you told them not to go back because of only 1 out of 100,000?

Dr. HOUK. We recommended that people should not continue to be exposed to that amount of dioxin.

Mr. SUNDQUIST. Because of 1 out of 100,000?

Dr. HOUK. Yes.

Mr. SUNDQUIST. Now, is that what you would call an environmental exposure?

Dr. HOUK. Yes.

Mr. SUNDQUIST. How would that differ from someone working with dioxin in an occupational exposure? What are the numbers there?

Dr. HOUK. There are no numbers that I know of. The dioxin is not purposely produced. It is a contaminant of the manufacturing process. The exposure of workers to dioxin is quite different than exposure in the general population. More is likely to come through the skin because of the content of it.

In Times Beach we were talking about the dioxin that was available in the soil, that people would breathe in dust, that people would eat the dust, the children would eat through their normal hand-to-mouth activities.

Mr. SUNDQUIST. Would it be less of a problem, then, at Times Beach than it would be for someone who worked with chemicals that had some exposure to dioxin?

Dr. HOUK. I would expect that the exposure of the occupational workers is probably greater than the general population from the environment.

Mr. SUNDQUIST. What do you recommend for those workers receiving exposure through occupational contact?

Dr. HOUK. We would recommend, and I will let Dr. Fingerhut amplify this, but we would recommend to try to prevent that exposure in the first place. If the exposure had already occurred, we'd recommend to do some studies on that.

Marilyn.

Dr. FINGERHUT. At this time, sir, there is no 2,4,5-T or trichlorophenol production being conducted.

Mr. SUNDQUIST. But there has been.

Dr. FINGERHUT. There has been. It is all terminated now. So currently there wouldn't be any exposure by working with the process.

Mr. SUNDQUIST. Have you done any studies on the numbers of those who worked with it?

Dr. FINGERHUT. The NIOSH dioxin registry has a listing of demographic and work history information for, we think, all U.S. production workers who ever would have made those substances and several others. We have identified about 5,000 workers, production workers, to date. And we anticipate identifying possibly an additional 1,000 by the end of this year. So that would be a cohort of all production workers in the United States who made substances contaminated with dioxin.

It doesn't include people who used the substances. And that number we don't have an estimate on.

Mr. SUNDQUIST. Dr. Houk, that would be 1 death per 100,000. What about other body functions or changes in that individual?

Dr. HOUK. We don't know enough about that, Mr. Sundquist, to be able to make predictions. We did our prediction on the things. I think that in the dioxin issue you have the production workers, now one can say with a great deal of certainty that chloracne is caused by dioxin exposure, that there is good evidence that in the acute effect, there is some abnormality of nerve conduction and neurological problems.

In the short term acute thing, there is pretty good evidence that there is an association with altered immune system, and with very high dosage, probably a very firm association with prophyria cutanea tarda, a very specific prophyria that is mediated through the liver. I point out those are very acute effects.

We know very little about the long-term effects and may never know very much about the chronic long-term effects of these chemicals either one way or the other.

If we are looking at adverse reproductive outcomes, let me give an example. The base line against what we are looking at is that people newly married and practicing unprotected intercourse for a year, about 20 percent fail to conceive. Once pregnancy has been diagnosed, about 20 percent end in miscarriage. If the pregnancy is carried to term, about 3 percent are stillborn. Of those who are born alive, 5 percent have structural birth defects detectable during the first year of life, and about 15 percent of birth defects detectable during the first 15 years of life; adverse reproductive outcomes are a relatively common event in people.

If you are trying to add to that, add a 20 percent increase or a 10 percent increase or 5 percent increase, it is almost an impossible task to verify.

Mr. SUNDQUIST. Two more questions.

Going back just a step, can you outline what poses the greatest threat, greater risk to human health? Are we talking about environmental exposure or are we talking about occupational exposure?

Dr. HOUK. Occupational exposure is an environmental exposure. It is an exposure in the environment of the occupation or the environment of where the individual works. Generally if you relate it to chemicals or materials, exposure in the occupational setting——

Mr. SUNDQUIST. How about saying nonoccupational versus occupational?

Dr. HOUK. OK. Generally chemicals, the occupational exposures are higher I believe. That is generally true. There are instances where the chemicals are taken home on the clothes, affect the children, affect other members of the house, et cetera. The occupational exposure to these chemicals generally is as small as the companies can make it.

I mean there are accidents, they are inadvertent. But it is to the interest of the companies very much to have a good safety record, not to have the accident, not to have the adverse health effects to their workers. In the environment in the nonoccupational environment, generally the exposures are lower.

We don't know, however, what the results of those exposures are. Indeed, we don't know how to measure exposure. We do not have techniques of measuring exposure for most of these chemicals.

Mr. **SUNDQUIST**. You said earlier science can't give us the answers. Is it possible to compensate victims of toxic chemicals?

Dr. **HOUK**. Science cannot give you a firm number of—for each case for cancer of something "X" percentage of them are caused by an exposure to "X." That is impossible.

I do not have opinions on compensation. I don't think that is my job. My job is to try to determine associations, where they exist or don't exist. It is up to the public policymakers and others to grapple with these very difficult questions of public policy.

Mr. **SUNDQUIST**. Dr. Fingerhut, is it possible, the same question, to fairly compensate victims of toxic chemicals?

Dr. **FINGERHUT**. I also, work as a scientist. I don't have an opinion on compensation, sir.

Mr. **SUNDQUIST**. Dr. Jasanoff.

Ms. **JASANOFF**. In certain cases I think it is. Dr. Houk mentioned, for example, the known rare diseases that are uniquely associated with certain kinds of exposures, mesothelioma, asbestos being perhaps the best known example. In those cases, I think there is very little doubt when the victim appears demonstrating that symptom that the causal chain leads back to an identifiable exposure. Those are the easiest cases.

I think one can trace from there a continuum across the whole array of people exposed to toxic substances for sure, but in unknown concentrations and demonstrating effect that become more and more like ordinary risks of life. Obviously as you go along that continuum, the problem of compensation becomes very difficult, and there is a large segment at one end where I would say, no, you cannot compensate these people under any reasonably fair theory of compensation.

Mr. **SUNDQUIST**. Thank you, Mr. Chairman.

Mr. **LEVITAS**. Thank you, Mr. Sundquist.

Dr. Rowland.

Mr. **ROWLAND**. Thank you, Mr. Chairman.

I wish to go back to the seven reported cases we talked about earlier. And five were histologically verified.

Dr. **FINGERHUT**. That is right.

Mr. **ROWLAND**. Two were not. What were those two that were not verified?

Dr. **FINGERHUT**. They were carcinomas, sir.

Mr. **ROWLAND**. Mistaken diagnosis?

Dr. **FINGERHUT**. I believe.

Mr. **ROWLAND**. Is that usual?

Dr. **FINGERHUT**. Soft tissue sarcomas are very rare cancers and according to our consultant experts, they are difficult to diagnose in many instances. There are many, there are several dozens of subtypes, as a matter of fact.

In part, I think because of the rarity, there has been great growth in the understanding and designs of classification and diagnosis of these various subtypes of soft tissue sarcoma. So there has been growth in the area within soft tissue sarcoma.

I can't comment on frequency of diagnoses, because I am not a soft tissue carcinoma expert. I am not a physician.

Mr. **Rowland**. I guess what I am asking is that there may be other instances where the disease might be thought to have been due to one thing when it was due to something else.

Dr. **Fingerhut**. Yes; let me talk just for a minute about these seven cases. I think it is important. As I indicated to the chairman earlier, in 1981, the rough estimate from reviewing three company studies with three deaths due to soft tissue sarcoma in them, suggested that there might be, based on national statistics, a possible 40-fold excess in the finding.

NIOSH indicated that what it would do was to confirm the cases, a basic epidemiologic principle we call confirming the cases, means to find out if the case was soft tissue sarcoma and find out if the people were exposed.

When the letter was written, there were three cases known in the United States. In response to that letter to the medical journal, *Lancet,* in 1981, the other four cases were reported by letter to that medical journal.

Case No. 4 was reported by Dr. Ralph Cook of Dow Chemical Co., who said the person had soft tissue sarcoma and was a member of one of the Dow cohorts that had been reported. But he was alive, so was not included in this mortality study. Now, since that time, that person died. So, of the seven cases, four of the cases belonged to the four studies. Three additional cases were reported by their physicians who had seen them in their offices and so knew they had soft tissue sarcoma and knew they worked at a facility where the herbicides were made, but didn't know necessarily the location of the work.

NIOSH indicated it would perform basic epidemiology, confirm the cases. Did these seven people have exposure? Did these seven people have soft tissure sarcoma? And that is what we have described here in the testimony.

Now, the numbers were small. Originally, three were written about in 1981 in the four company studies. It turns out that of those three, one has pathologically confirmed soft tissue sarcoma, but case No. 4 from those four company studies also has soft tissue sarcoma.

Now, we have a situation, you see, where we still have small numbers. In the 1981 letter it was reported by the companies from death certificate information that three people had soft tissue sarcoma. When you use death certificate information, you can compare the information on the death certificates to information in national statistics because we get our population rates from death certificates.

So, the original comparison of .07 percent of deaths expected was from national statistics which are based on death certificate information.

As a result of this current review which we have done, which involved getting the death certificates, getting the original hospital reports, getting the tissue specimens for these seven people and having the tissue specimens reviews by soft tissue sarcoma experts, our conclusion now is that of the four people who were reported from deaths certificate data by the companies to have exposure and soft tissue sarcomas, we have confirmed that they have exposure, but upon pathologic review our experts say that two have soft tissue sarcoma, not four.

Of the other three, they have soft tissue sarcoma pathology, but their records don't show that they ever worked in the 2,4,5-T, or trichlorophenol.

Now, the situation is, of the four who have exposure and originally were reported as having soft tissue sarcomas from their death certificates, we know there are two pathologically confirmed in that group.

Is that an excess, I think is the question? And what we have concluded is that it seems that two is still more than we would have expected. However, we still have small numbers and the comparison is a little bit rough now, because we have ascertained the cases by pathology review from experts and we are comparing them to death certificate information in national statistics. So we can't make that comparison. It may be apples and oranges. But it may be accurate.

So, our conclusion is that two is a small number. Two seems to be more than we would expect. What must be done is the large study which is underway. And so we are proceeding.

Mr. ROWLAND. Thank you for that explanation. I want to ask something about dioxin and its half-life. I understand it is very photosensitive.

Dr. HOUK. Dioxin, if it is exposed to ultraviolet light, we are talking about 2,3,7,8-TCDD now, the most toxic form, will, as was the overall spraying on the trees, et cetera, its half-life is relatively short.

But when it gets into soil and when it is protected from ultraviolet light its half-life was said, the liturgy was a rather precise 200, and I think 36 days. We know from Missouri that it stays there at least 13 years. And if it is there for a decade, it is probably going to be there for centuries.

That will have to be our assumption since it has not deteriorated in that period of time. I would like to make a point of differentiating some of the past exposures that have happened, and it seems to me a major emphasis we should be placing on preventing future exposures.

Because the past exposures really have gotten us into a very complex, difficult kind of issue. And we may be able to prevent something. And without fixing any blame or even laying blame, I would point out that had the materials from the manufacturing plant, dioxin-containing materials, been appropriately disposed of in Missouri, we would not have the Missouri dioxin problem that we have today.

Mr. ROWLAND. 236 days half-life, would that still constitute, in your opinion, a severe hazard?

Dr. HOUK. We know the half-life of dioxin in soil will be measured in the decades, and probably a lot longer than that. It has shown from the horse arenas in Missouri where it was measured in 1971, and again in 1983, substantially no deterioration in the amount.

Mr. ROWLAND. Did I understand you to say that the decision was made to move the people out of Times Beach after the flood?

Dr. HOUK. Yes, the flood, that led to the decision to move the people out of Times Beach. They were moved out because the town was flooded. It was our recommendation because we were very much afraid, we had samples of dioxin into the hundred parts per billion—200, 300 parts per billion, if I believe correctly, was the highest.

We did not know what happened to the dioxin soil as a result of the flood. We were very much concerned that it had moved into the houses. That was our recommendation then, to EPA, not to have the people come back in. We would not make that recommendation again in those precise words.

The same results would be accomplished. We would probably say keep them from being exposed. And there is only one way you could do that, and that was not allow them back into their houses.

Mr. ROWLAND. Had the flood not occurred, would you have made that recommendation?

Dr. HOUK. Had the flood not occurred, we made the recommendation on Minker-Stout, which has dioxin up to 7, 800 parts per million in the residential area. The recommendation we made on Minker-Stout is that the people not be associated with that dioxin-containing soil.

Mr. ROWLAND. Then you would have made the recommendation anyway.

Dr. HOUK. Yes.

Mr. ROWLAND. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you very much, Dr. Rowland.

Mrs. Johnson.

Mrs. JOHNSON. Thank you very much.

Dr. Houk, on something you just said in your last statement, when you say people being associated with dioxin-related soil, Does living in a house, walking on the sidewalk, associate one's self with that soil?

Dr. HOUK. Yes. if you have a family and there are youngsters playing outside in the dirt, this is a major avenue of exposure of children because they do things children are supposed to do, explore their environment.

Mrs. JOHNSON. I appreciate that, but if you live in such a way, are you saying that you don't come in contact with the soil, that there is no danger?

Dr. HOUK. Yes, I believe that if you can separate the individual from any potential contact with that soil, then that soil does not make a hazard.

Provided that it is not volatile, comes up in the air or move into the water systems.

Mrs. JOHNSON. Then lastly, just to go back to a point that both you and Dr. Fingerhut have discussed in a number of different ways, in talking about the results of this study, and the fact that there were really two verified cases which does appear to be twice the expected experience, nonetheless in a study that is so limited, with such small numbers. Do you take this as an indication that we must do more research, or can you really base actions on this narrow study?

Dr. HOUK. The two verified cases of soft tissue sarcoma occurring in the two people that we know were exposed to those products give us more concern and have given NIOSH the impetus to proceed very rapidly with the 6,000 worker study to look at their causes of death.

I believe that it is hoped all the people will be registered in October or December of this year, and it is hoped the initial mortality study will be out, I think, in a year.

Dr. FINGERHUT. Mid-1985.

Mrs. JOHNSON. You are going to use the same registry?

Dr. HOUK. Yes.

Mrs. JOHNSON. Well, now, the registry apparently has registered all those workers who are known to have been potentially exposed to dioxin. It does refer here to a couple of cases. Then when you looked at them, they actually weren't exposed.

Are you going to do any work beforehand to find out which of these workers were exposed and weren't exposed before you go further?

Dr. FINGERHUT. The seven workers didn't come from the NIOSH registry. There is, I think, a little bit of confusion there.

Mrs. JOHNSON. Oh, all right.

Dr. FINGERHUT. NIOSH began this registry in November 1979 to identify production workers. As a part of this process, careful review of the literature was important. So the researchers were reading very carefully literature that was published.

We began work on the registry in 1979 and in 1980, four company studies were published in which three of the studies, each reported one soft tissue sarcoma. The NIOSH researchers looking at those studies knew that two major Swedish studies in 1978 had indicated there was an excess risk of soft tissue sarcoma.

Here are four U.S. studies, three of which each contained one death. One can never conclude anything from one death. If one puts the numbers together and looks at the whole picture, one sees three deaths out of 105. That seemed to be excessive. In 1981, NIOSH researchers reported that observation to the medical journal, *The Lancet*.

In response to the NIOSH letter, those other four people were reported by others into the medical journal, The Lancet. So what NIOSH has done recently is to find out whether the seven persons really had soft tissue sarcoma and were exposed.

Now, alongside of that effort, NIOSH has been putting together this production worker registry of 6,000 people. Our effort is still in the data collection phase, and we anticipate having identified all U.S. production workers by the end of this year. As we find the individuals, we proceed with a stage called followup to determine their vital status.

The first use of these data will be a mortality study which should be completed in the middle of 1985, which will look at the causes and numbers of deaths in the 6,000 U.S. production workers, and ask the question, are the causes and numbers different from what one would expect, based on the U.S. national statistics?

So, the registry and mortality study are underway at NIOSH. Review of the literature and confirmation of reported soft tissue sarcoma cases in the literature is also an activity. Now, I think the reason for some existing confusion is that the sarcoma cases who are production workers also are in the NIOSH dioxin registry, among the 6,000 workers, if they worked in the processes that we are collecting information on. There has been some confusion in saying that the sarcoma cases came out of the registry. Actually, they didn't.

Mrs. JOHNSON. The three studies you referred to were scientifically accepted?

Dr. FINGERHUT. Yes; these were published in the Journal of Occupational Medicine in 1980, by two companies that make the herbicides. We received the fourth in draft form in 1980, but it has been published recently in the literature.

Mr. LEVITAS. While Mrs. Johnson is framing her next question, I will announce that there is a quorum call in process on the floor which may be followed by a 5-minute vote, if ordered.

So I thought maybe if Mrs. Johnson had one more short question, with hopefully a short answer, we might take that, then recess briefly so we can be recorded, and vote, if necessary, then come back and try to complete this panel of witnesses before we break for lunch.

I know Mr. Young has some interest in the Times Beach aspect of this at the very least.

Mrs. JOHNSON. OK. I hope you can make a brief response. First of all, have you started the registry of the Times Beach people?

Dr. FINGERHUT. The workers at the Verona, MO, plant, where they made the hexachlorophene, are included in our registry. The environmentally exposed people are not.

Dr. HOUK. But there is a registry of the Missouri dioxin folks that is separate.

Mr. LEVITAS. Can I clarify that? Is that a separate registry for the Missouri exposed victims?

Dr. HOUK. It would be a registry for the environmentally exposed people of dioxin, most of them are from Missouri now. It will spread to other States where there is other contamination.

Mrs. JOHNSON. As opposed to worksite exposure?

Dr. HOUK. Yes.

Mrs. JOHNSON. Dr. Jasanoff, two questions. First, do you have any comment on the differences or strengths and weaknesses of the 2-tier compensation approach recommended by the section 301(e) Superfund study group, and the collective liability proposal in the NSF study?

Ms. JASANOFF. I have not followed the section 301(e) proposals in enough detail to be able to give you a quick answer on that.

Mrs. JOHNSON. Would you be interested in taking a look at that and seeing if you have any thoughts on that?

Ms. JASANOFF. Yes.

[The information received from Ms. Jasanoff follows:]

SUPPLEMENTARY STATEMENT OF SHEILA JASANOFF

Both the National Science Foundation (NSF) victim compensation report and the Superfund § 301(e) Study Group proposed a two-tier approach to compensation. Differences between the two proposals can largely be attributed to the timing of the two studies and to their divergent objectives. The NSF report was based on case studies carried out in early 1981. Its primary objective was to evaluate the scientific basis for compensation decisions in relation to existing mechanisms. The § 301(e) study, completed more than a year later, was concerned with making new policy recommendations to supplement available remedies. Accordingly, the proposals in the later study were worked out in considerably greater detail than the collective liability approach outlined in the NSF report. For these reasons, I do not believe an in-depth comparison of proposals in these two reports would be very fruitful.

There are, however, two conceptual differences between the NSF and § 301(e) Study Group proposals which I think are worth highlighting. They relate to the design and accessibility of the Tier Two remedy, and in each case I believe the § 301(e) proposal is the more advantageous. Let me briefly describe the discrepancies and explain my evaluation.

The Superfund Study proposed that the Tier One administrative compensation system should be supplemented by a Tier Two consisting of currently available state tort remedies in the fifty states, modified to remove specific procedural and other barriers to recovery. By contrast, the NSF collective liability scheme made a new federal cause of action available at the second tier as well as the first. In this system, different rebuttable presumptions operating at the two tiers would require the plaintiff to meet a higher burden of proof in second tier actions against named defendants. However, even in such cases, the plaintiff would not need to demonstrate that the defendant was the cause of his injuries in order to recover. Existing tort remedies in most states would not permit recovery under these circumstances. The Superfund Study recognized the need for reform in this area. But instead of proposing a uniform federal rule or rebuttable presumption, it recommended that states should revise their rules so as to hold potential contributors to injury or damage jointly and severally liable, placing on multiple defendants the burden of allocating responsibility among themselves.

Federalizing Tier Two would have the advantage of creating a nationally uniform compensation system. Against this, however, must be set the costs and uncertainties of setting an entirely new remedial process in operation, particularly one involving the interpretation of new legal rules. The cost of compensating victims under a uniform, relaxed standard of proof in Tier Two is especially difficult to predict in advance. Retaining the state tort remedy at the second tier, as proposed in the Superfund Study, would not only prevent sudden dislocations in the number and size of claims, but would permit healthy experimentation in the states with new approaches to the problem of collective liability. Moreover, if a clearly superior approach were to emerge after a period of varying state activity, it would always be possible to amend the federal compensation process to create a uniform Tier Two remedy based on the most successful state model.

The Superfund Study compensation proposal promises to be less costly than the NSF collective liability system in another respect as well. The § 301(e) Study Group was concerned about increasing the administrative costs of victim compensation by simply adding a new remedy to those already available. Members recognized that the entire victim compensation process could become hopelessly unwieldy and expensive if claimants could freely elect to pursue both Tier One and Tier Two remedies. The report proposed at least two types of entry barriers to Tier Two for claimants electing Tier One: either an "exit toll," subjecting the claimant to an assessment of costs for unsuccessfully pursuing Tier Two, or enhanced benefits at Tier One, including recovery for certain kinds of pain and suffering, so as to make this "normally" the exclusive remedy for most claimants. These mechanisms have no analogues in the more rudimentary two-tier process outlined in the NSF study. It seems clear to me, however, that federal victim compensation legislation following the two-tier approach should include some means of alleviating the problem of the add-on remedy identified in the Superfund Study.

This concludes my appraisal of the relative strengths and weaknesses of the NSF and Superfund Study compensation proposals. Thank you again for the opportunity to present my views on this extremely important subject.

Mrs. JOHNSON. Fine. Thank you.

Lastly, Dr. Jasanoff, the report that you discuss recommends compensating victims for their severe stress, in the absence of physical manifestations of injury.

Now, we have found here that the data around the issue of physical manifestation isn't very strong. It seems to me that the issue of stress is one that could be claimed in many, many situations.

And I wonder if you could give me some better understanding of why you would recommend compensation on the basis of severe stress?

Ms. JASANOFF. As I stated at the end of my prepared testimony, what was represented in the testimony was my personal opinion and not that of the National Science Foundation. So I must also disclaim personal credit for everything that shows up in the NSF recommendations, because that was based on three different case studies conducted by three different people.

I don't believe, in fact, that compensation proposals in which people would be compensated for stress alone are a realistic way of going about it, any more than, for example, the use of purely psychological symptoms to base regulatory decisions on was considered appropriate by the Supreme Court following the Three Mile Island incident.

Mrs. JOHNSON. Thank you very much.

Mr. LEVITAS. Thank you very much.

The subcommittee will stand in recess for 15 minutes.

[Recess.]

Mr. LEVITAS. The subcommittee will please come to order.

The Chair recognizes the gentleman from Missouri, Mr. Young.

Mr. YOUNG. Mr. Chairman, I want to thank you and the committee for allowing me to participate this morning. I don't know if Mrs. Johnson had a chance at the end of that question, to Dr. Houk I guess it was, about the psychological effects.

I know in your statement, Doctor, that was not a part of your study, isn't that right?

Dr. HOUK. Yes, sir.

Mr. YOUNG. Why was that left out? I am trying to remember back to the middle of February.

Dr. HOUK. The study was done so rapidly in response to the concerns of the citizens there, that it was the judgment of ourselves and everybody that we called in, that the instruments of the study were not sufficiently valid and therefore any results obtained and disseminated would probably be harmful.

We are grappling with that issue, Mr. Young, for the future, if some instruments can be designed to be able to measure that.

Mr. YOUNG. Just one question I have, Doctor. We have gone through this. You briefed me on October 16 of this year. As you had indicated earlier, the CDC had warned the State of Missouri back over 10 years ago, I think you said 1971 or 1972, that dioxin was the cause of all the deaths of the horses and other farm animals that died in a very short time period.

Why did not, or did CDC then realize that the health studies on humans should have been immediately started so that here we could be maybe 10 years down the road a little better involved on the search and data that could have been gathered over that time?

Dr. HOUK. When the original study of the Shenandoah Stables and stables in Missouri where all the horses died, and the chickens and the little girl got sick in 1971, we knew that the material in that soil was some of the most toxic that we had seen.

We did not know what it was. In fact, it was not until 1975 that further studies at that time demonstrated that the material was dioxin. We made a recommendation then that the sludge in that tank be appropriately disposed of and appropriately burned.

As you probably remember, Mr. Young, there is a great deal of difficulty getting permits across State lines for that to go where the incinerations were, and people not wanting it passing through in their State.

Why that did not happen, I don't know. I doubt, sir, whether we would have recommended at that time a health study, because then in—the liturgy was that the half-life of dioxin in soil was less than a year.

And so, it had been placed there in 1971. Then, it was 1975 when we found out what it was. So, therefore, it had been reduced and it was probably not important to go back and do that.

The other thing I think, and this also partially addresses some of the chairman's concerns, that the environmental health issues in the Center for Disease Control are relatively new.

The Center for Environmental Health was established, I believe, in 1980. There were some little activities going before that, but not major, great activities.

Mr. YOUNG. At that time, then, in 1975, I didn't realize that, Doctor, was the organization aware of Mr. Bliss' activities?

Dr. HOUK. No, sir.

Mr. YOUNG. Fine. Thank you very much.

Thank you very much, Mr. Chairman. I appreciate that.

Mr. LEVITAS. Thank you, Mr. Young. I appreciate your continuing interest in this. Dr. Houk, you were talking in answer to Mr. Sundquist's questions about the use of 1 death per 100,000 as a threshold level for unacceptable risk. Are there different levels of risk per 100,000, or whatever number, for different types of activity, or different types of risks that people incur?

For example, 1 death per 100,000 in the case of exposure to a chemical is one we have talked about. Would that be the same level for driving across town in your automobile, or having a heart bypass operation or smoking cigarettes?

Dr. HOUK. By no means, Mr. Chairman. The risk that some of us unfortunate ones continue today, like smoking cigarettes, is probably one of the greatest risks to our long-term health, not only for bronchogenic carcinoma, but for heart disease, chronic lung disease, a whole host of different things.

The risk of driving, of being injured in event of an automobile collision, if you don't wear your seatbelts, you markedly increase that risk. You can eliminate the risk of—essentially eliminate the risk of toddler deaths in automobiles by having all the toddlers and the young kids in the appropriate child restraint device.

These sorts of risks are voluntary risks that people take. As I travel the country and deal with these issues, the unacceptable risk to the American population is the risk that is imposed upon them by somebody else.

Mr. LEVITAS. The study that you referred to, Dr. Fingerhut, or that Dr. Millar had referred to in his testimony, concerning the lumberjacks, where was that reported in the literature?

Dr. FINGERHUT. They were reported in Swedish journals and also in the Scandinavian journal, Work Environment. I can give you the references. I have them in my briefcase. They are usually published in Swedish and then again in an English journal.

Mr. LEVITAS. I recall, and I haven't been able to put my finger on it yet. I am going to ask staff to look for it in Dr. Karrh's testimony. He testified at our last series of hearings. He is with the Du-Point Co. and was testifying on behalf of the Chemical Manufacturers Association.

I am going to ask some questions about that testimony. But there was one particular point that he made, as I recall it, that there were no reported scientific medical studies of injury from occupational exposure to dioxin.

I believe that was the substance of what he said.

Dr. HOUK. The statement is incorrect. Let me be as blunt as I can. In the acute exposures, there is association of dioxin and chloracne. Chloracne is something other than just childhood acne.

In the Seveso incident, which people keep referring to as chronic exposure, it is not. It is acute exposure. Numbers of those children had abnormalities of their nerve conduits. Numbers of those children had problems with logic when they were tested.

In the adult population of workers in this country, there have been problems identified of immunologic difficulties. And there have been in the acute exposure and for a short term afterwards, and there certainly is a very strong association with prophyria cutanea tarda.

One of the difficulties, Mr. Levitas, when we talk about dioxin, and I don't want a dioxin hearing, is that there are 75 compounds that are generally referred to as dioxin. And these vary in toxicity from almost nothing to among the most toxic compounds that we know in this country.

It is also said that you produce dioxins when you barbecue your steak in your backyard, which is a true statement. But you produce the kind that aren't toxic. You don't produce 2,3,7,8-TCDD. I think that statement from the DuPont witness is in error. I have said so before other committees.

Mr. LEVITAS. Now, in this Swedish lumberjack case, there was evidence, I gather, from what I read, that exposure over a period of time by these lumberjacks to this material apparently did result in some associated illnesses.

Is that a fair statement?

Dr. FINGERHUT. There were actually three occurrences. In about 1977, in a clinic where lumberjacks were treated, it was noted by the physicians there that there were several cases of soft tissue sarcoma.

That was surprising because it is a rare cancer. That stimulated the conduct of two separate full-scale epidemiologic studies. The design of those studies was different from the ones we have talked about so far, so let me just explain them.

It is called the Case Control Study. Each of them had a similar design. One was in northern Sweden where there were many forests, the other in southern Sweden where there is agriculture.

The design is to use the cancer registry to select male cases of sarcoma recorded in the registry between 1970 and 1977. Those were the cases. The researchers then used the national population registry and selected three controls for each case, who were matched by age, sex, race, and location.

In the first study, there were 50 cases and 150 controls. In the second study, there were 100 cases and two controls for each person. What they then did was to use a questionnaire approach, which inquired about all facets of the person's activities and work exposure and environmental exposure and so on and so forth.

Using epidemiologic methods of analysis, what they learned and what they reported was that people who had prior exposure to phenoxy acids and chlorophenyls had a five-fold increased risk of getting sarcoma, as compared to people who didn't have prior exposure to phenoxy acids or herbicides. The information on phenoxy acids was obtained by asking people, did you use this, where did you work, et cetera.

The two studies are respected epidemiologic studies. They have been critiqued by many peers in part because of the EPA hearings on 2,4,5-T in 1978. There is a range of opinions from one extreme to the other, regarding the studies but in general, they are considered to be solid epidemiologic studies, with flaws, which all studies have.

Mr. **Levitas**. When EPA banned the spraying of some herbicide out in the Northwest, did CDC or NIOSH participate in the science that led to that decision?

Dr. **Fingerhut**. I was not with NIOSH then, sir. I can't speak to that.

Dr. **Houk**. I do not believe so. That is 2,4,5-T early in 1975, something like that. Perhaps 1977.

Mr. **Levitas**. I recall that Dr. Karrh also testified that he would have no hesitancy about moving himself and his family and children into Times Beach. Would you like to be a neighbor of Dr. Karrh at Times Beach?

Dr. **Houk**. I would not under any circumstance conceive and raise a family in the conditions of many of the sites that there are at Missouri. This is the difficulty we face in Missouri. And Mr. Young knows this as well as I do. Both of us have discussed this.

The poor people in Missouri don't know what to think. You have one group coming in, saying you can eat the stuff and it has never caused any harm to anybody. You have another group coming in saying, if you are within 1,000 miles of one molecule, you are going to die tomorrow.

Obviously, the truth lies somewhere in between. We can prevent, we can prevent exposure if we handle the soil in Missouri correctly. We can prevent the future availability of dioxin-contaminated products if we no longer allow the manufacture of products that can do that.

We still may have some, through inadvertent fires, polychloride biphenyls and a few products like that that are going to be around.

One of the issues is to prevent exposure of people. As you know, we are embarking on the study of agent orange for the Vietnam veterans. And people try to relate these two issues and they are not relatable. We cannot prevent the exposure to agent orange that happened 10 years ago or 15 years ago in Vietnam.

We can; however, prevent future exposure to people living in dioxin-containing soil, in future industrial accidents, these sorts of things.

Mr. LEVITAS. I just have two final questions.

Does it make any difference if exposure to a toxic chemical occurs in a workplace, or in a neighborhood that is adjacent to a toxic waste dump?

Dr. HOUK. As to the disease outcome? No.

Mr. LEVITAS. And with respect to the Seveso data, has CDC done any analysis of it?

Dr. HOUK. We have been trying our best over several years, both NIOSH and ourselves, very unsuccessfully to get the raw data to do an analysis of those results. There is a very serious question as to the validity of conclusions when data are unavailable for examination. It is better the way we do things, as in the Missouri dioxin study, that as soon as all data are arrayed we will make public access tapes so any scientist can have that data and analyze it in any way the scientist wants to.

One of the problems of Seveso that we have is the number of dead animals that were brought in assumed to be contaminated. The fact is people were paid, residents were paid to bring in dead animals. No one ever analyzed those animals for body content of dioxin, so we have no knowledge of how many of those animals were killed. We do know that the people living now, the soil in Seveso is contaminated with dioxin at 0.15 parts per billion, which is 7 times lower than our action level we established for Missouri. We do know that, I think 40, or an *x* number of drums, I will not say how many of them from that accident, turned up in France in some butcher's backyard shed that have now been found and put back into the appropriate system. I think the primary basis for our concern is data from the animal models indicating that these compounds do cause harm.

Frequently people will give a very nice liturgy of the animal toxicological data and then follow by saying but that has not been shown to be true in humans, technically an accurate statement but a very misleading statement because the truth of it is, the adequately designed studies in humans have not been done. For various complex reasons they may never be able to be done. Then you have to rely on the animal data and the inadvertent exposures in industry or outside of industry.

Mr. LEVITAS. I remember several years ago there was a substance known as kepone. Was that a dioxin-type compound?

Dr. HOUK. Kepone is a pesticide and I would have to submit information on that for the record. I do not know all of the toxicity to kepone off the top of my head, but it is not a dioxin compound.

[The information received from Dr. Houk follows:]

Kepone is a persistent chlorinated aromatic pesticide of moderate acute toxicity. In humans, the following toxic effects have been observed: tremors, rotating eye movements, and reversible effects on male reproduction. In rodents, it has been shown to be a carcinogen and to affect reproduction. Kepone is entirely chemically and toxicologically unrelated to the dioxins. The only similarity is its lipid soluability and the fact that it is preferentially stored in adipose tissue and liver.

Mr. LEVITAS. I am going to——

Dr. HOUK. It is not a safe compound.

Mr. LEVITAS. I am going to ask you to work with our staff. I would like some additional information about Seveso. It may be that we, this committee, the Congress, working through the State

Department, might be able to get some additional information. I am very interested in pursuing that situation. So I would like for you and our staff to get together on that. I do not have any further questions of the panel at this time, except let me say that I want to express my appreciation to all three of you for your being here today. You have answered a number of questions that I felt needed to be addressed from experts such as yourself. I would like to, on behalf of myself and the rest of the subcommittee, express my appreciation to you. Thank you very much.

The subcommittee will be in recess until 1:45 p.m.

[Whereupon, at 12:35 p.m., the subcommittee recessed, to reconvene at 1:45 p.m., the same day.]

Mr. LEVITAS. The subcommittee will please come to order.

This afternoon we have a panel of witnesses to address some of the concerns that industry has with respect to this issue. It has been my purpose to seek input from every possible source of every interested group and person. And I would like to join in the remarks that Congresswoman Johnson made this morning. I am especially grateful for the participation this afternoon of Mr. Browning and Mr. Kusch, because we need to get the particular expertise that you will bring to us from your perspective.

The witnesses this afternoon are Jackson B. Browning, director of Health, Safety and Environmental Affairs, Union Carbide Corp., and Albert Kusch—am I pronouncing that correct?

Mr. KUSCH. Kusch.

Mr. LEVITAS. Albert Kusch, vice president of Atlantic Richfield Co., senior vice president for manufacturing, research and engineering. Mr. Kusch is accompanied by Gary E. Kovall, manager, environmental, legislative, and regulatory affairs, and Joan DiNal—is that pronounced correctly—DiNal, senior counsel, environmental affairs, of Atlantic Richfield Petroleum Products Co.

We welcome all of you here today and look forward to your testimony. Why don't we begin, Mr. Browning, with you and then we will hear from Mr. Kusch?

TESTIMONY OF JACKSON B. BROWNING, DIRECTOR, HEALTH, SAFETY AND ENVIRONMENTAL AFFAIRS, UNION CARBIDE CORP., DANBURY, CT; ALBERT W. KUSCH, VICE PRESIDENT, ATLANTIC RICHFIELD CO., SENIOR VICE PRESIDENT FOR MANUFACTURING, RESEARCH AND ENGINEERING, ARCO PETROLEUM PRODUCTS CO., LOS ANGELES, CA, ACCOMPANIED BY GARY E. KOVALL, MANAGER, ENVIRONMENTAL, LEGISLATIVE, AND REGULATORY AFFAIRS, ARCO PETROLEUM PRODUCTS CO.; AND JOAN DiNAL, SENIOR COUNSEL, ENVIRONMENTAL AFFAIRS, ARCO PETROLEUM PRODUCTS CO.

Mr. BROWNING. Thank you, Mr. Chairman.

Union Carbide is a diversified manufacturer of chemicals and plastics, consumer goods, industrial gases, carbon products, and many other specialty products.

I appreciate the invitation to testify before the subcommittee on the issue of compensating people injured by exposure to hazardous wastes. This is one of a number of important issues before the Congress which we at Union Carbide are following through corporate policy groups as well as through trade associations.

Our view of the potential health effects and compensation issues associated with exposure to hazardous wastes continues to evolve. These are unusually complex scientific, legal and policy issues for which there are no easy answers. On some points, however, our position is clear.

We support the ongoing efforts under the Superfund law to undertake promptly appropriate cleanup of hazardous waste sites. And we are willing to pay our fair share of the cleanup costs at those sites at which we disposed of hazardous substances. We take this position even though our actions at those sites were not in violation of laws in effect at that time. Timely and effective cleanup efforts serve to protect the environment as well as help to minimize any risk of exposure of the public to potentially harmful substances.

We are committed to minimizing the risk of exposure to these wastes in other ways as well. We currently recycle, reuse or reclaim over 80 percent of the hazardous wastes we generate annually. We are continually seeking new ways to further reduce waste disposal through process improvements, increased recycling, and so on. We also believe additional economic incentives can be used to further the goal of source reduction and encourage alternatives to land disposal.

Most important would be replacing the current feedstock tax under Superfund with a waste-end tax. Such a tax on wastes disposed of, rather than on chemical feedstocks, would, we believe, provide direct economic incentives to complement other environmental statutes which encourage resource conversation and minimize waste generation and disposal. We strongly support such a change to the Superfund law.

We are committed to the proposition that anyone injured by exposure to hazardous wastes should be justly compensated by those responsible.

A key question is defining in scientific terms the health problems associated with exposure to hazardous wastes. There has not been epidemiological or other scientific evidence which supports the need for broad compensation legislation for hazardous waste exposure. Nor have large numbers of injured parties been identified.

The number and nature of injuries are important considerations in determining the need for and characteristics of a new system of compensation. Consideration of existing remedies is also important. In several instances where injuries have been claimed, the tort system has provided compensation. It is imperative that Congress determine that there is a verifiable need for legislation before it takes any action.

With the need for hard facts so critical, we do see the public and congressional debate on the issue serving an important purpose. Through the efforts of many groups, including the scientific community, the administration, the chemical industry, environmental groups, the legal progress, Congress, and many others, we look forward to some better insights in the coming year.

You have heard from previous witnesses regarding efforts to review the available body of scientific information and the data available from known exposure incidents to provide a sound basis for any major legislative action. The Universities Associated for Research and Education in Pathology, or UAREP, study commissioned by the Chemical Manufacturers Association, for example, will be a comprehensive review of available scientific literature to provide better guidance as to the nature and extent of possible health effects associated with exposure from waste sites. The results of the study shall be available next summer.

The Department of Health and Human Services, through its Agency for Toxic Substances and Disease Registry, has agreed to the terms of a court order and will provide needed information in the next year as follows: Information on methods of reporting of diseases and for establishing appropriate registries of persons exposed to toxic substances through environmental exposure; information on health effects of toxic substances organized in a readily accessible data base for all to use; and a list of areas closed to the public or otherwise restricted in use because of toxic contamination.

Efforts such as these will make policy decisions better informed.

While we await information on the extent and nature of the health problems associated with hazardous waste sites which may show the need for compensation legislation, we at Union Carbide continue to look broadly at the policy and legal issues from the two different perspectives. First, what are the key policy and legal issues which we must consider in developing a corporate position, but which cannot be decided until the scientific community better determines if there are health problems associated with hazardous waste sites? Second, what are the key policy and legal issues which should be considered and addressed independent of the extent and nature of the problem?

Let me begin by first highlighting some of the most important of those policy elements which are tied to these scientific questions which we are looking at. Our view is that the present tort system will probably continue for some time to be the principal means for handling claims arising from exposure to hazardous wastes.

We do see a need within that system to improve the basis for making decisions on the critical issues of causation. One recently suggested approach which we are exploring and which is promising is the use of independent expert witnesses to assist the courts on the complex questions of causation.

The traditional authority to call upon expert witnesses could be formalized, for example, to establish a body of scientific experts knowledgeable on the hazards of a particular substance or evaluating site-related exposures. Kept current, this would assure court access to current scientific knowledge. We are not suggesting the outside experts replace witnesses called by the parties involved but rather provide an independent and impartial source of expert testimony.

Exclusivity of compensation, is another very important issue we are looking at. At some point it may be determined that there is a public health problem arising from hazardous waste sites that is sufficiently large and complex to warrant some sort of new compensation system. At that time we will have to come to grips with the question of exclusivity and the continued role of the tort system. It seems to us at that point a decision must be made on the compensation that is to be provided for various injuries and how it is to be provided. In a given situation a potential claimant with injuries arising from hazardous waste exposure should be able to obtain reasonable recovery in a reasonable period of time. However, the compensation for that claimant should be an exclusive remedy.

On the second key point—policy issues to be addressed independent of an understanding of the health problems—there are at least two which we believe should be considered at this time.

The first of these deals with the question of statutes of limitations. Virtually all States have adopted a statute of limitation for tort claims which ensures an opportunity to consider claims for injuries manifested long after initial exposure by requiring only that such claims be filed within a certain period. However, a few States do not base their statutes of limitation on the discovery of injury. We believe this is an unwarranted barrier to recovering for injuries resulting from exposure to hazardous waste. However, we also believe that efforts to remove this barrier should be left for the few State legislatures involved.

The other policy issue to be addressed at this point is minimizing generation and disposal of hazardous wastes, which I have previously discussed. In this regard, again, we believe action by the Congress to replace the Superfund feedstock tax with a waste-end tax is particularly important.

Both of these issues can and should be addressed independent of the extent of health problems tied to hazardous waste. The actions we propose are constructive steps we are prepared to support at this time. We will continue to evaluate the other policy elements associated with hazardous wastes which we believe are so dependent upon the answers from the scientific community.

Thank you for this opportunity to testify. I would welcome your questions.

Mr. LEVITAS. Thank you very much. We will have some questions.

Mr. BROWNING. Very good.

Mr. LEVITAS. Mr. Kusch, if you would.

Mr. KUSCH. Thank you, Mr. Chairman.

My comments today will closely parallel those presented on June 29 of this year before the Subcommittee on Commerce, Transportation and Tourism of the House Energy and Commerce Committee.

As you have already indicated, I have brought with me two colleagues today, Joan DiNal, senior counsel, environmental affairs, and Gary Kovall, manager of environmental legislation and regulatory affairs for ARCO Petroleum Products Co., to aid me in addressing any questions you may have.

I have been associated with the petroleum industry since 1941, and have been privileged to have been party to the tremendous technical and economical changes to our industry which have taken place during that time. We, and other industries, are now entering another era of change, of which the subject of today's discussion is a potentially significant part.

Atlantic Richfield Co. has for some time had under consideration the issue of compensation for exposures to hazardous substances.

We have attempted to develop a corporate position responsive to several key points. However, due to the complexity of this issue, we have not yet reached a conclusion on determining a company position on all aspects of this issue. Our efforts to date have identified several important considerations with respect to the need for victims compensation legislation.

First, we recognize that there is a public perception that environmental exposures to toxic substances present a significant human health risk. Second, as a cardinal principle, we believe that injured persons should be fairly compensated by those responsible for the harm. Third, any legislatively established compensation system must include clear guidelines upon which industry can rely and plan its activities. Fourth, we find that the dimensions of the problem of environmental disease are poorly defined. This leads us to conclude that passage of legislation to compensate persons for non-work place exposures to hazardous substances is premature.

Further, it is difficult to identify causation and to create a compensatory scheme which isolates the cause of disease and allocates responsibility for harm.

There may be instances where toxicological and corroborating epidemiological evidence establishes a reasonable link between exposure and disease. If that link is established, compensation is warranted. We do not believe, however, that any existing legislative vehicle creates an appropriate or workable scheme for distinguishing legitimate from spurious claims for compensation.

In addition, federally mandated studies—such as those called for under section 301(e) and 104(i) of CERCLA—have either not contributed to our medical understanding of environmental disease or are as yet incomplete. Devising legislation—or a company position—in an emotionally charged atmosphere and without adequate factual underpinnings is fraught with difficulty and peril.

We recognize, however, that Congress has begun consideration of systems to compensate persons who may be harmed by exposure to hazardous substances. Therefore, we believe that several key aspects of victim compensation legislation currently under consideration by the Congress deserve discussion at this time.

First, an observation is necessary regarding whether a Federal administrative compensation scheme should be an exclusive remedy. The legislative proposals now under consideration in Congress would not require that the claimants seeking compensation elect one remedy. We view this as a serious defect. Claimants should be required to make a binding election between litigation and an administrative remedy. Election of an administrative remedy should foreclose further judicial remedies. Otherwise, litigation and its attendant costs to society will surely mushroom.

Second, from industry's point of view, Congress should seriously consider the current problem of inconsistent State laws governing compensation. We live in a complex and litigious society. Business is faced with increasing litigation over exposure to toxic substances both within and outside of the work place. Judicially fashioned remedies are unpredictable and costly. This evolving system holds no prospect of providing in the near future clear guidelines upon which industry can rely and plan its activities. If compensation legislation is enacted, it should provide business with a legitimate degree of certainty in knowing by what rules it will play the game.

Several proposals would create a new Federal cause of action. Instead of creating one more remedy to choose from, Congress should consider a separate preemptive Federal statute to assure consistent and equitable standards for all claimants who elect to litigate. Such standards should be based upon actual causation and not upon unproven assumptions.

Third, if Congress determines that a compensation fund is a desirable part of compensation legislation, subrogation provisions pose difficult issues. Subrogation preserves the fund and penalizes undesirable conduct. However, subrogation also continues litigation and elevates transaction costs that any compensation program should be designed to eliminate.

However, if subrogation rights are eliminated, we firmly believe that the legislative scheme should offer strong incentives to eliminate improper conduct and reward those whose conduct is exemplary. For example, criminal penalties could be imposed on certain types of conduct which result in claims against the fund.

Fourth, the method of funding any Federal administrative compensation scheme deserves careful attention. Several proposals have adopted the CERCLA "feedstock" tax approach to financing administrative compensation. This would be totally inappropriate and grossly inequitable. A tax on petroleum and chemical feedstocks bears no logical relationship to the broad-based problem this legislation is designed to address.

Additionally, such an approach would place the burden of taxation on a few companies rather than on a broader segment of commercial, industrial, governmental and community activities which may involve human exposures to hazardous substances. We urge you not to perpetuate the inequities in the current CERCLA tax system. If Congress decides to establish a program to compensate persons exposed to hazardous substances, we urge you to develop a financing mechanism which taxes all entities who are identified as being significantly involved in activities utilizing hazardous substances, and which taxes all such entities in a manner reasonably commensurate with their proportionate contribution to the total problem. Moreover, such a program must be designed to achieve administrative simplicity.

Atlantic Richfield Co. fully recognizes the difficult challenges before you on these compensation issues. We offer our assistance in developing a technically sound and equitable solution to these issues. We have not touched upon all of the troublesome mechanical and legal problems presented by this type of legislation. We look forward to providing the committee with additional specific views as consideration of this issue progresses both in the Congress and in our company.

I want to thank the members of this committee for giving me the opportunity to present our views today.

[The statement of Mr. Kusch follows:]

STATEMENT OF ALBERT W. KUSCH, VICE PRESIDENT, ATLANTIC RICHFIELD CO.

VICTIM COMPENSATION LEGISLATION

Good morning. My name is Al Kusch. I am Vice President of Atlantic Richfield Company and Senior Vice President for Manufacturing, Research, and Engineering of the ARCO Petroleum Products Company, a division of Atlantic Richfield Company.

My comments today will closely parallel those presented on June 29 of this year before the Subcommittee on Commerce, Transportation and Tourism of the House Energy and Commerce Committee.

I have brought with me today Joan DiNal, Senior Counsel Environmental Affairs and Gary Kovall, Manager of Environmental Legislation and Regulatory Affairs for ARCO Petroleum Products Company to aid me in addressing any questions you may have.

I have been associated with the petroleum industry since 1941, and have been privileged to have been party to the tremendous technical and economical changes to our industry which have taken place during that time. We and other industries are now entering another era of change, of which the subject of today's discussion is a potentially significant part.

Atlantic Richfield Company has for some time had under consideration the issue of compensation for exposures to hazardous substances. We have attempted to develop a corporate position responsive to several key points. However, due to the complexity of this issue, we have not yet reached a conclusion on determining a Company position on all aspects of this issue. Our efforts to date have identified several important considerations with respect to the need for victims compensation legislation. First, we recognize that there is a public perception that environmental exposures to toxic substances present a significant human health risk. Second, as a cardinal principle, we believe that injured persons should be fairly compensated by those responsible for the harm. Third, any legislatively established compensation system must include clear guidelines upon which industry can rely and plan its activities. Fourth, we find that the dimensions of the problem of environmental disease are poorly defined. This leads us to conclude that passage of legislation to compensate persons for non-work place exposures to hazardous substances is premature.

Two recent studies suggest the potentially limited scope of environmental disease. These studies—one by the Office of Technology Assessment and the other reported in the Journal of the National Cancer Institute—estimate that 5 to 7 percent of cancer deaths are related to three factors: occupation, pollution and industrial products. These same reports relate 25 to 40 percent of cancer deaths to tobacco and 10 to 70 percent to dietary habits.

Further, it is difficult to identify causation and to create a compensatory scheme which isolates the cause of disease and allocates responsibility for harm.

There may be instances were toxicological and corroborating epidemiological evidence establishes a reasonable link between exposure and disease. If that link is established, compensation is warranted. We do not believe, however, that any existing legislative vehicle creates an appropriate or workable scheme for distinguishing legitimate from spurious claims for compensation. In addition, federally mandated studies—such as those called for under Section 301(e) and 104(i) of CERCLA—have either not contributed to our medical understanding of environmental disease or are as yet incomplete. Devising legislation—or a Company position—in an emotionally charged atmosphere and without adequate factual underpinnings is fraught with difficulty and peril.

We recognize, however, that Congress has begun consideration of systems to compensate persons who may be harmed by exposure to hazardous substances. Therefore, we believe that several key aspects of victim compensation legislation currently under consideration by the Congress deserve discussion at this time.

First, an observation is necessary regarding whether a federal administrative compensation scheme should be an exclusive remedy. The legislative proposals now under consideration in Congress would not require that the claimants seeking compensation elect one remedy. We view this as a serious defect. Claimants should be required to make a binding election between litigation and an administrative remedy. Election of an administrative remedy should foreclose further judicial remedies. Otherwise, litigation and its attendant costs to society will surely mushroom.

Second, from industry's point of view, Congress should seriously consider the current problem of inconsistent state laws governing compensation. We live in a complex and litigious society. Business is faced with increasing litigation over exposure to toxic substances both within and outside of the work place. Judicially fashioned remedies are unpredictable and costly. This evolving system holds no prospect of providing in the near future clear guidelines upon which industry can rely and plan its activities. If compensation legislation is enacted, it should provide business with a legitimate degree of certainty in knowing by what rules it will play the game.

Several proposals would create a new federal cause of action. Instead of creating one more remedy to choose from, Congress should consider a separate pre-emptive federal statute to assure consistent and equitable standards for all claimants who elect to litigate. Such standards should be based upon actual causation and not upon unproven assumptions.

Third, if Congress determines that a compensation fund is a desirable part of compensation legislation, subrogation provisions pose difficult issues. Subrogation preserves the fund and penalizes undesirable conduct. However, subrogation also continues litigation and elevates transaction cost that any compensation program should be designed to eliminate. However, if subrogation rights are eliminated, we firmly believe that the legislative scheme should offer strong incentives to eliminate improper conduct and reward those whose conduct is exemplary. For example, criminal penalties could be imposed on certain types of conduct which result in claims against the fund.

Fourth, the method of funding any Federal administrative compensation scheme deserves careful attention. Several proposals have adopted the CERCLA "feedstock" tax approach to financing administrative compensation. This would be totally inappropriate and grossly inequitable. A tax on petroleum and chemical feedstocks bears no logical relationship to the broad based problem this legislation is designed to address. Additionally, such an approach would place the burden of taxation on a few companies rather than on a broader segment of commercial, industrial, governmental and community activities which may involve human exposures to hazardous substances. We urge you not to perpetuate the inequities in the current CERCLA tax system. If Congress decides to establish a program to compensate persons exposed to hazardous substances, we urge you to develop a financing mechanism which taxes all entities who are identified as being significantly involved in activities utilizing hazardous substances, and which taxes all such entities in a manner reasonably commensurate with their proportionate contribution to the total problem. Moreover, such a program must be designed to achieve administrative simplicity.

Atlantic Richfield Company full recognizes the difficult challenges before you on these compensation issues. We offer our assistance in developing a technically sound and equitable solution to these issues. We have not touched upon all of the troublesome mechanical and legal problems presented by this type of legislation. We look forward to providing the committee with additional specific views as consideration of this issue progresses both in the Congress and in our Company. I want to thank the members of this committee for giving me the opportunity to present our views today.

Mr. LEVITAS. Thank you very much, Mr. Kusch. Let me start by asking you something that you said in your testimony. Maybe you could provide a little elaboration. You talked about the need for a degree of certainty in the event legislation is enacted on victim compensation, so that there must be clear guidelines, I think you said.

Mr. KUSCH. Yes.

Mr. LEVITAS. And business needs to plan its activities. Could you give me some further understanding of what you have reference to? What is the problem that you foresee, and what is it that you mean by degree of certainty in this area?

Mr. KUSCH. May I defer to my legal arm over here?

Mr. LEVITAS. Sure.

Mr. KOVALL. Mr. Chairman, what we are referring to really is specifically the two-tiered type of approach that was recommended in the CERCLA 301(e) study. The fact that because there is no election of remedies, and because there is no specific type of Federal statute for the cause of action specified in that report, that we are concerned that we are going to see a rather dramatic evolution of tort law. Hand in hand with any compensation scheme, this evolution begins to break down traditional rules of causation which we think any compensation scheme ultimately is going to have to do.

And we are concerned that that—we are sort of faced with that situation today. The courts have been very creative in this country in fashioning new remedies for plaintiffs in so-called toxic tort litigation. I think we are right at the very beginning stages of that evolution. We are concerned if Congress tackles this issue and does not tackle the issue of specific liability rules, limitation, reasonable and ensurable defenses so we can predict our conduct and ensure our conduct, that we are going to lose total control of any type of compensation. It will end up being another black lung type of program. We are very concerned about that.

Mr. **Levitas**. Both of you advocated a waste-end based tax to fund Superfund in general; and if not in general, certainly then in any additional areas that would be added on. Obviously there is some secondary benefit from a waste-end tax in that it would encourage recycling and other types of methods as alternatives to land disposal.

But we have heard some concerns about a waste-end based tax approach, particularly in terms of its regulatory and cost implications, both to the Government and to industry. I wonder if both of you would let me have your thoughts on that aspect of it, recognizing, as I have already said, that there is certainly incentive built into a waste-end tax to achieve another purpose, which is to reduce reliance on land disposal.

And there is also, I think both of you indicated, a fairness issue that would be addressed by the waste-end tax. But what about the regulatory and cost implications?

Mr. **Browning**. Worth noting I guess that I met Mr. Kusch this morning, so I had no idea of which side of that issue he might come down on. The fairness issue aside, and I think that is fairly evident to everyone, the discipline placed today on waste disposers by the feedstock tax is nonexistent. Once we pay the tax we do what we can, and when times are good we are successful; when they are not so good, we are not, to pass that tax on to the ultimate consumer.

It serves as no discipline on those of our customers and the public who ultimately dispose of the vast majority of the materials that we produce and sell. The question of the waste-end tax was addressed at the time of the enactment of the original Superfund as I recall it. And at that time the—well, the overriding argument was that it would be impractical to collect.

I would suggest today that with the experience we have had with RCRA and the manifests that are available which do track hazardous waste, that particular objection is no longer quite as telling as it might have been. And I am also reminded that beginning I think it was October 1 or thereabouts, people who do dispose of materials in the ground are required to pay, I think it is a closure fee. So the recordkeeping system is much more with us today. And I think that we might be able to do this now without some of the problems that certainly were real when we began to do this 2 years ago.

Mr. **Levitas**. Would you like to add to that, Mr. Kusch?

Mr. **Kusch**. Mr. Kovall.

Mr. **Kovall**. Mr. Chairman, in your letter to Mr. Kusch you asked about whether or not we have been able to develop a corporate position; and if not, why not. I think it is pretty clear we don't have one—a full position yet. But if not, you asked what were some of the troublesome points. Certainly, I think you have hit right upon one of the real keys. We have perhaps an unusual corporation in that we have, we are not just a petroleum or chemical company operation, but we have solar, we are into solar engineering and mining coal and nonferrous metals. And the impact of any revenue-raising device is being very heatedly debated within our corporation.

It is no secret that we have been a strong proponent for switching to waste-end tax for purposes of CERCLA authorization. That comes primarily from what we think has been a positive experience in the State of California which has enacted a Superfund law with a multitiered waste tax revenue-raising device. And for some years it has had a waste fee process for funding its administrative programs in California.

We recognize, however, that a waste tax provides perhaps only a window of financial revenue flow, because if the tax in fact accomplishes what you would want it to accomplish, which is to reduce the wastes for disposition, the wastes do in fact become reduced. If in fact the tax is too high, I am sure you can imagine a scenario that one might be more encouraged to violate the law and not report the waste. In the State of California there has been, grossly speaking, a 30- to 40-percent reduction in wastes reported for disposition and subject to the tax in the last 2 years since the tax came into place.

So we are looking at the role that a waste tax should play in a total revenue picture. I think we would want to leave this message with you and this subcommittee very clearly, though, that we are very much opposed to the feedstock tax which we are convinced is inequitable. A recent Fortune magazine article indicated that 50 percent of that tax falls on five U.S. companies. Two of them are sitting here at this table today. We don't think a feedstock tax provides any appropriate environmental incentives, and it is terribly, terribly cumbersome as the newly proposed Internal Revenue Service regulations could demonstrate.

So we are looking at a wide variety of funding mechanism ideas. We really hope in the very near future that we will come out in the open concerning those ideas as we have about our basic advocacy. But we think a waste tax can play some role in at least CERCLA reauthorization for some period of time.

Mr. LEVITAS. I think you have addressed some of the concerns that we have in your comments. We would appreciate it if you would share with us any further conclusions you come to as your company goes through these considerations.

Mr. KUSCH. We will as we get to them.

Mr. LEVITAS. I would appreciate that. What we are looking at is not only an extension of Superfund, but I think that there is certainly overwhelming consensus, if not unanimity, that we are not only going to have to extend Superfund, but we are going to have to expand it. Instead of $1.6 billion we are looking at many billions more. And I think maybe I am understating that. We want to devise a system which is fair and which equitably shares the historic social cost that is a result of what we now know that we didn't know. At the same time, we want to provide for the future adequate resources and appropriate incentives to avoid perpetuation of problems of the past now that we do know what they are. Accordingly, I would look forward to receiving anything that you might have on that.

Mr. KUSCH. Yes.

Mr. LEVITAS. Let me move to another point. Both of you addressed the possible changes in the compensation system. However, I don't recall that either of you addressed the question of shifting burden of proof, which has been discussed by a number of witnesses before this committee, as being a minimum change that would be required if we or even if we don't go to a new administrative remedy. The difficulty remains in establishing the causal relationship and the fact is that most of the information probably lies, in this instance, with the defendant, rather than the plaintiff. The only way that we can have any sense of justice in the existing court system would be to shift in some way the burden of proof once certain threshold criteria were met.

Now, I would like to get your thoughts on that. I think, Mr. Kusch, you mentioned it. In the discussion, on page 4 of your testimony, you talked about this. However, I am just curious as to how you feel about shifting burdens of proof.

Ms. DINAL. One of the problems of shifting the burden of proof is that you have to have some criteria for doing so. I think, as was indicated by the previous panel, the problem is that there is just no certainty out there. The studies, the epidemiological and toxicological studies that have been done, have been inconsistent, and/or inconclusive. You can talk about shifting the burden but there has to be some threshold for doing so. And without some agreement on the part of the scientific community as to what might be a trigger exposure, it is very difficult to establish an appropriate threshold.

Another problem is when you are talking about a cancer I think the prevailing view among scientists is that the cause of such cancer is multifactorial. And so what we are talking about is a person being exposed to a number of triggers throughout his or her lifetime. In traditional causation you are looking for a "but for." You don't find that, perhaps, in the situation of cancer.

As we have seen with asbestos exposures, if you smoke and you are exposed to asbestos, your likelihood of developing certain types of asbestos injury are enormous as compared to someone who is exposed to asbestos and doesn't smoke. I think that when you talk about shifting the burden and we hear about uncertain evidence from the medical and scientific community over lifestyle factors, diet, etc. we just don't know what the triggers for disease are. And until we get some better guidance from the medical and scientific community, it is hard for lawyers to decide where the threshold should be for shifting that burden. If you shift the burden immediately to the defendant he has to prove a negative which is impossible.

Mr. LEVITAS. Let me pursue that just for a minute and then I will yield to Mrs. Johnson. This is obviously a very difficult situation. It is even slightly more vague than the whole discussion we have had for years about acid rain, when everybody was telling us, as you know, that we don't have enough proof. Yet, the fish are still dying in the lakes. Now, we are beginning to think we maybe do have some proof. I think that with or without that recent report, you would have seen legislation coming forward.

Certainly people in the Northeast—Connecticut, Massachusetts, Minnesota, Wisconsin—places like that, have a feeling about this. And there is, I think, if nothing else, a rather widespread and growing perception that there is a relationship between toxic chemicals and cancer or other illnesses. Associated with that, a like relationship between hazardous waste, dumps and the same type of problems.

Now obviously there is litigation going on right now. We heard from lawyers a few months ago who were telling us about the cases that are coming. They are increasing. There is a whole new segment in the bar handling toxic tort cases. Obviously there is somebody who is proving something to the satisfaction of the juries and the courts that are hearing them.

One of the questions and one of the paradoxes that we are confronted with from time to time was again raised yesterday by Assistant Attorney General McGrath. He says one of the ways you go about establishing causation is that you look at what the expected or normal rate of illness or disease, cancer deaths, is in a particular situation.

You then introduce the factor of the hazardous waste site or the nuclear fallout or whatever it is. And you show an increase of 5, 10, 15, whatever the percentage is. Now, you have your plaintiff come in and say, oh, there is a relationship here. But how do you know whether that plaintiff falls within the 15 percent or whether it is part of the first 100 percent?

As I said to Mr. McGrath yesterday, that is a very nice lawyer's problem that we can play around with either in a law school class on torts or in a philosophy class on some other part of the campus. However, I don't think it is going to go very far either with juries or Congress. Once you find that situation where there is obviously something going on that is out of the ordinary, I think the response is that we have got to provide compensation for whoever it is that might have fallen within that category, even if it is overly inclusive.

Now, my question is how do you respond to the issue of causation, recognizing the fact that there is litigation going on? And you might from your perspective, Ms. DiNal, share with us, what is the experience at Arco? Do you have a lot of claims from people who claim injuries? Do you have any ongoing cases?

Ms. DiNal. No; we don't have a lot of claims. I do think that the causation requirements probably would have to be relaxed if we are talking about a tier 1 compensation-type scheme. I think that is going to be one of the tradeoffs in tier one, that there is going to be a lesser burden for those people. But, at the same time, their damages are going to have to be limited in some way to medical expenses, and some lost income. They will have to forego the types of damages they would be able to ask for in litigation such as pain and suffering and emotional distress. I think that there probably will have to be a mechanism set up so that the information about particular substances is pulled together by the appropriate agencies, and so that there is some presumption created if a person can show a certain exposure over a certain length of time to a certain substance which is known to increase the likelihood of developing cancer.

I think it can be done in a compensation-type scheme. It is probably similar to, something, like workers' compensation. In exchange for the worker being able to sue for everything, if he is injured in the work place and can prove negligence he receives all his medical expenses and a disability income if necessary on a no-fault basis. I think we are talking about that kind of a tradeoff.

Mr. LEVITAS. I am curious if either company could just give us an indication. You are two large companies. Are you getting a lot of litigation on this? Do you have 10 cases, 50?

Mr. KUSCH. I think we have on the order of 50, isn't it, Joan?

Ms. DINAL. Over waste sites we have really just one.

Mr. LEVITAS. Waste sites, one.

What about chemical exposure in general?

Mr. BROWNING. We have—I am not aware of any cases that we have involving waste site exposure. I am not aware of any pending litigation that is prolonged in the workmen's comp area. We have a number of product liability cases, our materials in somebody else's workplace. They don't sue the employer, they sue us. There are a few of those.

Ms. DINAL. Yes; we have maybe about 50 some odd of those.

Mr. BROWNING. Yes.

Ms. DINAL. And about 20 asbestos cases, I think.

Mr. BROWNING. Mr. Chairman——

Ms. DINAL. The bulk of the asbestos cases have come in, of course, since the chapter 11's.

Mr. LEVITAS. Mr. Browning.

Mr. BROWNING. On the previous question, I don't have an answer for you, but some observations which might give you some lines of thinking that would be helpful that we have been pursuing.

In the first place, it has been implicit in your questions I think and in the observations here that we do make a distinction between acute illness and the chronic illness. The burn, the nausea, the dizziness. Those things fit into the traditional workmen's compensation or tort litigation without too much difficulty. Normally the sums involved are not great. The relationship between the event and the cause, the manifestation of disease, is fairly easy to prove. So those cases seem to be handled reasonably well.

The chronic case, the multiple employer, the long, latency period, those are the things that create the difficulties, where the emergencies are greater, and in this case you would be thinking of a large number of litigants; there appears to be an opportunity to get resources necessary to get the scientific proof to begin to establish causation.

I think the case we really need to focus on in the kind of system which you are considering here is where we have few claimants relatively in any given instance, and the case just doesn't permit the expenditure of the sums of money necessary to carry forward the purpose.

We have looked, and I don't have this system fully evolved in my own mind yet, but we have looked at the use of science panels, people who are expert in the disease processes, who could help establish a relationship, a causal relationship between a particular exposure and a particular disease process.

In my remarks here I mentioned a variation of this which I have personally found intriguing. And that has to do with a court-called expert, perhaps someone who is qualified to expound on the views that have been educed from studies by the National Academy of Science or some group like that.

Now, unfortunately, if that system is in place you will find that in most cases they will tell you that with lung cancer and this exposure you are dealing with a 15 to 25 percent increase in the disease process you are talking about. And that is compounded with smoking, and a few other things, as you certainly would be aware of.

I can only say in situations like that, where the causal relationship is so tenuous, or is tenuous to some extent, and you are going through the outside expert in an administrative system, the amount of the award might have to take into account that there is a likelihood that this person did not get that disease from that particular exposure.

It is some of the give and take that we have gone through in the regular Workmen's Compensation System where we are dealing traditionally with the finger in the drill press, the more acute emergency. Just observations which we are working on.

Mr. LEVITAS. Thank you.

Mrs. Johnson.

Mrs. JOHNSON. I would like to continue with the subject that we are discussing, the issue of an independent expert witness. How does that differ from toxic substance documents? Or do you think there is any advantage? What are the pros and cons of panels of independent witnesses and documents?

Mr. BROWNING. Well, yes. I do see some differences. In the first place, let me say to you that this idea of the court-appointed expert has been around for some time. My recent interest in it was stimulated by Mr. Robert Bernard who presented a paper on it a couple of weeks ago at one of the meetings here in Washington. If you are going to work in the tort system, it seems to me that you cannot deny constitutionally the parties the right to bring in their own expert witnesses.

I am not sure how you might introduce a paper, which just evolved from a National Academy of Science or some similar body, into the court of law. Certainly a witness who is basing his testimony on that would be in a position to bring it into play. And you would have to permit cross-examination of the expert, I believe, to satisfy the requirements of the court.

So a document which cannot be explained and updated and defended doesn't seem to me to be too useful in a court of law. In an administrative process, you might legislate in a way which would make that a more likely event. But even there I think both parties would need to have some access to either the author of the document or to the—someone who would defend the reasoning behind it, and also to make sure that the document was up to date.

Now, this is a field, most of these things we are learning about, you know. Not as much as we would like, and certainly not as quick as we would like. But it does—we are learning more every day. And we need to have that most recent expert testimony.

Mr. **Kovall**. I agree with Mr. Browning. I want to steer away from using the term technical substance document because it is a term that is being used in a couple of pieces of legislation in this Congress. But that concept I think has some value in an administrative proceeding, and I am assuming those types of documents which go to the causation in question are going to be subjected to rather rigorous regulatory proceedings and they are not going to just be a creation of some individual scientist's imagination—that these documents will go through some sort of review and be used for the administrative proceeding, where I visualize that of necessity you are dealing with a different type of causation showing than you are in the courts.

I think there is a tremendous danger—and I think it has been testified to perhaps before this subcommittee by Mr. Horowitz of OMB—that there is a tremendous danger of not making it very clear that if you are going to relax causation, don't carry that over to the litigation sense because you do run into the constitutional problems. And it is fundamentally disruptive of the judicial proceeding.

Mrs. **Johnson**. Do you think causation should be relaxed only if other remedies are foregone? If there is a selection made?

Mr. **Kovall**. Yes; we have stated that there ought to be a very clear binding election of remedies, and if you go one route, that is the route you go. You go to court with perhaps tougher standards, more clear——

Mrs. **Johnson**. If you establish the system with a binding election of remedy, then you would not object to some loosening of the causation standards?

Mr. **Kovall**. That is true.

Mrs. **Johnson**. Then in the area of subrogation, what would be your reaction to limited subrogation that dealt only with those situations where there had clearly been deliberate and willful misconduct?

Mr. **Kusch**. I don't think we are prepared to speak to that question. We are having quite a debate right within our own corporation on this. It has anything but come to a conclusion.

Mrs. **Johnson**. OK.

Mr. **Kusch**. I would guess that the debates that are going on on a number of these issues right within our corporation, among the various entities, companies, if you would, that there are probably parallels in all the debates that are going on before committees like yours, and in many other places.

Mr. **Browning**. My company doesn't have a position on that, but I will give you a personal observation.

The question of subrogation is a very difficult one. And, of course, the idea initially is to get a system which eliminates, to the extent one can, the transaction costs involved in getting money to the injured party. Subrogation would seem not to go in that direction, if you are just going to move the litigation off to another arena at another time.

Although if carefully defined, the kind of exception that you have raised would seem to me to be worth serious consideration.

Willful misconduct should be punished, and the system ought to be designed so that it militates against those people who have gone beyond the ordinary care——

Mrs. JOHNSON. Would you just clarify for me because this came up in yesterday's hearing as well as today's hearing. Could you just clarify for me what kinds of cases are being already taken care of through existing systems? Which kinds of cases are addressed through which systems?

Mr. BROWNING. You mean which cases are being taken care of today in the court system?

Mrs. JOHNSON. For instance, your testimony says, Mr. Browning, the present tort system will probably continue for some time to be a principal means. Are there other systems besides the tort system that is taking care of or addressing some aspect of it? And what aspects is the tort system handling?

Mr. BROWNING. Well, to get to the second part first, there has recently been an announcement I understand, somewhat premature but not too much, of the settlement of the claims of the people who lived around Love Canal. Certainly a few months ago there was some evidence, or a settlement of the claims of those who had been injured, I believe it was in Louisiana as a result of some environmental episode there that occurred several years ago.

So, again, you have large numbers of claimants, and you have a situation where the tort system is working. Presumably to the not total dissatisfaction of both sides, although I am sure that the people paying think they paid too much, and the people who are getting paid don't think they are getting enough. But that is the system.

Mrs. JOHNSON. Wouldn't you say that was primarily a negotiated settlement, or was that a court settlement?

Mr. BROWNING. It was litigation, yes, in both instances as I recall it.

Ms. DINAL. Yes.

Mr. BROWNING. Certainly in Love Canal, lots of suits were filed. And I am told that while the announcement is premature, that they are close to some kind of an accommodation there. So the tort system does work in some cases. When you get away from the tort system and you deal with the administrative system, the only one that comes to my mind quickly is the Workmen's Compensation System. And it is used to take care of a good many of these so-called toxic torts.

I don't know of a single system that is operable in environmental problems.

Mrs. JOHNSON. Do you have any idea how many of the workmen's comp cases are related to this kind of a cause? Do you have any idea how much is being——

Mr. BROWNING. In our case, it is certainly a very small fraction. We don't have many claims.

Mrs. JOHNSON. Ms. DiNal?

Ms. DINAL. No; that is the same situation at ARCO. In relation to all the workmen's comp claims, very few of them are for some kind of hazardous substance exposure.

Mr. KUSCH. As a matter of fact, they are rare.

Ms. DiNal. Yes, and most of them are for skin irritations or burns.

Mrs. Johnson. Thank you, Mr. Chairman.

Mr. Levitas. Thank you.

Mr. Browning, I understand that you are chairman of the Chemical Manufacturers Association's Strategic Options Group, which has examined policy alternatives on victims compensation.

Mr. Browning. I was, the committee is in a state of limbo at the moment.

Mr. Levitas. That was my question. My question is that I wanted to know how that group is coming along with its deliberations.

Mr. Browning. I think this is true, Mr. Chairman. The board of directors of CMA met yesterday. And our original charter was to explore many of the issues and sort of define the issues that needed further elaboration, and we made that report. The board of directors, I am told, agreed yesterday to reconstitute the committee, and it isn't clear or isn't certain at this point that I will be the chairman, although there has been that indication.

Mr. Levitas. What do you see as the direction? Obviously you can't anticipate what is going to happen, but what sort of strategic options are you viewing other than those which we have all been hearing about from the 301(e) study group?

Mr. Browning. This is largely the sort of thing we would review. The merits of the various proposals and the interrelationship of these things. Now I think Congresswoman Johnson raised the point, if you go for a lessening of the burden of proof, does that push you toward an administrative system that is exclusive?

These are the kinds of tradeoffs that I think ultimately we are going to have to weigh and decide, and how much do you lessen the burden of proof in that situation?

Mr. Levitas. I think that there are obvious differences of opinion, but I also think that to the extent you reduce the burden of proof, you go for some tradeoffs which may be a limitation on liability.

Mr. Browning. Yes.

Mr. Levitas. On the other hand, the problems, without getting into all of them, are so many as you go down the line. For example, you have a compensation system, and presumably you are being compensated for an ailment which you now elect exclusively to receive from an administrative tribunal.

A year later, instead of having an ailment here, you now have an ailment there. Have you elected exclusively to deny compensation for that ailment? If that ailment is one which is more precisely related to a particular type of chemical to which there was exposure, you think well maybe I won't go into the administrative tribunal for that ailment. I will go into court because I have got a better case here.

I am not suggesting any answers. I am simply saying, looking at it even superficially, the problems begin to unfold almost in front of your eyes.

Mr. Browning. The only quick answer that comes is you have a time limit. If you have taken a bite of the apple today, you don't get another shot at it for three or four years, something of that sort. But it is a difficult problem.

Mr. LEVITAS. I am just curious as to the direction in which CMA's strategic options group was headed, and I think you have answered that question.

Mr. BROWNING. I think one of the things we would be doing certainly is working more closely with the insurance people to see if we can make sure that we are in an insurable area with the claims that might arise. And explore—I think we need to get into more details on the workings and adequacy of the compensation being provided by the Workmen's Compensation Systems for these chronic injuries. We have both given some general thoughts. But that needs a further review.

Mr. LEVITAS. Mrs. Johnson.

Mrs. JOHNSON. Just sort of along the same line, in your estimation, being as familiar as you are with the complexities and the needs, do you think if left to its own evolutionary trends that the tort system would be able ultimately to deal with the problem? Leave it up to judicial interpretation?

Mr. BROWNING. I guess if I answer the question on the basis of my knowledge today of the number of claimants and those that I perceive to be uncompensated, I would have to say that the answer is yes, with the exception of small claims.

Now, I think there probably are some people who couldn't go for the million dollars or so. But they do have some difficulties. Perhaps there is an area there that needs to be thought through more carefully. But our experience and what I read doesn't tell me that there are a great number of people uncompensated as a result of exposure to hazardous materials, particularly around dump sites.

Mrs. JOHNSON. Thank you.

Mr. KUSCH. The problem that you as legislators are facing is the tremendous public perception that they are really being harmed. And I would guess that back in various districts who are represented in the Congress are people like you folks who run into a lot of that when you go back home. There is no way, even if you get all the evidence and science in the world, you can tell those guys, you know, nothing is happening to you. There is no way you are going to get away with that.

So then you are faced with, well, what kind of legislation do I create to mollify that sort of thing? It is a very difficult, difficult problem.

Mrs. JOHNSON. Yes.

Mr. LEVITAS. Yes; I just would make a comment about that, because you are absolutely correct, and I take what you say as being a substantive statement.

Mr. KUSCH. It is my own opinion. It is not——

Mr. LEVITAS. I take it as a substantive statement.

Mr. KUSCH. All right.

Mr. LEVITAS. But I will tell you that in the minds of some people that gets translated into the fact that Congress is simply reacting to witches and goblins and making a political response. However, it is my sense, and I am not running down science in this regard, that there is some sort of commonsense understanding that people have. It is like, and I don't know if this is an appropriate analogy, but it is like pornography. It is hard to describe, but everybody knows what it is when they see it.

It may be hard to establish to the satisfaction of scientists and certainly it is incapable of being defined satisfactorily to statisticians and epidemiologists. But there is sort of a correct understanding by people that say, look, there are more people getting sick right here near that toxic waste site than there are everyplace else. So there must be something wrong there.

Mr. KUSCH. Yes.

Mr. LEVITAS. So I take what you say as a substantive observation. If I could predict, I would predict that Congress is going to respond in some way as a result of that.

Mr. KUSCH. I think you have no choice.

Mr. LEVITAS. I think that is right. I am sorry.

Mrs. JOHNSON. You just pursue that.

Mr. LEVITAS. The last question I have is something which I think, Mr. Kovall, or Mr. Kusch alluded to and which we asked at our earlier set of hearings. You, talking about the need for certainty, said in order to make your plans, above all you had to be able to have an insurable system.

When we were talking with the representatives of the insurance industry, I remember asking the question, how do you actuarially provide for insured coverage for this type of situation when there is no experience? I mean, there really is no experience at this point, at least adequate experience, to define what should be the premium costs associated with this type of risk.

I wonder if any of you at the table had any thoughts on that subject because obviously it will take a period of time of insurance coverage and experience in order to find out what we really are talking about regardless of what system is put in place.

Mr. BROWNING. It is really the thought that I had in mind when I was talking about the direction of the CMA in the future. I think we do have to work with the insurance industry to begin to define the boundaries. If the burden of proof is relaxed this much, what does that do to the risk that we are running that you would have to insure? We don't have good understandings of that.

Now, everybody understands preponderance of evidence. We have lived with it a long time and actuarially you can predict that. If we begin to play with that, it is a whole new ball game. But we would have to call on their experience in other arenas and to help us put some price tags on it.

Mr. LEVITAS. I have no further questions.

Mrs. JOHNSON. I don't have any further questions. Thank you.

Mr. LEVITAS I want to thank each of you for your willingness to participate in our review of the victims' compensation issue and also my thanks to the other witnesses who appeared before the subcommittee during the six days of hearings we have held so far on the subject. Your contributions have been invaluable and have provided us with a record that will enable us to go forward with our tasks and measurably assist us in more knowledgeably addressing the issue of hazardous waste and victim compensation.

I think the more we hear, the more obvious it becomes that the issue is complex. There are differences of opinion. There are areas of uncertainty that will not be easily resolved, if ever resolved. In view of this, it would be premature to attempt any conclusions.

But we are going to be analyzing this extensive record over the next weeks and months ahead with the intent of providing some specific legislative proposals or other proposals on this subject in order that we can ensure that we will provide all those who wish to comment an opportunity to do so.

Accordingly, I will, without objection, order that the record of these hearings be held open for two weeks. Specifically I might say with respect to your comment concerning the 3M Corp., we will certainly be more than happy to accept their filing for that purpose.

Mrs. JOHNSON. Thank you.

Mr. LEVITAS. With that, again, thank you for your participation. I appreciate your being here.

The subcommittee stands adjourned.

[Whereupon, at 3:02 p.m., the subcommittee was adjourned, subject to the call of the Chair.]

[The following additional items were received for the record:]

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

DOC # ____
CODE ____ REC # ____
DATE. ____
PARA. ____

AUG 25 1983
OFFICE OF
POLICY AND RESOURCE MANAGEMENT

AUG 23 1983

Honorable Elliott H. Levitas
Subcommittee on Oversight and
Investigations
Committee on Public Works and
Transportation
House of Representatives
Washington, D. C. 20515

Dear Congressman Levitas:

Thank you for your letter of August 4, 1983, concerning EPA's risk assessment and risk management policies. We have begun to collect the information you requested and will provide you with as much information as possible by September 9. In the meantime, I would like to indicate how we intend to proceed and perhaps provide some preliminary answers to your questions.

Your first question concerned EPA policies for risk assessment and risk management prior to Mrs. Burford's tenure as Administrator. It is my understanding that during this period generic policies and uniform guidance on risk assessment were not articulated by the Agency in a formal way. We are searching the record, but it is likely that policy in this area will have to be inferred from an analysis of individual cases.

Second, you ask if there were any changes in these policies during Mrs. Burford's tenure as Administrator. Again, I believe that risk assessment and risk management policies at that time were not explicitly stated and will have to be inferred from an analysis of individual cases. A memorandum from Dr. John Hernandez was specifically referenced in your letter. We have asked each program office to review and comment on this memorandum and submit to us any comments generated at the time the memorandum was issued.

Finally, you asked whether there had been any changes in these policies since Mr. Ruckelshaus became Administrator or if any are being contemplated. The Administrator is at present engaged in several efforts that we hope will result in a careful articulation of policies in the areas of risk assessment and risk management. The impetus for this activity is the broad public concern that surrounds policies for dealing with environmental risks. Some of the issues are scientific--how hazards should be identified and assessed. Others are managerial--ensuring that we reduce total risks in the most efficient possible way, which suggests, among other things, coordination of total efforts across media and hazards. Still others are primarily philosophical--including the extent to which risk reduction should be balanced with other important goals.

The Administrator has initiated discussions with the heads of other Federal agencies and departments involved with risk assessment and risk management. The goal is to achieve greater cooperation among the relevant agencies, to improve the use of scientific knowledge in regulatory decisions, and to seek to reduce overlaps, gaps, and inconsistencies in the way agencies handle related regulatory issues. At EPA, we are assembling the analytic basis to help with that reexamination. We have set up a Task Force charged with:

- Finding out where we stand on uniform risk assessment guidelines for EPA and developing a plan and schedule for updating and writing these as needed;
- Developing policies for the management of significant health risks, and assuring that risk management information on such factors as exposure, hazard, uncertainty, risk reduction, inter-media transfer and cost are presented to Agency management in a consistent, comparable format;
- Improving our ability to respond quickly and efficiently to problems of immediate national concern--dioxin contamination, for example; and
- Improving coordination between EPA and other Federal agencies responsible for controlling toxic substances.

The final report to the Task Force, due November 30, will be an important input to an ongoing process of developing coherent and consistent strategies for risk assessment and risk management. We will provide you and your committee by September 9, however, some tentative answers to your third question. We also shall keep you apprised, of course, as this process develops.

I will be happy to meet with you at your convenience to discuss these issues and the progress we are making in developing policies for risk assessment and risk management.

Sincerely,

Milton Russell

Milton Russell
Special Assistant
to the Administrator

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

SEP 12 1983

OFFICE OF
POLICY AND RESOURCE MANAGEMENT

Honorable Elliott H. Levitas
Subcommittee on Oversight and
Investigations
Committee on Public Works and
Transportation
House of Representatives
Washington, D.C. 20515

RECD [illegible] 1983
Subcom[illegible]igations
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Comm[illegible] on Public Works
[illegible] Transportation

Dear Congressman Levitas:

This letter and the enclosed materials are submitted in response to the questions raised in your letter of August 4, 1983. The program offices have provided a variety of materials that I hope will be of interest to you. Let me summarize the highlights and elaborate on my original response to you of August 23.

Risk assessment and risk management prior to Mrs. Burford's tenure as Administrator

The recent National Academy of Sciences report on risk assessment and risk management has done much to clarify some difficult topics. It has been especially useful in clarifying the distinction between the two concepts, and in encouraging staff and managers at all levels to make it as clean as possible. Simply put, assessment is a description of risk, management is a judgment on what to do about it.

Risk assessment interprets toxicological and other data on the hazards of pollutants, usually relying heavily on animal experiments, and arrives at conclusions on that pollutant's human health effects. This is then combined with data on the patterns and extent of human exposure from a particular pollution source or set of sources. The result is a calculation (for carcinogens this is generally expressed as a probable upper bound) of the health risk of a pollutant. In contrast, risk management is the process of weighing the results of the risk assessment, along with economic and other important factors, and arriving at a decision on actual control of the target source(s). The NAS report stressed the importance of keeping the assessment component entirely free of judgments that anticipate desirable degrees of control. All such evaluations should remain in the domain of risk management.

This distinction was less obvious three years ago, and, as I stated to you in my letter, risk policies prior to Mrs. Burford were not explicitly stated as such, and are best interpreted from the actual decisions made. On the other hand, certain procedures, especially those affecting what we now regard as the purely scientific and technical process of risk assessment, had by that time been made formal, at least in draft form. Copies of the pertinent documents are enclosed.

The Interim Cancer Assessment Procedures (1976) and the proposed Guidelines for Mutagenicity Risk Assessments (1980) were issued prior to the tenure of Mrs. Burford. They represented the state of a new science as it was then. They were not altered under her administration, but are now being reviewed to incorporate new topics and update old ones.

The major questions on risk assessment, both prior to Mrs. Burford and during her administration, relate more to how consistently the available guidelines were applied than to any conflict between competing scientific approaches. Imperfect as risk assessment was then and is now, there is no credible scientific alternative to the basic approach embodied in the existing draft guidelines.*

The major controversies since the publication of risk assessment guidelines have centered instead on issues of risk management. The major questions are:

> What weight should be given to the results (and especially the uncertainties) of both qualitative and quantitative components of risk assessments?
>
> To what degree are formal policies on the management of risk possible or desirable (especially in relation to the requirements of statutes)?

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* There was discussion and consideration of distinguishing among carcinogens with differing modes of biological action, including some that might have exposure thresholds below which no risk is present. I enclose a copy of a report prepared and circulated by the Carcinogen Assessment Group on this subject, along with the peer review commentary it evoked. It was decided that the available scientific evidence did not yet support the use of such an approach. Dr. Hernandez testified to this effect shortly before he left the agency.

The first attempt to deal formally with these questions prior to Mrs. Burford's administration resulted in the enclosed draft Cancer Policy Statement, produced by a subgroup of the then-active Toxics Substances Priorities Committee (TSPC). In addition to the general questions above, the statement dealt with the following specific types of judgments:

> At what level of risk to the population or to individuals should the agency act?
>
> What should be target levels for regulatory control?

The group itself was unable to reach full consensus (see enclosed memorandum from Dr. Arnold M. Kuzmack and Dr. Elizabeth L. Anderson to the TSPC). Though the work was begun under the previous administration, it was completed during the Burford administration. Mrs. Burford and Dr. Hernandez commissioned no further work in this area, and did not formally implement the draft cancer policy in this or any other form.

Risk assessment and risk management during Mrs. Burford's tenure

As stated above, there was no change in the content or use of risk assessment guidelines during the Burford administration. Differences of opinion did, however, exist in relation to risk management.

The memorandum from Dr. Hernandez which you forwarded to us for comment is the only instance we have come across where an attempt was made to create a formal alternative to the risk management policies suggested by the TSPC. We have reviewed the headquarters files and are unable to find any written commentary in response to Dr. Hernandez on his memorandum. The only possible reference forwarded to us by the program offices is a memorandum to the Deputy Administrator from Marilyn Bracken; I do not think it is relevent to the question you raise, but have included it for your review.

Dr. Hernandez's memorandum is aimed at the evaluation of local incidents (I believe it was written with the Dallas lead case in mind), rather than at regulatory policy in general. We believe it reflects Mrs. Burford's and Dr. Hernandez's emphasis on the need to have quality assured data and peer reviewed studies prior to taking any action on potential environmental problems, and to review every decision on a case by case basis.

This philosophy was also applied to broader regulatory action under Mrs. Burford. Case by case review does not lend itself to extensive or formal policy statements, and to our knowledge none was ever written. Some decisions on individual regulations made under Mrs. Burford, however, do appear to depart from the draft TSPC Cancer Policy.*

Current efforts on risk assessment and risk management

Since Mr. Ruckelshaus took office there has been a strong emphasis on clarifying, improving, extending, and utilizing uniform risk assessment procedures. There is equal interest in the development and implementation of better risk management policy.

The vehicle for this work is the Task Force on Toxics Integration set up by Deputy Administrator Alm, whose basic activities I described in my first letter. Richard D. Morgenstern, Director for the Office of Policy Analysis within the Office of Policy and Resources Management, leads this group. Membership includes Dr. Anderson and Dr. Kuzmack (who had prime responsibility, respectively, for risk assessment guidelines and the draft cancer policy). The task force also includes senior representatives from the regional offices and each of the Assistant Administrators.

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* I have included an article from a recent issue of "Science," which quotes a memo written by Burford to the White House referring to cancer policy as "a policy this Administration is changing." The Burford memo anticipates controversy over a decision on the pesticide permethrin -- controversy that never in fact materialized, to our knowledge. As part of our Toxic Integration Task Force (see next page) we have in fact reviewed the risk management aspects of the permethrin decision, and found nothing unusual (see enclosed report, referenced next page). The important conclusion here is that Mrs. Burford was aware of case-by-case departures being made from previous policy, and anticipated that it would generate controversy.

I enclose copies of the pertinent reports the task force has produced to date. Of particular interest is the report of the subcommittee on risk management, which analyzes 27 recent regulatory actions for their positions on risk management. The report displays, for each action:

- The name of the regulation, standard, or guideline;
- The pertinent route of human exposure to risk;
- The source of exposure to be controlled;
- The population whose risk was considered as part of the regulatory decision;
- The health effect of principal concern;
- The strength of evidence of risk presented to the decision maker;
- Estimated levels of pre- and post-regulatory risk;
- Any treatment by the regulatory analysis of intermedia transfer of pollution (air to water, water to solid waste, etc.);
- The regulatory alternatives considered;
- The results of any cost-effectiveness analysis performed; and
- Considerations on which the decision was finally made.

The analysis has revealed wide variations in the way different programs treat these factors. Much of the variation is attributable to the statutes under which the actions are taken. However, the Administrator is anxious that:

1. Unnecessary inconsistencies in the control of risks are minimized or eliminated;

2. That analytic presentations on risks and other factors affecting the decision are clear and complete; and

3. That his discretionary powers (a) reflect consistent policy and (b) are exercised with full awareness of the impacts of previous regulations affecting the same chemicals and industries.

The issues here are difficult, and I do not want to convey the impression that we have reached any final conclusions or are about to make major changes in current policies. We do think, however, that this work is on the right track, and will produce substantial benefits in helping us to make clearer and more consistent decisions.

I am grateful for your interest, and hope that you will follow our work as it progresses. I hope the enclosed materials are useful, and will be glad to be of additional assistance if you need more.

Sincerely,

Milton Russell

Milton Russell
Special Assistant
to the Administrator

ENCLOSURES

In apparent response to Dr. Hernandez's memorandum:

MEMORANDUM: Marilyn Bracken to John W. Hernandez
SUBJ: A Generic Work Plan or Guidance for EPA Study
DATE: October 23, 1981

Risk assessment:

INTERIM CANCER ASSESSMENT GUIDELINES
DATE: May 19, 1976

PROPOSED GUIDELINES FOR MUTAGENICITY RISK ASSESSMENTS
DATE: October 30, 1980

THE CARCINOGEN ASSESSMENT GROUP'S METHOD FOR DETERMINING THE UNIT RISK ESTIMATE FOR AIR POLLUTANTS
DATE: August 8, 1980

WATER QUALITY CRITERIA DOCUMENTS
DATE: November 28, 1980

OAOPS DOCUMENT: Risk Analysis Program Plan and Accomplishments
DATE: February 1981

MEMORANDUM: Victor Kimm to Merna Hurd
SUBJ: Outline for Background Documents on Environmental Human/Health Effects and Risk Assessment
DATE: April 9, 1982

STAFF DRAFT: Guidance on Suspect Carcinogens with Specific Reference to Water Quality Criteria
DATE: June 21, 1983

COMMENTS FROM REVIEWERS OF "ADDITIONAL USEPA GUIDANCE FOR THE HEALTH ASSESSMENT OF SUSPECT CARCINOGENS..."
DATE: August 23, 1982

Risk Management:

MEMORANDUM: Arnold Kuzmack/Elizabeth Anderson to TSPC
SUBJ: Request for Review of Draft Cancer Policy (contains copy of draft cancer policy)
DATE: July 22, 1981

REPRINT: Paper by Victor Kimm, Arnold Kuzmack, and David Schnare
SUBJ: Waterborne Carcinogens: A Regulator's View
DATE: undated

REPRINT: Article by Dr. Bernard H. Haberman
SUBJ: EPA planned to Alter Carter Policy on Cancer
DATE: undated

STAFF DRAFT: Guidance on Response to Contamination Detected in the Ground Water Supply
DATE: April 29, 1981

STAFF DRAFT: Proposed Process for Evaluation and Control of Toxic Air Pollutants
DATE: March 23, 1983

Documents pertaining to the Toxic Integration Task Force

MEMORANDUM: Richard Morgenstern to prospective members
SUBJ: Outline and goal statement for task force
DATE: July 1983

INTERIM REPORT: Subcommittee on Risk Management
DATE: August, 1983

INTERIM REPORT: Subcommittee on Risk Assessment
DATE: August, 1983

INTERIM REPORT: Subcommittee on Interagency Liaison
DATE: August, 1983

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

SEP 16 [illegible]

OFFICE OF
POLICY AND RESOURCE MANAGEMENT

Honorable Elliott H. Levitas
Subcommittee on Oversight and
  Investigations
Committee on Public Works and
  Transportation
House of Representatives
Washington, D.C 20515

Dear Congressman Levitas:

The letter I sent to you dated September 12, 1983, contains an inadvertant error. I have enclosed a substitute page and would appreciate your cooperation in replacing the erroneous page.

Thank you for your assistance in this matter. I regret any inconvenience this may have caused.

Sincerely,

Milton Russell

Milton Russell
Special Assistant
 to the Administrator

Enclosure

This philosophy was also applied to broader regulatory action under Mrs. Burford. Case by case review does not lend itself to extensive or formal policy statements, and to our knowledge none was ever written.*

Current efforts on risk assessment and risk management

Since Mr. Ruckelshaus took office there has been a strong emphasis on clarifying, improving, extending, and utilizing uniform risk assessment procedures. There is equal interest in the development and implementation of better risk management policy.

The vehicle for this work is the Task Force on Toxics Integration set up by Deputy Administrator Alm, whose basic activities I described in my first letter. Richard D. Morgenstern, Director for the Office of Policy Analysis within the Office of Policy and Resources Management, leads this group. Membership includes Dr. Anderson and Dr. Kuzmack (who had prime responsibility, respectively, for risk assessment guidelines and the draft cancer policy). The task force also includes senior representatives from the regional offices and each of the Assistant Administrators.

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* I have included an article from a recent issue of "Science," which quotes a memo written by Mrs. Burford to the White House referring to cancer policy as "a policy this Administration is changing." The memo mentions this in reference to anticipated controversy over a decision on the pesticide permethrin. Frankly, I do not know what Mrs. Burford meant by this statement -- whether she refers to the still-continuing OSTP effort to revise cancer policy, whether she believed the permethrin decision itself embodied a change in cancer policy, or some other reason.

A number of key officials involved in the decision, such as Ed Johnson, the Director of Pesticide Programs, have testified that the permethrin action was consistent with previous agency policy. As part of our Toxic Integration Task Force (see next page) we did in fact review the risk management aspects of the permethrin decision, and found nothing unusual in its risk management policies (see enclosed report, referenced next page). The conclusion I draw is that Mrs. Burford's preference for case-by-case risk management policy certainly may have allowed for departures from TSPC policy, but we have no evidence that the regulatory judgment regarding permethrin departed from accepted policy.

February 1981

U.S. Environmental Protection Agency (EPA)
Office of Air Quality Planning and Standards (OAQPS)
Strategies and Air Standards Division (SASD)

RISK ANALYSIS PROGRAM PLAN AND ACCOMPLISHMENTS

In 1979, EPA's Science Advisory Board (SAB) Subcommittee on Health Risk Assessment recommended that OAQPS develop and "establish the credibility" of a risk assessment method for use in evaluating alternative national ambient air quality standards, and by implication recommended that EPA address how risk evaluation can best be incorporated into the standard-setting process. In late 1979, OAQPS started a Risk Analysis Program. This plan delineates both the projects undertaken since that time and those proposed for FY 1981-83.

For purposes of this program plan, risk analysis is broken into two components, risk assessment and risk evaluation. Risk assessment is the process of making risk estimates and describing the severity of harm associated with adverse events. Risk evaluation is the process of evaluating how much risk is acceptable in light of all relevant circumstances.

RISK ASSESSMENT

The primary objective of the risk assessment portion of the program is to choose a risk assessment method to use in assessing health risks associated with setting alternative national, ambient air quality standards (NAAQSs).* Main elements of the risk assessment program are:

- further development and evaluation of the F/B Risk Assessment Method.+
- development and evaluation of alternative risk assessment methods.
- investigation of issues related to risk assessment in general.

---

[*] It is recognized that there are possible outcomes of the program not as simple as the choice of a single "method." A possible outcome is that a general approach to doing risk assessments will be deemed appropriate no matter what the circumstances, and the particular method within this general approach judged to be appropriate will be a function of nature of the pollutant and time and resources the user chooses to expend in performing the assessment.

A spin-off of the program may be the identification of one (or more) risk assessment method(s) suitable for use in setting national emission standards for hazardous air pollutants (NESHAPS). The resources appropriate to spend for a NESHAPS risk assessment are presumably less than that for a NAAQS risk assessment.

[+] The F/B Risk Assessment Method is an approach to estimating health risks associated with alternative ambient air quality standards. It was developed by SASD analyst, Thomas B. Feagans, and a contractor, Dr. William F. Biller.

Specific projects have been and will be undertaken to address these three elements. Project summaries follow.

A. INVESTIGATION OF ISSUES RELATED TO ASSESSING RISK IN GENERAL.

The SAB Subcommittee and others have recommended that work be undertaken to address issues related to the use of expert judgment in assessing health risks associated with alternative NAAQS. These issues are common to the F/B and alternative methods currently under consideration. Another common problem is how should exposures to ambient air concentrations be estimated. A third general area of interest is to understand what issues should be addressed in a risk assessment and how other federal agencies currently address them. A brief description of past and proposed projects focused on these general issues and problems follow.

Completed Projects.

° Address behavioral psychology issues. The first project undertaken on issues associated with the use of expert opinion in assessing health risks of alternative ambient air standards was a review of empirical literature on subjective probability encoding. This review was undertaken by Drs. Thomas S. Wallsten and David V. Budescu of the University of North Carolina's Thurstone Physhometric Laboratory (Psychology Department). The review is published in:

Thomas S. Wallsten and David V. Budescu.
Encoding Subjective Probability: A Psychological and Psychometric Review. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, 1980.

° Describe instrumental factors inherent in a NAAQS risk assessment. A small project was undertaken to describe factors that must be addressed in a health-focused risk assessment of alternative ambient air standards. The project also inventoried and classified existing non-carcinogenic health risk assessment approaches according to how they handled various conceptual issues inherent in assessing health risks. A typology of approaches was developed, focused on (1) output measures, (2) methods used to obtain output measures, and (3) inputs required.

This project was contracted to Dr. David Moreau, Professor of Environmental Planning, University of North Carolina. Results are contained in:

David Moreau.
Quantitative Risk Assessment of Non-Carcinogenic Ambient Air Quality Standards. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, 1980.

° Describe Existing Risk Assessment Approaches Used by Federal Agencies. An inventory of "state-of-the-art" non-carcinogenic risk assessment approaches used by selective Federal agencies was pursued under another small project to Dr. David Moreau. The typology developed in the previous project was used to characterize and classify the various approaches. The inventory is contained in:

David H. Moreau and Mary Elizabeth Anderson. Quantitative Assessments of Health Risks by Selected Federal Agencies. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, 1980.

° Estimate exposures to NO2 ambient air concentrations. An exposure analysis is an integral part of a risk assessment in that it provides the estimate of "dosage" seen by sensitive and normal population groups. A nitrogen dioxide (NO2) exposure analysis was undertaken in four urban areas, where population exposure was estimated using actual air quality information, population location data, and transportation movement estimates. In addition, indoor-outdoor relations of the pollutant were estimated and data on gas-stove usage was obtained to account for indoor exposure to NO2. Human activity level, which affects ventilation rate was also estimated. Exposure estimates, therefore, were time and space dependent and varied by population group, location, and physical activity. Sensitivity analyses were undertaken on key variables to understand their importance in contributing to total NO2 exposure.

The exposure analysis was done by the "SASD exposure team," consisting of EPA staff, Dr. William Biller and PEDCo Environmental, Inc., contractors. An interim report was written; it is:

Ted R. Johnson and Roy A. Paul. Analyses of Population Exposure to NO2 in Selected Urban Areas. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, 1981.

On-Going Projects

A-1. Investigate behavioral psychology issues.

Dr. Thomas Wallsten of the University of North Carolina is undertaking an experimental investigation into two important issues associated with the use of expert opinion in setting ambient air standards. They are: (1) the question of "stability" of encoded probability judgments over time, and (2) the related question of the degree of randomness in encoded probability judgments by the means of encoding two corresponding dose-response probability surfaces.

Dr. Wallsten will present his findings in an EPA report. Completion date: October 1981.

A-2. Estimate exposures to SOx/PM/and CO ambient air concentrations.

The exposure analysis already developed for NO2 will be modified and applied to three additional pollutants, sulfur oxides (SOx), particulate matter (PM), and carbon monoxide (CO). In essence, three separate exposure analyses will be produced. Further methodological development of exposure modeling is the main purpose of this project from the risk assessment program perspective. The analyses will also be used in review of existing standards for the three pollutants.

Depending upon whether CO or ozone is chosen as the pollutant of interest in Project C-1, below, the CO exposure analyses may be used to further develop the F/B and alternative approaches risk assessment methods.

The SASD exposure team is undertaking all analyses in this project. Three reports are expected, one for each pollutant. Report due dates are: CO, July 1981; SOx, September 1981; PM, October 1981.

Future Projects

A-3. Estimate exposures to O3 ambient air concentrations.

Again, the SASD team will modify and improve its exposure analysis by applying it to ozone (O3). This project may be substituted for the CO part of the A-2 Project, if O3 is chosen as the pollutant of interest in Project C-1. A report will be the final product; it will be finished in late 1981.

A-4. Develop evaluation criteria for Project A-6.

This project will be focused on developing explicit criteria against which the remaining risk assessment methods will be evaluated in Project A-6. It will be done largely by SASD staff, with input from the SAB Subcommittee on Health Risk Assessment and Argonne National Laboratories. This project will be undertaken in mid-1982.

A-5. Estimate exposures to Pb ambient air concentrations.

A full-scale exposure analysis of lead (Pb) will be undertaken in four major U.S. urban areas. Results from this project will be extrapolated to the nation as a whole to obtain estimated national population exposures to this pollutant. This analysis provides inputs into the application/evaluation project. (Project A-6). The project will be undertaken in late 1982.

A-6. Apply and evaluate (the remaining) risk assessment approaches.

This project will be a "third-party" technical application and evaluation of the risk assessment approaches still under consideration by OAQPS in mid-1982. (There will be three at most; if there is only one, this project will not be undertaken). It is proposed to use lead as the subject pollutant and to possibly use results of this project in OAQPS's NAAQS-review effort. It is proposed to use the Decision and System Sciences group in Argonne National Labs as the third-party contractor. All developers of risk assessment approaches still under consideration will be directly involved in this project, doing specific tasks related to their particular approach under Argonne's general guidance. Argonne will prepare a report on its technical evaluation. The approximate completion date for this project is June 1983.

A-7. Review the application project and evaluate policy implications.

The purposes of this task are to review findings of Project A-6 and choose a risk assessment method (or methods) that will be used in the NAAQS-setting process. As part of the review, OAQPS--with assistance from OPE*--will evaluate policy implications of using the risk assessment method(s) found to be technically adequate. The project will be done in-house in mid-1983.

A-8. Investigate how risk assessment outputs can be best presented to EPA managers and the public.

Two interrelated questions will be addressed: first, how to best present results of a risk assessment to EPA decision-makers; second, how to best inform the public about health risks associated with alternative air standards. This project will be undertaken in mid-1983 by SASD, with perhaps some assistance by an outside contractor. The project must be closely coordinated with project D-4.

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*Office of Planning and Evaluation in EPA headquarters.

B. DEVELOPMENT OF THE F/B RISK ASSESSMENT METHOD

The SAB Subcommittee made several suggestions with regard to work that would aid development, explanation, and evaluation of the F/B method. The following series of projects is designed to continue development of the F/B method in a way that will be responsive to suggestions of the Subcommittee. (Note: work of a general nature that was suggested by the Subcommittee appears in Section A, above.) Future projects in this section are contingent upon receiving a "decision to continue" from OAQPS in the Spring of 1981. This decision will be based upon results of the technical review project (Project B-2) and other evaluations.

Completed Projects

There were a number of projects and papers completed on the F/B method prior to institutionalizing a formal OAQPS risk program. These are not delineated here. In 1980, F/B had a paper published that discusses their view concerning the nature of probability estimates suitable for regulatory risk assessment. This paper is: Thomas B. Feagans and William F. Biller. "Fuzzy Concepts in the Analysis of Public Health Risks," pp. 391-404 in: Paul P. Wang and S. K. Chang (eds.), Fuzzy Sets (New York: Plenum Press, 1980). In addition, both gentlemen presented papers related to specific aspects of the F/B method at meetings and conferences during 1980.

On-Going Project

B-1. Complete "A General Method for Assessing Health Risks Associated With Primary National Ambient Air Quality Standards."

This project essentially involves completing a major report describing the F/B approach to regulatory risk assessment. The report is scheduled for completion in March 1981.

Future Projects

B-2. Conduct a technical review of the F/B approach.

Four-to-six-experts in probability theory, risk analysis, and air quality analysis will be engaged to provide an in-depth technical review of the F/B approach. Their review will focus on the report produced in Project B-1. The technical review will occur in late April and early May, 1981. The document will be released publically simultaneous with this review. Results of the review will be presented to the SAB Subcommittee on Health Risk Assessment in May 1981.

OAQPS will evaluate comments received from the reviewers, the SAB Subcommittee, and the public. Based upon this evaluation, OAQPS will make a decision whether or not to continue developing the F/B approach to regulatory risk assessment. The remaining projects in this category are contingent upon receiving a decision to continue from OAQPS management.

B-3. Publish peer-reviewed papers explaining various aspects of the F/B method.

Mr. Feagans and Dr. Biller, with assistance from other EPA staff, will write and submit papers on the F/B method to peer-reviewed journals. This is an on-going intermittent project starting after the OAQPS evaluation is completed.

B-4. Apply the F/B approach to CO.

This project involves a full scale application of the F/B approach to carbon monoxide. It includes modules to: select experts, conduct panel meetings to exchange health information, encode experts, develop probabilistic models and software, and produce "benchmark" and "headcount" risk estimates. The application will generate a more complete risk picture than was possible with the initial application to ozone; outputs will include both headcount and benchmark risk estimates.

This project will be undertaken by Mr. Feagans and Dr Biller, with assistance from Dr. Wallsten and PEDCo Environmental. The project will begin in early 1982 and should take about six months to complete. The main output is a report that will be used by OAQPS to determine if the F/B approach will be further considered in Project A-6.

C. DEVELOPMENT OF ALTERNATIVE RISK ASSESSMENT METHODS

The SAB Subcommittee recommended that OAQPS formulate alternative quantitative approaches for assessing risk. The Subcommittee wanted the Office to involve a number of outside experts in developing these alternative methodologies. This part of the risk assessment program is intended to address those recommendations.

Completed Project

- The first project undertaken in developing alternative risk assessment approaches was a highly-interactive effort involving six distinguished researchers and groups. Purposes of the project were to (1) elicit and develop conceptual risk assessment approaches suitable for use by EPA in setting ambient air standards, and (2) narrow down these alternatives to 2-3 approaches for further development.

  A two-day open meeting was held to discuss risk assessment issues and how they could be formally addressed. Information was shared and views were exchanged; audience participation was encouraged and obtained. Another two-day public meeting was held by the SAB Subcommittee at project conclusion. All six groups presented and defended their approaches. Again, audience participation was high.

  The SAB Subcommittee formally reviewed the conceptual approaches and stated that two, and possibly another, warrented further development. The SAB review is contained in: "Approaches to Health Risk Assessment for Alternative National Ambient Air Quality Standards: A Report of the SAB Subcommittee on Health Risk Assessment." Washington, D.C.: U.S. Environmental Protection Agency, Science Advisory Board, 1980.

  The conceptual approaches were described in the following papers that will be combined by OAQPS into a single EPA report (due date from the printer: June 1981).

  Richard deNeufville and Marie-Elisabeth Pate.
  "A Conceptual Risk Assessment Procedure" (1980).

  Baruch Fischhoff and Chris Whipple.
  "Assessing Health Risks Associated with Ambient Air Quality Standards" (1980).

  H.O. Hartley, K.G. Manton, and M.A. Woodbury.
  "Estimation of Risk of Adverse Health Effects Associated with Air Quality Standards for Pollutants" (1980).

M.W. Merkhofer.
"A Procedure Based on Decision Analysis for Assessing the Health Risk Associated with Alternative Ambient Air Quality Standards" (1980).

Howard Raiffa and Richard Zeckhauser.
"Reporting of Uncertainties in Risk Analysis" (1981).

Robert L. Winkler and Rakesh K. Sarin.
"A Risk Assessment Methodology for Environmental Pollutants" (1980).

Future Projects

C-1. Development of alternative risk assessment methods.

This project includes tasks designed to both further develop and apply the two conceptual approaches found by the SAB Subcommittee and OAQPS to be most promising for NAAQS purposes. The pollutant of interest will be either CO or O3. All exposure work needed for the assessment will be done by the SASD exposure team (see Projects A-2 and A-3).

Contractors for this project will be SRI International, who will develop Dr. M.W. Merkhofer's risk assessment approach, and Woodward-Clyde Consultants, who will develop Drs. R. Winkler and R. Sarin's approach. (The two professors are consultants to Woodward-Clyde; Dr. Ralph Keeney of the firm will also be involved in the effort.)

Each group will describe its methodology and the rationale behind it in a report; it will also describe output measures used and all inputs required. Technical appendices to the report will document algorithms and statistics used in the method, as well as its application to a specific pollutant. Finally, both research groups will evaluate their method for suitability and usefulness in a formal decision analysis approach to setting ambient air standards. (The final report is due in January 1982).

The approaches will be presented in an open session of the SAB Subcommittee. EPA will invite the Subcommittee to make recommendations concerning which method(s) should be evaluated further by Argonne National Laboratory in Project A-6.

This concludes the risk assessment part of the risk analysis program.

D. RISK EVALUATION

The main objectives of the risk evaluation portion of the program are to (1) determine the extent to which formal evaluation analysis can be used in the NAAQS-setting process and (2) develop a feasible risk evaluation approach that is compatible both with whatever risk assessment method(s) is (are) chosen for use in estimating health risks and with EPA's air programs mission.

Specific projects already completed and planned for to advance risk evaluation of NAAQS follow:

Completed Projects

There were two small projects undertaken in 1980 relating to risk evaluation. Both were with Dr. Ralph L. Keeney of Woodward-Clyde Consultants. The first involved an evaluation of how currently-generated information could best be analyzed and presented to EPA decision-makers. The emphasis was on the use of decision analytic aids in EPA's NAAQS standard-setting process.

The second project involved a trial application of decision analysis methodology to a specific NAAQS pollutant: carbon monoxide. The application was quite limited due to bugetary constraints.

Results of the two projects are contained in:

Ralph L. Keeney.
"A General Decision Framework for Primary NAAQS-Setting decisions" (1980).

Ralph L. Keeney and Vladimir M. Ozernoy.
An Illustrative Analysis of Ambient Carbon Monoxide Standards. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, 1980.

On-Going Projects

D-1. Describe existing conceptual approaches to risk evaluation.

This project involves a critical literature search of conceptually distinct ways posited to evaluate policy and societal implications of risk associated with governmental programs. The project addresses such issues as choosing an adequate margin of safety, using risk-benefit analysis (or one of its variants), and explicitly evaluating a decision-maker's trade-off function. The project will be undertaken by Dr. James Vaupel of Duke University. Output will be a report describing findings. (This may be combined with the report developed for Project D-2.) The due date is August 1981.

D-2. Describe existing risk evaluation/decision analytic approaches used by federal agencies.

A literature search and site visits will be undertaken to determine what risk evaluation methods are currently being used by Federal agencies involved with the Interagency Regulatory Liaison Group. In addition, the extent to which risk evaluation is part of a broader decision analytic framework will be investigated and described. Findings will be published in an OAQPS report (which may be combined with the D-1 report). Report due date is August, 1981.

Future Projects

D-3. Develop criteria/constraints on the use of risk evaluation in the NAAQS-setting process.

This project essentially involves developing a "White Paper" describing what types of evaluation criteria are appropriate to use in setting air standards. It will also discuss legal/procedural/resource constraints facing the Agency in evaluating risk associated with alternative air standards. This is an in-house project with extensive staff involvement. It will begin after revisions to the Clean Air Act have been made, which is expected to be in the Fall of 1981.

D-4. Develop a risk evaluation method for appraising alternative NAAQS standards.

This project is designed to develop an integrated risk evaluation/decision analytic method suitable for use with the risk assessment method(s) found to be most appropriate for NAAQS pollutants. Both quantitative and qualitative aspects of decision analysis will be investigated as possible risk evaluation techniques. The project will be done under contract, and must be coordinated closely with Argonne's work on Project A-6.

The impact of using a risk evaluation methodology on other EPA programs and offices will also be addressed. Thus, SASD will cooperate closely with OPE and other interested Agency offices on this project.

Output will be a report. The project is slated to start in late-1982 and be completed in mid-1983.

This concludes the risk evaluation portion of the risk analysis program.

For more information, contact:

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U.S. Environmental Protection Agency
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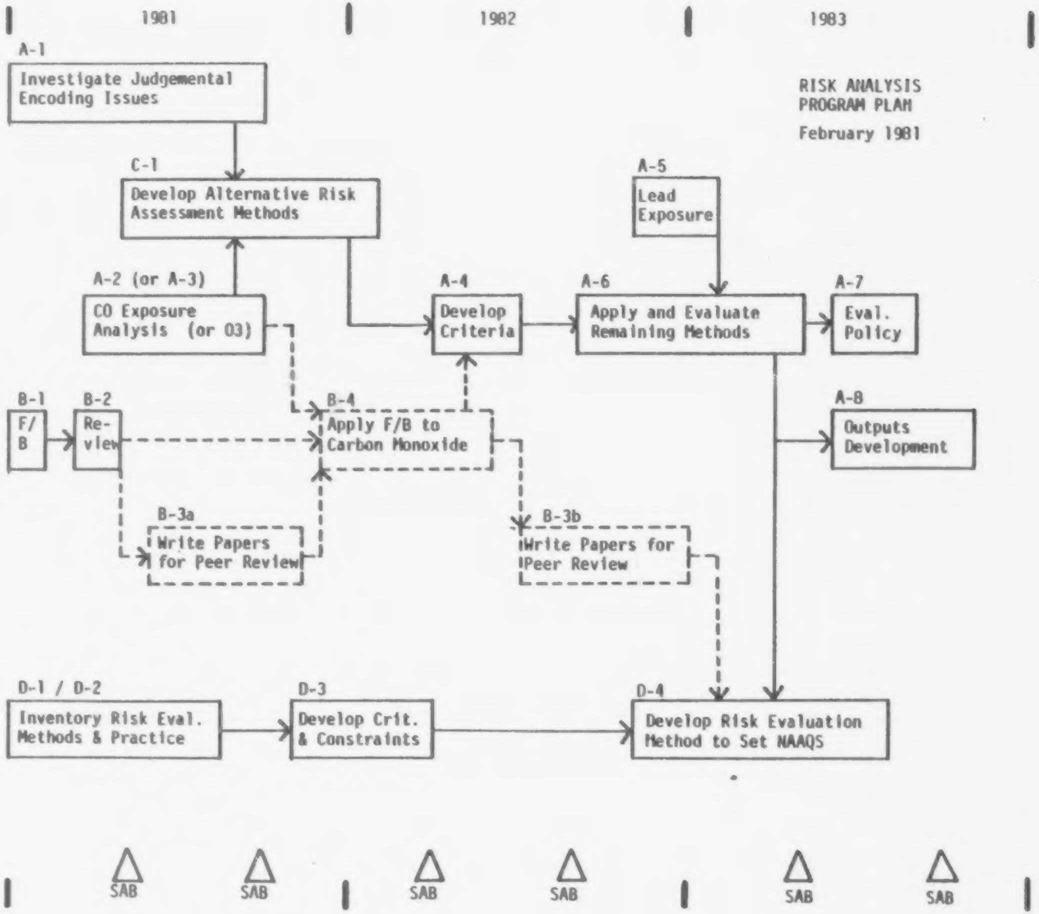

RISK ANALYSIS
PROGRAM PLAN
February 1981

1981
1982
1983
A-1
Investigate Judgemental Encoding Issues
C-1
Develop Alternative Risk Assessment Methods
A-2 (or A-3)
CO Exposure Analysis (or 03)
A-4
Develop Criteria
A-5
Lead Exposure
A-6
Apply and Evaluate Remaining Methods
A-7
Eval. Policy
A-8
Outputs Development
B-1
F/B
B-2
Re-view
B-4
Apply F/B to Carbon Monoxide
B-3a
Write Papers for Peer Review
B-3b
Write Papers for Peer Review
D-1 / D-2
Inventory Risk Eval. Methods & Practice
D-3
Develop Crit. & Constraints
D-4
Develop Risk Evaluation Method to Set NAAQS
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APR 29 1981

Guidance on Response to Contamination Detected in the Ground Water Supply Survey

The Office of Drinking Water (ODW) in cooperation with the States, is undertaking a national survey on the quality of ground water (The Ground Water Supply Survey). The survey is underway and it will include sampling and analyses of drinking water derived from ground water at 1,000 locations in the United States. The analytical procedures will identify purgeable halogenated organic chemicals such as the chlorinated solvents (e.g., trichloroethylene) and non-halogenated volatile aromatic hydrocarbons (e.g., benzene). Out of a total of 1,000 samples, 500 will reflect a random selection of sites. Another 500 sites will be selected by the States and EPA as areas with ground waters potentially contaminated by organic chemicals. Among other things, we will use this latter group to determine characteristics that may allow better predictability for locating contaminated sites.

The purpose of this guidance is to assist the States or other appropriate authorities in determining the type of response that they should make in a particular case where organic chemicals are detected in a sample from the survey. We are providing a perspective on the relative health significance of various concentrations of chemicals that might be detected, as well as information on the type and relative urgency of possible follow-up actions to protect the public. Depending upon the circumstances of a contamination incident, one of the appropriate actions could be to identify and control the source of contamination. There are ample legislative authorities for this purpose under existing pollution control legislation. However, such activities are not dealt within this guidance.

Two tables are attached which list a number of chemicals which may be detected in this investigation. The concentration levels in Table 1 reflect a scientific judgment on the health effects of specific chemicals using non-carcinogenic toxicity data. The values in Table 2 are based on the carcinogenic aspects of the chemicals. For each chemical, there are four concentration values listed, shown as categories I-IV. The categories represent differing degrees of concern and differing types of action that should be considered depending upon the level of concentration of the contaminants. Concentrations values are not listed for several of the chemicals; the Office of Drinking Water is continuing to collect and evaluate the available data and the tables will be revised as more information becomes available.

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When an unregulated chemical is detected in drinking water, the EPA regional drinking water office should be contacted if assistance is required by the State. The Office of Drinking Water's Criteria and Standards Division (CSD) is also available to provide advisory assistance for case-by-case decisions. In instances where there is not a concentration value listed in the table, or an unlisted chemical has been detected, the CSD should be notified and every effort will be made to provide immediate recommendations. The person to contact is Dr. William Lappenbusch, phone 202-472-6820.

Both tables should be referred to when a chemical has been detected. For the same chemical, the concentration levels listed in the two tables in the same category may differ. This is due to the fact that each table was generated from a separate data base which used different toxicological end points. For certain chemicals, there may be questions as to which table to use and what level of concern is appropriate. Examples of the use of Tables 1 and 2 are given below:

- If trichloroethylene was found in drinking water and assumed to be consumed for approximately a year (chronic exposure) before remedial action was implemented, 0.075 mg/l (Table 1) would not pose significant health risks to the most sensitive members of the population. On the other hand, should the duration of the exposure be anticipated to last several years, then an attempt to reach concentrations between 0.005-0.045 mg/l (Table 2 would be used which is based upon carcinogenicity) may be more prudent depending upon population exposure, treatment and associated costs.

- In some cases, like carbon tetrachloride, the excess cancer risk number (0.045 mg/l for 10^{-5} lifetime risk) is provided but no chronic non-carcinogenic number exists since existing dose/response data are not available for the proper duration to allow for such an estimation. In that case, a reasonable approach would be to use the more conservative values in Table 2. In such instances the State should contact the CSD and advice will be provided based upon the specific toxicity of the chemical, the length of exposure, and other factors.

- In the situation where several chemicals are detected in the same water sample, a reasonable approach would be to treat the carcinogenic risks as additive (Table 2). For example:

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- If trichloroethylene (TCE) and tetrachloroethylene (PCE) were found in drinking water at concentrations of 0.045 mg/l and 0.035 mg/l, respectively, the excess cancer risk over a lifetime may be estimated as follows:

TCE		1×10^{-5}
PCE	+	1×10^{-5}
Total		2×10^{-5}

  The total risk for these elements would still be in Category II of Table 2.

- If TCE and PCE were found at concentrations of 0.225 mg/l (5 x 0.045 mg/l) and 0.210 mg/l (6 x 0.035 mg/l), respectively, the excess cancer risk may be calculated as follows:

  Step 1

  TCE $5(1 \times 10^{-5}) = 5 \times 10^{-5}$
  PCE $6(1 \times 10^{-5}) = 6 \times 10^{-5}$

  Step 2

TCE		5×10^{-5}
PCE	+	6×10^{-5}
		11×10^{-5} or 1.1×10^{-4}

- The total risk for these chemicals would now be in Category I of Table 2.

Addition of the two or more calculated risks is obviously simplistic, but at least it provides a reasonably consistent decision process, assuming that other factors are not mitigating. However, there is a potential for synergestic effects and simple addition of the risks of each carcinogen could thereby underestimate the overall risk.

For mixtures of non-carcinogens, none of which exceed the concentrations in Category III of Table 1, the CSD should be consulted.

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In the case of trihalomethanes (THM) being detected, an MCL of 0.10 mg/l is included in the National Interim Primary Drinking Water Regulations. This value was derived taking economics and technical feasibility into account and levels above the MCL should be handled according to normal State procedures. Since THMs are a by-product of the chlorination process, detection of THMs in the raw water would indicate contamination of the ground water; depending upon the circumstances at each site, the source of contamination should be identified and controlled.

Table 1 - Acute and Chronic Toxicity Factors

The values in Table 1 are computed from acute, short-term and chronic toxicity considerations (Categories I, II, III, respectively). Category IV values are based upon taste and odor detection data and do not include toxicity factors. This compilation was derived from a number of sources, including Office of Drinking Water SNARLs (Suggested No Adverse Response Levels), recommendations from the National Academy of Sciences (NAS) report, Drinking Water and Health (1977), and other literature as evaluated by the CSD. Those concentration levels in Table 1 which are not identified by asterisks were prepared by the ODW. The other levels, identified by asterisks, were calculated from values delineated in the National Academy of Sciences report or EPA Ambient Water Quality Criteria Documents (1980).

The categories, values and suggested follow-up responses for Table 1 are as follows:

Category I. Acute Concern Level, High Risk

This is an unusually high concentration level that may introduce risks from brief exposure (one or a few days) in high risk individuals.

Even though large safety margins normally are included in the computed values, emergency type action should be taken.

- Immediate resampling and verification of the concentration levels.
- Identification of the source of contamination and rectify problem, if possible.
- A ban on water consumption for drinking and cooking.

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Category II. Subacute Concern Level

This is a high concentration level but the concentrations are about 1/10 of Category I. Action should be immediate.

- Immediate resampling and verification of the concentration levels is essential.
- Identification of the source of contamination and rectify problem if possible.
- A ban on water consumption for drinking and cooking should be considered, especially if the level is likely to persist for more than approximately 10 days.
- Alternate sources should be seriously considered as well as treatment to reduce the levels.

Category III. Chronic Exposure Concern Level

Exposure near this level for an extended period would represent a relatively low risk situation, especially if the chemical is not bioaccumulative.

As soon as practical, the following actions are appropriate:

- Resample and verify concentration levels.
- Identification of the source of contamination and rectify problem if possible.
- Initiate long term surveillance monitoring, as concentrations may increase in the future.
- Alterations in the treatment process or consideration of alternate sources.

Category IV. Taste and Odor Detection

Detection by taste or odor usually causes public rejection of the water for drinking and cooking uses and may indicate severe contamination and a potential imminent health risk (e.g. the case of benzene where the taste and odor threshold is 2 mg/l but the acute number is 0.35 mg/l). In some cases, the taste and odor levels listed in Table I are lower than the Category I, II or III levels and therefore provide protection as in the case of 1,4-dichlorobenzene. In either case, the cause should be identified and corrective action taken.

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Table 2 - Potential Carcinogenic Risks

The concentration levels in Table 2 for the potential carcinogens are derived from risk extrapolations from the NAS's Drinking Water and Health report, or from EPA's Carcinogen Assessment Group (CAG). The Crump multi-stage model was used, and the computed incremental risks assume lifetime (70 years) consumption of 2 liters of water per day containing the chemical at the indicated level. No additional exposures to the chemicals other than in drinking water are considered in these values.

Both the NAS and CAG numbers were included in this table to indicate that risk estimation represents a scientific judgment as to the health risk resulting from a chemical's presence in water. The two values are not identical because different data and parameters were included in each model. For example, the trichloroethylene concentrations associated with a lifetime exposure and cancer risk of 10^{-5} via the CAG and the NAS at the 95% confidence limit are 0.028 mg/l and 0.045 mg/l, respectively. However, for the majority of chemicals, the two values are fairly consistent and indicate the general range of concentration levels that would elicit a response action.

For the purpose of providing advice on the types of follow-up actions that would be recommended when certain levels of contaminants are found, four categories were arbitrarily selected as a means of indicating relative concern levels based on the lifetime risks at these concentrations. It is emphasized that these values are projected from lifetime exposure, and therefore, response actions usually would not be commensurate with risks from relatively short exposures.

The categories, excess cancer risk levels and suggested responses for Table 2 are as follows:

Category I. Projected Lifetime Exposure Risk Larger than 1 in 10,000 (> 10^{-4})

This is a situation which would represent a high priority; because of the relatively high concentrations in some cases, immediate action may be warranted (non-carcinogenic vs. carcinogenic data should be compared and other factors such as population exposure, duration of exposure, treatment and associated costs should be evaluated).

- Resample and verify concentration levels.

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- Identify source and rectify problem if possible.
- Consideration given to:
    - switching to alternate source
    - banning water for cooking/drinking purposes

Category II. Projected Lifetime Exposure Risk Between 1 in 10,000 and 1 in 100,000 (10^{-4} - 10^{-5})

This is a situation that would represent a high priority concern, but somewhat less than Category I and should generate expeditious action.

- Resample and verify concentration levels.
- Identify source and rectify problem, if possible.
- Take steps to reduce human exposure.
- Initiate frequent monitoring.

Category III. Projected Lifetime Exposure Risk Between 1 in 100,000 and 1 in 1,000,000 (10^{-5} - 10^{-6})

This is a relatively lower risk situation that would not require accelerated action. Action should be taken as resources permit.

- Resample and verify concentration levels.
- Identify source and rectify problem if possible.
- Long range surveillance monitoring should be considered because concentrations could increase in the future.

Actions should reflect whether the concentration value detected is at the higher or lower part of the risk range.

Category IV. Projected Risk Less than 1 in 1,000,000 ($< 10^{-6}$)

This is a concentration level that represents a negligible risk; this situation should be considered to be low priority.

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- Depending upon available resources:
  - Resample and verify concentration levels.
  - Investigation of the source of contamination and possible rectification of problem.
  - Long range surveillance monitoring should be considered because concentrations could increase in the future.

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Table 1: Concentration Levels (mg/l) for Select Chemicals, Based on Toxicity Other Than Carcinogenesis

Chemical Name	Acute I	Short-term II	Chronic III	Taste and Odor IV
Benzene	0.35			2
Carbon Tetrachloride	0.2	0.02		
Chlorobenzene			0.072**	0.1
Dichlorodifluoromethane	100*	43*	1.6*	
1,2-Dibromo-3-chloropropane			0.00005	
1,2-Dichlorobenzene				0.01
1,3-Dichlorobenzene				0.02
1,4-Dichlorobenzene			0.13*	0.0003
1,2-Dichloroethane				29
1,2-Dichloropropane				.0014
Ethylbenzene				0.1
Methylene Chloride	13	1.3	0.15	
Isopropylbenzene				0.1
Styrene			1.3*	0.05
Tetrachloroethylene	2.3	0.18	0.02	0.3
Toluene	120*	10*	0.10*	1.0
1,1,1-Trichloroethane	140*	20*	1.1*	
Trichloroethylene	2	0.2	0.075	0.5
Trichlorofluoromethane	25*	2.2*		
m-Xylene	6.1*	3.2*		1.1
o-Xylene mixture				1.8
p-Xylene				0.5

* = Numbers calculated from NAS Drinking Water and Health for a 10 kg child consuming 1 liter water/day.

** = Numbers calculated from Water Quality Criteria for a 10 kg child consuming 1 liter water/day.

Studies are underway to obtain values for the following chemicals:

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Bromobenzene
o-Chlorotoluene
p-Chlorotoluene
1,1-Dichloroethylene
1,1-Dichloroethane
Cis-1,2-Dichloroethylene
Trans-1,2-Dichloroethylene
N-Propylbenzene
1,1,2,2-Tetrachloroethylene
1,1,2,2-Tetrachloroethane
1,1,2-Trichloroethane

Table 2: Concentration Levels (mg/l) for Select Potential Carcinogens

Carcinogens		I (> 10^{-4})	II (10^{-5} to 10^{-4})	III (10^{-6} to 10^{-5})	IV (< 10^{-6})
Carbon tetrachloride	NAS*	.45	.045-.45	.0045-.045	.0045
	CAG†	.04	.004-.04	.0004-.004	.0004
1,2-Dichloroethane	NAS	.071	.0071-.071	.00071-.0071	.00071
	CAG	.095	.0095-.095	.00095-.0095	.00095
1,2-Dibromoethane	NAS	.0055	.00055-.0055	.000055-.00055	.000055
	CAG				
Tetrachloroethylene	NAS	.35	.035-.350	.0035-.035	.0035
	CAG	.09	.009-.09	.0009-.009	.0009
Trichloroethylene	NAS	.45	.045-.45	.0045-.045	.0045
	CAG	.28	.028-.28	.0028-.028	.0028
1,2-Dibromo-3-chloropropane	NAS	(TO BE DETERMINED)			
	CAG				
1,1,2-Trichloroethane	NAS				
	CAG	.061	.0061-.061	.00061-.0061	.0061
1,1,2,2-Tetrachloroethane	NAS				
	CAG	.017	.0017-.017	.00017-.0017	.00017
Vinyl chloride	NAS	.1	.01-.1	.001-.01	.001
	CAG	.2	.02-.2	.002-.02	.002
Benzene	NAS				
	CAG	.067	.0067-.067	.00067-.0067	.00067

* NAS: Calculated by EPA/ODW using carcinogenic risks specified by the Safe Drinking Water Committee, National Academy of Sciences.

† CAG: Calculated by EPA/ODW using carcinogenic risks specified by EPA's Carcinogen Assessment Group. Modification made to reflect carcinogenic risks associated with lifetime exposure to drinking water. The contribution from fish/seafood has been excluded.

Cancer Policy

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

JUL 22 1981

OFFICE OF
WATER

SUBJECT: Request for Review of Draft Cancer Policy Statment

FROM: Arnold M. Kuzmack, Director Arnold M. Kuzmack
Office of Program Development & Evaluation, ODW (WH-550)

Elizabeth L. Anderson, Director
Office of Health & Environmental Assessment (RD-689)

TO: TSPC

This memorandum reports on progress by the Cancer Policy Task Force in its effort to develop meaningful statements of principles for use in controlling human exposures to carcinogens. Attached for your consideration at the July 31 meeting is a draft statement on cancer policy worked out by the task force. As the draft shows, and the discussion below explains, in various areas the task force did not reach consensus. In one of the two areas in which policy principles were developed, two options are provided because the task force was unable to agree. Individual members do not support certain portions of the reasoning, or certain emphases. However, all agree that it is appropriate to submit this draft to you at this time for your review and further guidance.

Specifically, we solicit your views on the following:

- The policies laid out in Parts III and IV.
- Other areas in which you wish us to attempt to articulate policy or decision rules (or alternatives).
- The background generalities and lines of reasoning in the draft.
- Is the extent of detail and explanation about right, too much or too little?
- Is the statement as a whole both useful and appropriate to the broad range of Agency programs and activities? (See two unresolved issues discussed at end of this memo).

In addition to discussion at the TSPC, we would be most grateful if individual members would provide us with written responses to help us in coming up with a final draft.

We were charged with developing recommendations on two questions:

(1) The weight to be given to individual risk vs. total population risk in Agency decision making, and

(2) Whether the Agency should adopt a target risk level for control of carcinogens.

It proved exceedingly difficult to articulate a useful set of generally applicable principles as a result of the differences in program experience and perspective that one would expect in a controversial area such as this. In addition, the differing decision criteria and requirements of EPA's authorizing statutes and many extra-statutory decision criteria tend to confound attempts to formulate simple, generally applicable principles based on cancer risk alone.

The attached draft is thus limited in scope, and at this stage hedged about with conditions, despite the very substantial amount of effort devoted by the members of the task force. The task force addressed the first of the two question only in the context of setting priorities for investigation of potential actions, since we did not think we could make a useful general statement about how to make final decisions in situations where a small number of people are exposed to a high individual risk. In this context, two alternative statements are offered (see pages 7 and 8. Option A indicates that the Agency will emphasize situations of high total population risk and will generally assign a lower priority for Federal regulatory action to situations of low total population risk and high individual risk. Option B give equivalent weight to both high individual risk and high total population risk. The group was not able to reach a consensus on which of these options to recommend and was about evenly divided on this issue. Some group members expressed reservations about the desirability of including the numerical illustration shown as part of Option B and felt strongly that the general statement may be much more acceptable without the specific numerical references. Others felt that without numerical examples the usefulness of the statement was largely vitiated.

With respect to the second question, the group considered an array of options involving risk targets, floors, and ceilings, both as single numbers and as ranges. The task force consensus was that the general positions on page 10 is supportable and that any statement which went further would not be supportable. The task force rejected the notion of a risk target in favor of a risk "floor", that is, a risk level below which the Agency generally would not go in controlling carcinogens. We felt that we could not give guidance that would be both specific and generally applicable as to how close Agency actions would get to the "floor". We have tried to state very clearly that the level is not a target and that particular actions will frequently result in a higher level of risk after control. While the task force reached a consensus on the recommendation of a risk floor, some group members disagree with the final paragraph of the draft and would delete as irrelevant and as conveying an unwise impression the statement that the risk level after control would "sometimes be much greater" than the floor. There was also a general feeling that this language would be much less acceptable if the level at which the floor was set, 10^{-6}, were to be changed to a much higher level such as 10^{-5} or 10^{-4}.

Two additional important issues were addressed by the task force only at the end of our last meeting, and hence were not considered in sufficient depth to provide a basis for resolving them in the draft submitted with this memo. We solicit your guidance on these issues. The first issue is whether the two policies stated in the draft should apply to all Agency control actions dealing with the hazards of carcinogens, or just to decisions on prospective controls. Should they apply not only to the prescribing standards or practices before environmental discharges have taken place, or also to the steps to be taken to deal with discharges to the environment after they have taken place and require cleanup or other action? Should these policies be extended to guidance as to how to deal with existing environmental problems (e.g., SNARLs), to advice to State officials in emergencies, to cleanup decisions under Superfund, to enforcement actions? Or should they be limited to regulatory-type standards typified by effluent guidelines, drinking water standards, and air new source performane standards? Part of the group felt there was no good reason not to apply these policies to all such actions; others felt that it was unwise, or at least not sufficiently thought through to recommend. The draft as submitted to you generally applies to all control actions, both preventive and cleanup, but we agreed to flag the issue for your guidance.

The second issue is whether the policy should apply to carcinogens one-by-one or should apply to the mixture of carcinogens found in the industrial source or other problem to controlled. Some task force members felt that where a given action would control exposure to several carcinogens, the two policy positions should apply to the aggregate risk of cancer presented by the several carcinogens. Others felt that the policy positions had been prepared on an assumption of dealing with a single carcinogen. While there was a tendency toward the former position, it was agreed that the issue deserved a more careful consideration with respect to the two policy statements.

The task force earnestly solicits your comments on the issues raised above, the draft policy statement, and the general direction of further effort.

EPA POLICY GUIDELINES ON THE CONTROL OF CARCINOGENS

I. INTRODUCTION

Cancer is a major problem from both a health and economic point of view. Chemicals in the environment cause an unknown but perhaps substantial number of cancer cases, and the identification of known and suspected chemical carcinogens is increasing with the passage of time. In order to ensure that the Agency is according an appropriate priority to carcinogenic chemicals for study and control, EPA has formulated the general approach set forth in Section III below. To set a point beyond which control of carcinogens is generally not appropriate, the Agency has developed the statement in Section IV.

EPA is authorized to control carcinogens under a number of statutory provisions. Some of the statutory provisions make risk reduction the predominant decision-making criterion; some focus on technology to reduce risks; and others require decision-makers to balance the costs and benefits of possible regulations (or the risks of a chemical and the benefits of its use). These different statutory provisions are among the reasons that the regulation of carcinogens is a highly complex matter.

One important issue that has arisen in connection with the control of carcinogens is whether and how to use quantitative risk estimates.

In 1976, EPA published interim guidelines on cancer risk assessment[1] which stated, among other things, the Agency's intention to use quantitative risk estimates when making decisions on chemical carcinogens under certain statutory provisions. These guidelines remain in effect. Since their publication, the Agency has had a great deal of experience in the use of quantitative risk estimates and now believes that it is appropriate to make some further general policy statements on the use of these risk estimates.

Therefore, in order to improve the decisionmaking process and make the control of carcinogens more consistent, the Agency has established benchmarks to be used in conjunction with other relevant factors in the control of carcinogens. They are intended to achieve reasonable goals consistent with the requirements of the various statutes which the Agency implements and the principles of E.O. 12291.

II. NATURE OF CANCER RISK ESTIMATES

Cancer risk estimates contain two basic components: an evaluation of the weight of evidence that a chemical is a carcinogen, and, assuming that it is a carcinogen, an estimate of the magnitude of the cancer burden resulting from it. The latter estimate (the quantitative risk estimate) is derived from a chemical's potency as a carcinogen and the numbers of people exposed to various levels of the chemical.

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1. 41 FR 21402, May 25, 1976.

The analysis of human exposure will involve an estimate of the size of the exposed population and the level of exposure. The analysis may also consider such factors as exposure sources, routes, and conditions; the duration, frequency, and intensity of exposure; and the relevant characteristics (e.g., age, sex, and health) of the exposed population.[2]

The assessment of cancer risk resulting from a given exposure is characterized by great uncertainty, because of frequent limitations in the evidence of carcinogenicity and because of the necessity of extrapolating results from experimental animals to humans, from high doses to low doses, and (if human evidence is available) from one population group to another. The evidence of a chemical's carcinogenicity and of its potency will, in most cases, consist principally of the results of animal tests performed at doses much higher than those to which humans are exposed in the environment. In a few cases, adequate epidemiological studies may also be available, but these also usually involve exposures at high

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2. Recognizing the complexity of estimating exposure, EPA is preparing procedural guidelines for the preparation of exposure assessments. The guidelines are intended to provide a general approach and framework for performing human and nonhuman exposure assessments, as well as to promote consistency, to the extent possible, in the data used, in the assumptions made, and in the presentation of ranges of estimates.

levels. In order to estimate the likely effect of exposure at much lower levels, perhaps by several orders of magnitude, mathematical extrapolation must be used.[3]

Any assessment of the effects of low exposures to carcinogens based on animal tests necessarily involves an extrapolation whose accuracy cannot be verified by existing scientific data. Accordingly, quantitative risk estimates are subject to a wide range of uncertainty. However, the only alternative to using these estimates is to attempt to make decisions without any estimate of the magnitude of the effects the Agency is attempting to control; this alternative is clearly much less desirable. Hence, despite these uncertainties, it is necessary to use estimates of the magnitude of the cancer risk along with other factors to make sensible regulatory decisions.

Because of the importance of many factors other than the degree of cancer risk, EPA may (to the extent permitted by law) rely on both risk and other considerations in reaching decisions on whether to attack a particular problem involving cancer risk, whether to implement controls for specific

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3. Because it is not possible to identify a level of exposure to a carcinogen that would totally eliminate risk, the mathematical extrapolation models used by EPA assume that there is no threshold to the action of a carcinogen. The models assume, further, that the probability of response is proportional to the dose at low dose levels.

for specific sources and, where the pollutant is emitted from many sources, which sources to control and in what order. Although cancer risk is the focus in this statement, other factors may be more important in a particular case. Other factors that may be considered along with the reduction in cancer risk include:

- feasibility of control;
- control costs;
- benefits of the compound or activity that may be lost through control;
- presence of other toxic pollutant emissions;
- availability of regulatory resources;
- alternatives to federal control action (e.g., voluntary or state action, technical assistance, and provision of information);
- status of implementation of other regulatory programs;
- other health and environmental effects;
- multimedia (e.g., air, water, solid waste) sources and cross-media impacts;
- impact on the controlled industry and the economy; and
- legislative authorities and constraints.

When pollutants enter the environment via multiple routes or when exposure occurs from more than one medium, consideration will be given to implementing a coordinated multimedia, rather than a single-medium, control program since this may result in a more effective and econmical control strategy.

III. POLICY ON USE OF CANCER RISK ESTIMATES IN ESTABLISHING PRIORITIES FOR CONTROL ACTION

EPA is concerned about cancer risk to individuals because it is inequitable for some individuals to bear very large risks, often without choice and with little or no compensating benefits, even if other members of society benefit from the risk-producing activity. EPA is also concerned about the magnitude of total (nationwide) risk to the population as a result of exposure to a suspected carcinogen.[4/] Therefore, in establishing priorities to control exposure to carcinogens in the environment (whether by issuing regulations or other actions), EPA will consider both the magnitude of the risk to individuals and the total population risk.

Since decisions about the degree of control will depend on the costs and cancer risks as well as the requirements of the governing statute, a high priority for control does not necessarily imply that EPA

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[4] Individual risk, as used in this statement, is the projected probability that an individual exposed to a given level of a carcinogen (or mixture of carcinogens) will develop cancer as a result of that exposure over the course of a lifetime. It is expressed as a probability,, e.g., a probability of one in a thousand (10^{-3}) or one in a million (10^{-6}). Total population risk is the expected number of cases per year projected to result from a given distribution of exposures and depends on both the levels of individuals risk in the population and the number of people subjected to those risks.

will take action to control the source. "Establishing priorities" means establishing the order in which EPA will examine the risks, costs, and benefits of control alternatives for various sources of carcinogens and the level of its own resources it will devote to this task. Hence, a "high priority" only means that EPA will consider a given control opportunity before others of lower priority. After examining the costs and benefits of control options, EPA may decide to proceed with national regulation or other Federal action, to encourage action by state or local government, to take other non-regulatory action, or to take no action at all because there is no feasible control alternative that results in significant risk reduction at reasonable costs. In some situations in which an exposure is limited to one or a few geographical locations, or is otherwise amenable to reduction by means other than Federal action, EPA may encourage voluntary actions or actions by State or local authorities and may use its resources to encourage such action.

Because data are generally limited at the early stages of decision-making, EPA will not do a detailed quantitative analysis in order to establish priorities. Moreover, while EPA will use readily available information, it will not necessarily delay priority setting to await additional analysis.

OPTION A

In general, EPA will set priorities for the control of carcinogens based principally on nationwide cancer effects. The Agency may also [illegible] high priority for national action to protect subpopulation groups at high risk, e.g., lifetime risks of 10^{-3} or greater. However, EPA will usually encourage action at the State or local level rather than issuing national regulations if individual risks are high but the number of people exposed (and consequently total population risk) is very small and geographically localized.

OPTION B

In general, high priority (as described above) may be supported either by a high level of estimated risk of cancer to small groups of individuals at greatest risk or by high total population risk. A combination of moderately high risk to individuals and moderately high total population risk may also support a high priority. While numerical levels of risk to define levels of priority will not be fixed, some examples may help. If, for instance, a relatively small number of people -- perhaps a few hundred -- were estimated to be subjected to a lifetime cancer risk in the vicinity of one in 100 (10^{-2}) to one in 1,000 (10^{-3}) by a given exposure, this could be the basis for high priority for considering action to reduce the risk caused by that exposure. On the other hand, if 10 million people were estimated to be subjected to an increased cancer risk of in the vicinty of one in 10,000 (10^{-4}) to one in 100,000 (10^{-5}), this also could be a basis for high priority.

IV. POLICY ON EXTENT OF REDUCTION OF CANCER RISK

Once a problem has been selected for control, a key issue is the stringency of control that the Agency should impose. There are no laws that call for regulation on the basis of achieving a specified level of risk (other than zero). Furthermore, there is no such thing as an acceptable level of risk _per se._ Risks are acceptable only in light of the benefits to be gained from the action that produces the risk or the cost of reducing the risk.

In theory, control decisions should be made based on a careful balancing of benefits and risks (under statutory provisions which permit such balancing). However, there are a number of considerations which have led the Agency to establish some risk benchmarks to guide its actions. There are a variety of situations in which there is not time for the lengthy process of rulemaking. For example, environmental contamination may require an immediate response (e.g., the Agency must determine whether people should be allowed to drink contaminated water). In other cases, state officials may want guidance on levels of pollutants that are not sufficiently widespread to justify the establishment of formal standards. In taking action under "imminent hazard" provisions of various statutes, EPA must make judgments about how serious particular risks are, and these may involve chemicals for which formal standards have not been established.

This policy statement will not attempt to articulate a "target" for risk reduction -- that is, a level to which EPA will generally try to reduce risk. It is useful, however, to articulate a level of risk below which EPA believes further effort to reduce risk is only infrequently justifiable (e.g., where there is no significant cost of additional control or no significant benefit from use of a chemical). This level is stated as follows: To the extent consistent with law, EPA will generally not attempt to reduce lifetime individual risk from particular exposures to carcinogens below one in a million (10^{-6}) for highly exposed groups of people. Risks below this level are widespread and are not generally perceived as significant by the public. Further reductions in such risks are not likely to be warranted from the point of view of competition for use of EPA's resources, nor feasible except at disproportionate cost.

This concept means that the level of individual lifetime risk after control may and frequently will be greater than 10^{-6}. Where the circumstances require, this level may sometimes be much greater. The actual level to which risk will be controlled will be based in each case on the statutory requirements and other relevant factors, as discussed in Section II.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

October 23, 1981

OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: A Generic Work Plan or Guidance for EPA Study

FROM: Marilyn C. Bracken Marilyn C Bracken
Associate Assistant Administrator
for Toxics Integration (TS-777)

TO: John W. Hernandez
Deputy Administrator (A-101)

I have reviewed the attached studies as requested in your memorandum of October 5, and they appear to fall into two categories -- Group I - Multi-media/Intergrative studies and Group II - State/Regional support.

Group I - Memphis/Louisville

- These studies parallel the geographic approach that was studied by the Toxics Integration Project in the Kanawha Valley (Coordinated with Region 3).
- The Memphis/Louisville studies appear to have been initiated by the Region because of the potential environmental concern, and in some cases, some identified health effects.
- Both of these studies are likely candidates for the "Generic Approach" although I suspect that we could not establish sufficient evidence of exposure and/or potential exposure with existing data.
- The level of effort to complete these studies is greatly underestimated. I suspect their workload estimates do not project impact or need for headquarters support/review.

- I also suspect there are a number of such studies in the Regions that have been initiated that deserve identifying, tracking, reviewing, possibly stopping, etc. I think it is a rather sizable task to do this, but worth it in view of the time we end up spending if they get off track, and/or if they end up causing a great deal of Regional concern (e.g., Lead in Region VI).

Recommendation: I suggest we attempt to identify how many studies have been initiated in the Regions that fall in this Category I type. Furthermore, I recommend we review them, and determine the purpose for the study, the justification for the study, and establish a priority for continuing some of them, if appropriate.

Group II - Study Plans for Apalachicolo Bay, Brevard County and Hookers Praire.

- These studies are all some what different, but fall within the normal workload and purview of the Regional Administrators. They are one-time requests from State or Regional groups for technical assistance and advice. In general, I think the RA's must decide how their resources can best be spent to respond to these requests.
- Solutions to any problems in two of these three studies are state/local - i.e., waste load allocation and septic field leaching.

Recommendation: These studies are not candidates for Generic Analysis by Headquarters; they should remain within the decision-making authority of the RA's.

Attachment

UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

Office of the Deputy Administrator

October 5, 1981

SUBJECT: A Generic Work Plan or Guidance forEPA Study of Same Problem

FROM : John W. Hernandez, Jr. Marlene for

TO : Marilyn Bracken

Please look over the enclosed studies and see if you think that a generic approach can be developed. Should it? I know that some S&A labs do a good job but that others just start-out on a survey without doing the necessary planning.

Please give me your thoughts.

Enclosure

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MEMORANDUM

OFFICE OF
WATER

DATE: April 9 1982

SUBJECT: Outline for Background Documents on Environmental/Human Health Effects and Risk Assessment.

FROM: Victor J. Kimm, Director
Office of Drinking Water (WH550) ?

TO: Merna Hurd
Associate Assistant Administrator for Water
Office of Water (WH556)

I have asked my staff to look at the outline for the 'Health Effects' report proposed by the sludge contractor, and compare it with other similar documents in use within the Agency. Having done so, some interesting findings come to light. For example, the term 'risk assessment' is not used the same way by different groups. Within the Office of Water we are using a definition ~~which recognizes risk analysis as the review and evaluation of hazard and exposure information for purposes of estimating the likelihood that the hazards associated with a substance will be realized in an exposed population~~. Within the assessment, as defined this way, there must be a presentation of the change in exposure and risk as a result of the alternative regulatory and non-regulatory control options.

However, having reviewed ODW's most recent criteria documents, there appears a section entitled 'risk assessment' which is actually a hazard assessment, as defined in the "Criteria For Evaluating Risk Assessment Documents", a copy of which you received at the end of our meeting last Tuesday. Other documents we reviewed misuse this term as well, so it is no surprise that the recommended outline from the contractor uses the term, leaving me in some doubt as to exactly what is intended in that section.

In order to most effeciently capture my staff's review of these documents, while providing you our best advice on the structure of the contractor's reports, there is a recommended outline for the report attached.

Specifically, the report should consist of ~~three major~~ sections, the health assessment, the exposure assessment, and the risk assessment. By breaking the report into these three sections, development of the first two can go on simultaneously. Naturally, the risk assessment can not be written until the other material is drafted. The report may be viewed as a three volume piece. The content and purpose of each section of the outline is included, and I believe this approach will best serve your efforts.

# RECOMMENDED OUTLINE
for

Background Documents on Environmental/
Human Health Effects and Risk Assessment

It is possible to view the material in these documents as three distinct reports which together constitute a complete document. This outline contemplates such an arrangement.

Summary

- Executive Summary
  This section should summarize all three basic reports. It may constitute the useful decision supporting report, and would be a stand alone overview of the three in-depth reports.

Health & Environmental Assessment

The purpose of this report is to review and evaluate the scientific data in order to characterize the health hazards that may be associated with exposure to a substance, and to ascertain the strength of the scientific evidence supporting the characterization.

- Introduction
  This section should briefly identify the uses, sources and ambient levels of the chemical. The purpose is only to introduce the chemical, not to discuss material which will be presented in the latter reports.

- Pharmacokinetics
  This section should cover absorption, distribution, metabolism and excretion in humans and other species, as appropriate.
  phytotoxic problems

- Mechanisms of Toxicity
  This section identifies the biochemistry of the toxicity within the species upon whom a toxic effect is observed.

- Health Effects in Animals
  This section discusses acute effects, chronic effects, teratogenicity, reproductive effects, mutagenicity, and carcinogenicity in the animal species for which there is data, and especially for those ~~animals~~ concerned, aquatic and marine species likely to be exposed.

- Health Effects in Humans
  This section discusses case studies and anecdotal data on acute and long-term effects, as well as controlled studies and epidemiology which may be available.

- Hazard Estimation
  This section will discuss the quantification of the carcinogenic risk, the thresholds for acute and chronic risks, the impact on sensitive populations, the Acceptable Daily Intake, and interactions of the chemical with other chemicals.

Exposure Assessment

The purpose of this report is to review and evaluate the scientific data which identifies the size and nature of the human and other species exposure to the chemical, and to estimate the frequency, duration and intensity of its exposure. In addition, the amount of the exposure which can be controlled, and the technology to control the exposure should be identified.

- Introduction
  This section should briefly identify the hazardousness of the chemical. The purpose is only to introduce the chemical, not to discuss material which will be presented in other reports.
- Mass Balance
  This section discusses the occurrence of the chemical in the various media from source to use or disposal.
- Plant Uptakes
  This section discusses the information and assumptions associated with estimation of uptakes in sensitive plant species and the food chain.
- Animal Uptakes
  This section discusses the information and assumptions associated with estimation of uptakes in sensitive species and the food chain.
- Human Uptakes
  This section discusses the information and assumptions associated with estimation of human uptake, indicating the contribution from all source media.
- Exposure Control
  This section identifies the nature and efficiency of technologies which can be used to control human, animal and ecosystem exposures to the chemical. Cost data can be included.

Risk Assessment

The purpose of this report is to review and evaluate the hazard and exposure information for purposes of estimating the likelihood that the hazards associated with a substance will be realized in an exposed population. Within this assessment, a presentation is made of the change in exposure and risk as a result of the alternative regulatory and non-regulatory control options.

- Introduction
  This section should briefly identify the hazard and exposure associated with the chemical. The purpose is only to introduce the chemical, not to discuss material which will be presented in other reports.

- Regulatory and Non-regulatory Policy Alternatives

  This section will identify the analytical construct of the assessment. It will organize the readers perspective with respect to the alternatives considered, discarded and discussed in depth.

- Base Case Risk Estimate

  This section will discuss the existing situation, and will use the hazard and exposure data to estimate the national risk. In addition, there may be significant differences in risk, depending on regional or local conditions. These differences will be identified, as appropriate. Lastly, in order to support benefit-cost analysis, the risk at the decision-makers level should be identified. For example, if one of the policy alternatives is to leave control decisions to local government, the base case risk to localities should be estimated in a systematic manner.

- Policy Alternative Risk Estimates

  This section will discuss the estimates of national, regional and local risk associated with each of the policy alternatives. The format of these estimates should permit comparison between the base case and the alternatives.

- Sensitivity Analysis

  This section should identify the critical assumptions in the risk analysis, including those assumptions which were made in the hazard and exposure assessments, if they are significant to the risk estimate. The sensitivity of these assumptions to the estimate of risk should be identified in a manner which permits the decision-maker a full understanding of the significance of the assumptions.

Should cross-walk this to RIA requirements

CRITERIA FOR EVALUATING RISK ASSESSMENT DOCUMENTS

General

Risk assessments describe the changes in risk to human health associated with potential reductions in human exposure as a result of alternative regulatory and non-regulatory actions.

At minimum, a risk assessment must present information on five elements:

I Hazard Assessment (HA): HA is defined as the review and evaluation of scientific data to characterize the health hazards that may be associated with exposure to a substance, and to ascertain the strength of the scientific evidence supporting the characterization.

II Exposure Assessment (EA): EA is defined as the review and evaluation of scientific data for purposes of identifying the size and nature of the human population that may be exposed to a hazardous substance, and to estimate the frequency, duration, and intensity of its exposure. from all routes.

~~III~~ Controllable Exposure (CEx): CEx is that exposure which is within the ability of technology to control. It sets the limit of a regulatory approach, and must be related to the total exposure due to all routes.

Has

yes!!

III Risk Assessment (RA): RA is defined as the review and evaluation of hazard and exposure information for purposes of estimating the likelihood that the hazards associated with a substance will be realized in an exposed population. Within the assessment, there must be a presentation of the change in exposure and risk as a result of the alternative regulatory and non-regulatory control options.

subset 8/10

Sensitivity Analysis (SA): SA is a discussion of the uncertainty within the risk assessment. Since exposure estimates nearly always require use of uncertain data and and assumptions, the effect of varying critical assumptions must be presented, along with a clear specification of the assumption, as well as whether uncertainty is due to data or judgement.

Should be a part of each 5 sections, [illegible] 23, it should [illegible]

Purpose of the Evaluation

The evaluation criteria presented below are intended to ensure consistency in risk assessments within the Office of Water. In brief, the evaluation will review the adequacy of health effects assessments, the completeness and appropriateness of critical assumptions, the manner in which uncertainty is reflected, the sensitivity of assessment conclusions to assumptions, the completeness of consideration of regulatory and non-regulatory alternatives, and the extent to which multi-media exposures are incorporated into the assessment.

References

Risk assessment is a rich field of study, and much has been written on how to conduct these analyses. Two references which are especially helpful, and which indicate both the development and use of an assessment are:

National Academy of Sciences, Decision Making for Regulatory Chemicals in the Environment, Washington, D.C. 1975.

Hirshleifer, J. and Shapiro, D.L., "The Treatment of Risk and Uncertainty", Public Expenditures and Policy Analysis, Haveman, Margolis: Eds., Markham Publishing Co., Chicago, 1970.

Criteria

The following criteria are for use when evaluating risk assessment plans, draft assessments, or final assessments. It is assumed that risk assessments are for use in the policy making process, and are available before regulations, guidances or policies are proposed.

1. Health Effects Component. Does the risk assessment contain a segment which identifies the health effects basis for the assessment? Does that segment reflect:

- A thorough literature search strategy containing the following essential elements:
  - a) recognized standard sources;
  - b) sources relating specifically to the subject under study;
  - c) all relevant machine-readable data bases (i.e. MEDLARS, Lockheed Information System's DIALOG, etc.).

  The presence of primary reference should be mandatory to prevent misinterpretations.

— [illegible] EA ?

- A careful review of computer printouts and selection of relevant citations for hard copy retrieval.

- A comprehensive discussion of pharmacokinetics including:
  a) absorption;
  b) distribution;
  c) metabolism;
  d) excretion;
  e) bioaccumulation.

Problems arising in evaluating the pharmacokinetics data at different doeses should have been addressed (i.e., alteration of the rates and the pattern of metabolishm in different species and strains of animals and among sub-groups of human populations, etc.).

- A detailed evaluation of health effects in animals and in humans.

  Systems and organs affected should be identified, and a correlation of exposure and effect should be discussed. Major findings from animals studies should be discussed under the following subheadings:

  a) Acute effects
    *LD(50) (lethal dose, 50% kill)
    *LD(LO) (lowest published lethal dose)
    *LC(50) (lethal concentration, 50% kill)
    *LC(LO) (lowest published lethal concentration)
    *and other short term effects on target organs.
  b) Subacute or Subchronic effects (time depedent).
  c) Chronic effects, including but not limited to:
    * carcinogenic effects
    * mutaginic effects
    * teratogenic effects
    * reproductive effects.

- Any analysis of animal toxicity studies should include basic information on:
  a) sex, species, age, and health of animals showing adverse effects;
  b) interactions with other chemicals (antagonistic and/or synergisitc effects);
  c) inter- and intraspecies variability in response;
  d) dose-response data for acute or longterm effects, with emphasis on threshold exposures;
  e) effects of possibly cumulative exposure.

- A careful evaulation of animal toxicity studies that elucidate the mechanism of toxic action, with particular attention given to the validity of subtle effects described from exposure of humans to the chemical.

- A critical examination of carcinogenesis/mutagenesis/teratogenesis tests results to detect any occurrence of false-negative or false-positive for a given chemical;

- A discussion of major problem areas such as:
  - a) conflicting reports indicating that further research is needed to determine the significance of a particular toxic action;
  - b) possibility of unconventional methods being used and insufficient data as to their reliability, accuracy, consistency, and validity;
  - c) validity of experimental design and statistical methods used in animal studies, controlled human exposures, etc.;
  - d) disagreement between epidemiological studies, clinical reports, and chronic toxicological studies;
  - e) complication of longterm health effects of exposure arising from environmental alternations, biogmagnification, and dietary variability, etc.

2. Exposure Component . Risk is a function of both the intrinsic toxicity of the chemical and the pattern of use projected for it. Does the assessment discuss the realistic expected human exposure to the contaminants? Does the exposure segment reflect:

- Actual or modeled changes in patterns of contamination;

- Relative exposure estimates for regulatory options, including alternative standards, the ambient condition, the relative contribution from other sources of the contaminants.

3. Reliability and Sensitivity . Without perfect information, assessments must depend upon estimates and may be written even though some information is not available, even as an estimate. Does the assessment deal with missing or unreliable data in a manner which still permits rational decision-making, or its analog, reasonable judgement? Does the assessment contain a segment which reflects:

- Estimates of data uncertainty;
- Development of 'estimated ranges of probable risk' which capture the probable risk, taking uncertainty into consideration;
- Specification of those parameters to which the assessment is most sensitive.

4. Analytical Parameters . Does the risk assessment use standard parameters which reflect the Office of Water and EPA policies? Do inconsistencies exist in use of:

- Safety factors;
- Extremely low exposure toxicity models;
- Distribution of sensistive populations;
- Ingestion, inhalation, other exposure estimates;
- Human and environmental contaminant pathways;
- Engineering and technological efficiencies.

5. Association with Policy Alternatives . Does the risk assessment present the estimated risk for each of the major policy alternatives? Are the major policy alternatives diverse and innovative or are they very closely related in scheme? Does this presentation reflect:

- Quantitative estimates;
- A 'best estimates';
- A 'range of realistic estimate';
- A 'range of all possible estimates';

6. Data Presentation . Does the assessment present the risk data in a manner which is useful to the decision-makers and outside reviewers? Does the presentation reflect:

- Simplcity in data presentation;
- Understandability to an educated public;
- Differentiation power for the assessment of policy alternatives;
- Inter-media and inter-regulation comparisons (relative vs. absolute risk)
- a distinguishing between scientific data and value judgement estimates.

Recommended Outline for a Sludge Risk Assessment

In reviewing the sludge materials, it appeared to us that the critical policy questions deal with how to safely and efficiently dispose of sludges. Specifically, which are the significant contaminants in sludes? How and to what degree can these contaminants be transported to humans as a result of sludge disposal applications? Are there distinctions between types and sources of sludges and their possible end applications? What cirteria and operating mechanisms can be established to properly control this program? Procedurally, what should be the process for completing the analysis of all these issues and thereby producing a final policy? From these questions it is possible to structure an outline for a risk assessment. The outline below is an initial suggestion which reflects our experience in dealing with these human health issues, but also reflects our lack of experience in dealing with sludges. The italicized section below each outline point is our best estimate of what is now available and what is missing. In many cases, the information exists, but is dispersed throughout several background documents. We recommend that the existing material be brought together, and only then should new analytical efforts be started. It is clear that a great deal of analytical work will remain, but the structure and even the modeling capability to do it are already in existance. Lastly, we recommend that your staff review our comments and improve this outline. We recognize that their background on this subject is greater than our own, and no doubt they can put their hands on much of this data quickly. As this information is pulled together, we would expect that some conclusions would become clear before all the questions are answered. There might be some disposal methods that are clearly acceptable and some others that are clearly not acceptable. The Agency could then make these decisions known while continuing to work on the remaining, more difficult problems.

1. Introduction and technical background

   [illegible]

   o Volume, content and sources of primary, secondary and post-digestion sludges.

   This information was not presented in the basic briefing materials. It is dispersed throughout the background documents, but even in those materials the breadth of the problem is not discussed. This introduction must go beyond the pre-treatment issue, although pre-treatment could be highlighted.

2. Health Considerations of Sludge Components

   o Contaminants with significant potential for adverse impacts on human health, animals, ecosystems, plants and crops.

Seven contaminants have received special attention, to the point where draft recommended contaminant levels have been developed. This list should be expanded with respect to other metals, organics, and especially bacterial and viral components of sludges. It may be that as ADI's are established, several of these chemicals will drop off the sludge related lists.

- Acceptable Daily Intakes for the significant contaminants, including all human exposures from air, water, food, and sludge disposal methods. This section would describe the exposure routes which exist, the relative importance of each, and the basic computation of each ADI. Criteria must apply to the aggregate concentration of the contaminants of the soil/sludge mix at the application site, and should not apply to the sludge component alone.

ADI's have been developed for several of these chemicals. It remains for the sludge related exposures to be compared with other exposure routes. This should be done quickly, as some of the chemicals may well drop off the list, due to the insignificance of the sludge component of human exposure.

- Non-human impacts of the significant contaminants, describing impacts on soil, plants and crops, food chains, domestic and non-domestic animals.

This has not been aggregated into a simple presentation, but much of the data do exist in the background documents.

- Specification of the critical contaminants and exposure routes, based on the preceding discussions. A comparison of exposure levels of these contaminants with background levels.

This is a critical step which, when done, may immediately support some of the proposed technical options. It will drive the discussion technical discussion, in that it will limit the need to discuss some technical options which could have been developed, and may exist in the discussion now.

3. Technical options

- Specify the efficacy and cost of the various disposal options, including incineration, ocean dumping, and the many different types of land disposal.

The efficacy and cost data have not been packaged, or were not in materials you provided us. It is important to key on the technical costs in this section. Some costs of disease treatment were presented, and they are a surrogate for the benefits which might be derived from various regulatory or non-regulatory options. However, they are not the costs of the treatment/control technologies.

- Indicate the current mix of the technical approaches in use, and the optimum mixes which could be suggested.

One mix of land applications has been presented (fertilizer/soil conditioner for an assumed set of markets). The use or disposal by other techniques is not discussed in any way. It may be that land disposal is singularly less efficient than other techniques, and that a ban should be proposed. This is not a suggestion, but indicates the extent to which lack of information constrains the policy maker, as insufficient information on competing disposal methods is presented to dispell that potential conclusion.

- o Assess the administrative feasibility of controling the various technical approaches described above.

Administrative feasibility has been left undiscussed. The state and federal implementation activities may limit the technical approaches available. For example, if there is to be an enforcement component of potential regulatory alternatives, the feasibility of such enforcement must be identified and presented.

- o Assess the cost of these technical approaches.

Models ~~apparently~~ exist to quickly aggregate technical costs. These should be reemployed as necessary to develop the costs of each of the technical approached developed.

- o Assess the risks associated with these technical approaches.

This has been done only for the baseline and a pre-treatment option. It must be expanded to consider other reasonable technical approaches such as ocean dumping and incineration. It appears that a model exists to assist in this process.

4. Regulatory and Non-regulatory Alternatives

- o Specify which of the technical approaches would satisfy the needs of the legislative mandates.
- o Present the regulatory and non-regulatory approaches which stem from the previous chapter and section, indicating the risk estimates associated with each, as well as the sensitivity of the critical assumptions which were applied to each approach.

This presentation is, of course, the key decision maker's section. This is what you are aiming for, and what you are now missing.

United States
Environmental Protection
Agency

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EPA

Research and Development

ADDITIONAL U. S. ENVIRONMENTAL PROTECTION AGENCY GUIDANCE FOR THE HEALTH ASSESSMENT OF SUSPECT CARCINOGENS WITH SPECIFIC REFERENCE TO WATER QUALITY CRITERIA

Prepared for

OFFICES OF THE U. S. ENVIRONMENTAL
PROTECTION AGENCY

Prepared by

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ADDITIONAL U.S. ENVIRONMENTAL PROTECTION AGENCY GUIDANCE FOR THE HEALTH ASSESSMENT OF SUSPECT CARCINOGENS WITH SPECIFIC REFERENCE TO WATER QUALITY CRITERIA

A. Introduction

This document proposes an addendum to the guidelines adopted in 1976* under which the U.S. Environmental Protection Agency (EPA) has carried out its risk assessments on suspect carcinogens. These guidelines call for a qualitative weight-of-evidence judgment concerning the likelihood that an agent is a human carcinogen. The weight-of-evidence approach entails a thorough review of all of the available data from animal bioassays, human epidemiology, metabolism data, and in vitro studies, both positive and negative, with judgments based on the scope and quality of the available evidence. The guidelines also call for quantitative estimation of the incremental cancer risks associated with given levels of exposure using several mathematical extrapolation models relating dose to cancer incidence.

In practice, no formalized system for stratifying the qualitative weight-of-evidence has been used. Frequently the term "substantial" has been used where the weight-of-evidence was greater than "suggestive," recognizing that the same term was being applied to strong evidence in some cases and relatively weak evidence in others. Alternate ways of expressing the weight-of-evidence were also employed.

With quantitative assessments, the linear non-threshold extrapolation

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*"Interim Procedures and Guidelines for Health Risk and Economic Impact Assessments of Suspected Carcinogens," Federal Register 41(May 25, 1976): 21402.

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model has been used almost exclusively because it provides a plausible upper-limit estimate of low-level risks and no other extrapolation model has as much scientific acceptance. It has been recognized that the upper-limit risk estimates obtained by the use of the linear non-threshold extrapolation model, although plausible, involve large uncertainties and the true risk can range from the upper-limit to lower levels; i.e., approaching zero. Also, the plausibility of the linear non-threshold extrapolation model is predicated to a significant degree on the mutagenicity of carcinogens and not all carcinogens have this property.

In recent years there has been a growing awareness of the need to amend the EPA's cancer risk assessment guidelines (cited above) in two respects:

1) to adopt a formal approach that would stratify the weight-of-evidence judgments about the likelihood than an agent is carcinogenic for humans, and
2) to take mutagenicity into account in quantitative risk assessment.

Section B, which follows, proposes an approach for stratifying the weight-of-evidence for carcinogenesis. This is followed, in Section C, by a proposed approach to the quantitative assessment of suspect carcinogens according to the evidence for mutagenicity. Section D applies the above approaches to guidance on water quality criteria. Section E summarizes options to the proposed approach and guidance on water quality criteria.

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B. Stratification of the Weight-of-Evidence for Carcinogenesis

Although there are various possible methods of stratifying the evidence for carcinogenicity on the basis of factors such as the number of studies done, the species affected, the target organs affected, or the background tumor rates in target organs, etc., only one approach has had extensive institutional use and international acceptance, namely, the one developed and used for several hundred agents by the International Agency for Research on Cancer (IARC). As with the current EPA approach to risk assessment, the IARC approach requires the collective judgment of experts, thus providing a reasonable balance between flexibility and a consistent set of ground rules for evaluating the evidence for carcinogenicity. For this reason, the IARC approach is proposed for use by the EPA; it is presented below as published by IARC* with two editorial changes indicated by footnotes.

B.1 The IARC Stratification of the Evidence for Carcinogenicity

B.1.1 Assessments of evidence for carcinogenicity from experimental animal studies

These assessments are classified in three groups:†

a. Sufficient evidence of carcinogenicity indicates that there is an increased incidence of malignant tumours: (a) in multiple species or strains, or (b) in multiple experiments (preferably with different routes of administration or using different dose levels), or (c) to an unusual degree with regard to incidence, site or type of tumour, or age at onset. Additional evidence may be provided by data concerning dose-response effects, as well as information on mutagenicity or chemical structure.

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*IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Chemicals and Industrial Processes Associated with Cancer in Humans, IARC Monograph Supplement 1. Lyon, France. September 1979.

†The last two IARC categories have been deleted because of their lack of relevance to EPA needs.

b. Limited evidence of carcinogenicity means that the data suggest a carcinogenic effect but are limited because: (a) the studies involve a single species, strain, or experiment; or (b) the experiments are restricted by inadequate dosage levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or (c) the neoplasms produced often occur spontaneously or are difficult to classify as malignant by histological criteria alone (e.g., lung and liver tumours in mice).

c. Inadequate evidence indicates that because of major qualitative or quantitative limitations, the studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect.

8.1.2 Assessment of evidence for carcinogenicity from human studies

Evidence of carcinogenicity from human studies comes from three main sources:

1. Case reports of individual cancer patients who were exposed to the chemical or process.
2. Descriptive epidemiological studies in which the incidence of cancer in human populations was found to vary spatially or temporally with exposure to the agents.

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3. Analytical epidemiological (case-control and cohort) studies in which individual exposure to the chemical or group of chemicals was found to be associated with an increased risk of cancer.

Three criteria must be met for a causal association to be inferred between exposure and human cancer:

1. There is no identified bias which could explain the association.
2. The possibility of confounding has been considered and ruled out as explaining the association.
3. The association is unlikely to be due to chance.

In general, although a single study may be indicative of a cause-effect relationship, confidence in inferring a causal association is increased when several independent studies are concordant in showing the association, when the association is strong, when there is a dose-response relationship, or when a reduction in exposure is followed by a reduction in the incidence of cancer.

The degree of evidence for carcinogenicity in human studies is categorized as:

a. *Sufficient evidence* of carcinogenicity indicates a causal assocation between exposure and human cancer.

b. *Limited evidence* of carcinogenicity indicates a possible carcinogenic effect in humans, although the data are not sufficient to demonstrate a causal association.

c. *Inadequate evidence* of carcinogenicity indicates that the data are qualitatively or quantitatively insufficient to allow any conclusion regarding carcinogenicity for humans.

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Dividing lines are by no means firmly drawn between sufficient evidence and limited evidence from animal studies and between inadequate evidence and limited evidence from both human and animal studies.

B.1.3 Evaluation of the carcinogenic risk to humans

Presently, no objective criteria exist to interpret the animal data directly in terms of human risk. In the absence of sufficient evidence from human studies, evaluation of the carcinogenic risk to humans is based on consideration of both the epidemiological and experimental evidence. The quality of evidence discussed in B. 1.1 and B.1.2 is used by IARC to place the chemical into categories.

The chemical, groups of chemicals, or industrial process is placed into one of three groups:

Group 1

The chemical, group of chemicals, or industrial process is carcinogenic for humans. This category is used only when there was sufficient evidence to support a causal association between the exposure and cancer.

Group 2

The chemical or group of chemicals is probably carcinogenic for humans. This category includes chemicals for which the evidence of human carcinogenicity is almost 'sufficient' as well as chemicals for which it is only suggestive. To reflect this range, this category is divided into higher (group A) or lower (group B) degrees of evidence. The data from experimental

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animal studies play an important role in assigning chemicals to category 2, and particularly to those in group B.

Group 3*

The chemical or group of chemicals is possibly carcinogenic for humans.

C. Quantitative Cancer Risk Estimation with Respect to Mutagenicity

The linear non-threshold extrapolation model has had extensive use in quantitative risk estimation beginning in the field of ionizing radiation and extending to chemical carcinogens. The rationale for the use of the linear non-threshold dose-response extrapolation model depends heavily on the mutagenicity of carcinogens. The biological justification for its use lies in the correlation between carcinogenesis and mutagenesis, the commonality of DNA damage for carcinogens and mutagens, the quantal nature of the interactions of chemicals with DNA, and the linear nature of the dose-response relationships for the induction of gene mutations, particularly with organisms such as bacteria, in which the induced mutation frequencies have been determined at lower exposure levels than in other organisms. From a theoretical standpoint, the multistage mathematical model for carcinogenesis has considerable biological plausibility, and linearity at low dose levels is a part of this model.

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*The original IARC wording is as follows: "The chemical or group of chemicals cannot be classified as to its carcinogenicity for humans." Since agents in this category might or might not be carcinogenic for humans, the description "possibly carcinogenic for humans" is considered more appropriate, particularly since agents in this category might have "limited" evidence which is almost "sufficient."

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In the absence of the induction of gene mutations, the use of the linear non-threshold dose-response model becomes less plausible. Many mathematical extrapolation models fit most tumor data in the range of experimentally observed responses but give widely divergent estimates of risk at the low doses generally encountered with environmental exposure, where it is not possible to obtain direct experimental or epidemiological dose-response relationships. The selection of a particular mathematical extrapolation model requires some acceptance of a mechanism of carcinogenic action. The plausibility of the linear non-threshold extrapolation model depends heavily on acceptance of the somatic mutation theory of cancer. We currently do not know enough about the mechanism of action of non-mutagenic carcinogens to plausibly extrapolate dose-response relationships for such agents to low levels of exposure.

We are therefore proposing, in principle, for apparently non-mutagenic carcinogens, to use the conventional toxicologic approach to defining exposure limits in terms of the no-observed-effect-level (NOEL) of response together with an uncertainty factor. However, in practice, since the evidence for mutagenicity is frequently not decisive, it may be appropriate to use both the linear non-threshold extrapolation model and the conventional toxicological approach together with guidance as to the relative weights to be given to each. This is the approach which is proposed for water quality criteria in Section D.

In Section C.1 we describe the conventional toxicological approach. Section C.2 proposes the criteria for evaluating chemical substances according to mutagenicity.

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C.1 Conventional Toxicological Approach for Non-Mutagenic Carcinogens

The conventional toxicological approach to setting exposure limits is to select the highest dose that does not produce a response in test animals, and then to adjust this dose downward by dividing it by an uncertainty factor.

We propose to use the conventional toxicological approach for non-mutagenic carcinogens with an added refinement of making use of all the available data in a consistent fashion. This modification involves the use of the multistage extrapolation model to estimate the dose corresponding to a 10% response. The procedure has the advantage of standardizing the response level to which the uncertainty factor is applied. It is particularly appropriate when the no-observed-effect level is absent, as is often the case with NTP-type carcinogenesis bioassays that involve the use of maximum and half-maximum tolerated doses where both doses show tumorigenic responses. The 10% response level is a reasonable one because it is in the domain of the minimal level of response that can be observed in well-conducted bioassays. The extrapolated dose at the 10% response level is relatively insensitive to the particular mathematical model that is employed to determine it. The multistage model is recommended because it has had extensive use and has some biological rationale. The extrapolation can be done on the basis of a maximum likelihood estimate (MLE) or an upper confidence limit. In line with conventional practice, the proposed approach would use the maximum likelihood estimate and a dose conversion from animals to humans on a body weight basis rather than by surface area.

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primary concern because there is evidence that gene mutations caused by chemical agents that react with DNA follow a linear non-threshold mechanism; such evidence does not exist for chromosome mutations.

As with carcinogenicity, a weight-of-evidence approach is used for evaluating genotoxicity; the evaluations are described under the following categories: adequate, inconclusive, negative, and inadequate.

Adequate evidence involves positive responses in any two appropriately conducted different gene mutation systems, one of which is a eucaryotic system, or, by itself, a positive response in a whole mammal gene mutation test. It should be noted that the generally accepted definition of mutation is a relatively permanent change in hereditary material involving either a fundamental change in genes or a physical change in chromosome structure or number. However, only point mutation is considered as positive, whereas chromosomal mutation is not considered as positive for the reason discussed above concerning linear non-threshold mechanisms. Chromosome mutations and primary DNA damage (e.g., sister chromatid exchanges, unscheduled DNA synthesis, mitotic recombinations) are considered as supportive evidence for the ability of a chemical substance to cause gene mutations, since chemicals that cause these effects may also cause gene mutations.

Inconclusive evidence exists where definitive conclusions about mutagenicity cannot be made. In some cases it may be possible to indicate whether the evidence tends toward being positive or negative.

The choice of the uncertainty factor is somewhat arbitrary. A factor of 1,000 is recommended consisting of factors of 10 each for 1) the possibly greater sensitivity of humans in general compared to animals in general; 2) the sensitivity of the most susceptible human compared to the average human; and 3) an additional factor of 10 because of the general irreversibility and lethality of the carcinogenic response.

When carcinogenesis data are available in humans, the first uncertainty factor is considered to be a safety factor of 10 based on the greater certainty that the agent is carcinogenic for humans. Thus, in both humans and animals, the uncertainty factor is 1,000.

Given our current uncertainty about how to estimate cancer risk at low levels of exposure to non-mutagenic carcinogens, the proposed conventional toxicological approach does not necessarily imply the existence of a cancer threshold level below which exposures are completely safe. Hence, wherever possible, the formulation of exposure limits should tend toward the lowest feasible levels.

C.2 Criteria for Evaluating Chemical Substances with Respect to Their Ability to Cause Gene Mutations

Evaluating the ability of chemicals to cause gene mutations will assist in deciding whether to use a linear non-threshold extrapolation model to estimate cancer risk. The rationale for this approach, described above in Section C, is based on the somatic mutation theory for carcinogenesis. Somatic mutations include gene mutations as well as chromosome mutations. Gene mutations are of

Inadequate evidence exists when the evidence is insufficient as a basis for the evaluation of mutagenicity.

Negative mutagenicity is defined operationally as negative responses that are obtained in an adequate number of test systems capable of detecting gene mutations, chromosomal aberrations, and primary DNA damage. An adequate number and combination of test systems may vary from chemical to chemical and depends on many factors such as physical-chemical properties, structure-activity relationships, and toxicity of the chemical to the test organisms. As a consequence, professional scientific judgment is required on a case by case basis to determine the number and types of test systems necessary to classify a chemical as being non-mutagenic.

D. Application to Water Quality Criteria of Additional Guidance for the Health Assessment of Suspect Carcinogens

The Consent Decree Water Quality Criteria (44 FR 15974, March 15, 1979 and 45 FR 79347, Nov. 28, 19 were based on the 1976 guidelines for the assessment of suspect carcinogens and used the linearized multistage extrapolation model, regardless of the evidence for or against mutagenicity, to establish the concentration that would be associated with a maximum excess cancer risk of 10^{-5}. In order to incorporate evidence for mutagenicity, it is proposed here that both the linear non-threshold and the conventional toxicological approaches be used to provide a range of water quality criteria values. It should be recognized that the conventional toxicological approach

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specifies an exposure limit; the linear non-threshold extrapolation model provides the relationship between risk and exposure, and requires the selection of a risk value in order to define an exposure limit. It is proposed that the range of values for the water quality criteria would involve a lower limit, which would be the concentration associated with an excess lifetime cancer risk of 10^{-5}, and an upper-limit, which would be the concentration specified by the conventional toxicological method.

The weight-of-evidence for or against mutagenicity as well as the weight-of-evidence for carcinogenicity would provide the basis for the water quality criteria guidance as shown in Table 1. Adequate evidence of mutagenicity and sufficient evidence of carcinogenicity would tend to call for the use of the linear method. Sufficient human or animal evidence for carcinogenicity and negative evidence for mutagenicity would tend to call for the use of the conventional toxicological method as would limited evidence for carcinogenicity together with inadequate or inconclusive evidence for mutagenicity. Inadequate or inconclusive mutagenicity data, together with sufficient human or animal carcinogenicity evidence, or adequate evidence in mutagenicity with limited evidence for carcinogenicity, would tend to call for a water quality criterion in the domain of the average of the criteria determined by the linear and the conventional toxicological methods. The midrange would be the geometric mean, that is, the square root of the product of the bounding concentrations of the water quality criteria range, since this places the midrange equidistant from the extremes on a proportional basis. Such guidance, together with the usual considerations that go into regulatory

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TABLE 1. WATER QUALITY CRITERIA GUIDANCE*

IARC Rank for Carcinogenicity Evidence†	Weight of Evidence for Mutagenicity			
	Adequate	Inconclusive	Inadequate	Negative
Sufficient	L	M	M	U
Limited	M	U	U	U

*L = Toward lower limit.
M = Toward midrange.
U = Toward upper limit.

†"Sufficient" corresponds to IARC categories I, IIA, and IIB.
"Limited" corresponds to IARC category III.

decision-making, would determine where in the range the actual water quality standards would be set.

Table 2 illustrates the above approach to water quality criteria guidance for selected compounds. The carcinogenicity and mutagenicity categorizations are indicated for each compound. The water quality criteria concentrations listed in column (a) were obtained using the "linearized" multistage model with an upper 95% confidence limit for the linear non-threshold extrapolation and a surface area correction. Water quality criteria are presented in column (b) which were calculated by the proposed conventional toxicological approach using the multistage model to obtain the MLE of the 10% level of response, an uncertainty factor of 1,000, and an animal-to-man conversion on the basis of body weight. It can be seen in column (c) that for these chemicals the proposed NOEL approach results in water quality criteria concentrations that range from about 10 to 369 times greater than those obtained using the linear method.

The guidance given in column (e) is based on the criteria given above. In practice, the guidance would need to be formulated by a group of experts with due consideration of all of the available evidence. The guidance toward the midrange with many of these compounds reflects the fact that few suspect carcinogens have been tested adequately for mutagenicity. In some cases, it might be appropriate to defer guidance if additional information can be obtained in a reasonable time particularly where the evidence for mutagenicity is inadequate or inconclusive. The midrange values, calculated as geometric

TABLE 2. ILLUSTRATIVE GUIDANCE ON WATER QUALITY CRITERIA FOR SELECTED COMPOUNDS ACCORDING TO CARCINOGENICITY, MUTAGENICITY, AND METHOD OF ESTIMATION

Compound	Carcinogenicity Group	Mutagenicity	Method of Estimation					
			(a) Linear Multistage* 95% Lower Bound Dose at 10^{-5} Risk (ug/liter)	(b) Conventional MLE Estimate§ 10% Response with 1000 Uncertainty Factor (ug/liter)	(c) Ratio - Conventional: Linear	(d) Geometric Midrange Concentration (ug/liter)	(e) Guidance on Water Quality Criteria	(f) Conventional Non-cancer (ug/liter)
Aldrin	2B	inconclusive	2.8×10^{-2}	8.0	285	0.5	toward midrange (d)	0.54
1,1,2-Trichloroethane	2B	inadequate	6.0	2,085	348	112	toward midrange (d)	7,900
Hexachloroethane	2B	inadequate	19.0	7,018	369	365	toward midrange (d)	880
Chloroform	2B	inconclusive	1.9	322	169	25	toward midrange (d)	4,300
Hexachlorobutadiene	3	inadequate	4.5	477	106	46	toward conventional (b)	69
TCDD	2A	inconclusive	2.1×10^{-9}	3.8×10^{-7}	181	2.8×10^{-8}	toward midrange (d)	8.8×10^{-8}
Trichloroethylene	2B†	adequate	27.0	4,533	172	354	toward linear (a)	810
Chlordane	2B	inadequate	4.6×10^{-3}	0.7	141	5.7×10^{-2}	toward midrange (d)	4.5

*With surface area conversion from animals to humans.
§With weight conversion from animals to humans.
†Based on new New National Toxicology Program data (not finalized).

(continued on the following page)

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TABLE 2. (Continued)

Compound	Carcinogenicity Group	Mutagenicity	Method of Estimation (a) Linear Multistage* 95% Lower Bound Dose at 10^{-5} Risk (ug/liter)	(b) Conventional MLE Estimate§ 10% Response with 1000 Uncertainty Factor (ug/liter)	(c) Ratio = Conventional: Linear	(d) Geometric Midrange Concentration (ug/liter)	(e) Guidance on Water Quality Criteria	(f) Conventional Non-cancer (μg/liter)
Benzene¶	1	adequate	6.6	66	10	20.9	toward linear (a)	5,300
Benzidine	1	adequate	1.2×10^{-3}	1.2×10^{-2}	10	3.8×10^{-3}	toward linear (a)	35
Carbon tetrachloride	2B	inconclusive	4.0	811	203	57.0	toward midrange (d)	3,300
DDT	3	inconclusive	2.4×10^{-4}	3.8×10^{2}	158	3.0×10^{-3}	toward conventional (b)	0.1
Heptachlor	2B	inconclusive	2.8×10^{-3}	0.5	179	3.7×10^{-2}	toward midrange (d)	5.5
Vinyl chloride	1	adequate	20.	1761	88	187.7	toward linear (a)	1,800

*With surface area conversion from animals to humans.

§With weight conversion from animals to humans.

†Based on new New National Toxicology Program data (not finalized).

¶Based on the CAG's updated risk assessment on benzene (Response to Public Comments on Benzene, Nov. 13, 1981). The criteria presented above should be increased by a factor 1.37.

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averages, are given in column (d). As with the values for the range of water quality criteria given in columns (a) and (b), the midrange is not meant as a hard figure but as a target to be sought. Water quality criteria based on the most sensitive non-cancer toxic response is given in column (f) as a limit not to be exceeded by the water quality criteria based on cancer.

E. Options for Alternative Approaches to the Formulation of Water Quality Criteria

1. The 95% upper confidence limit could be used to estimate the 10% response level instead of the maximum likelihood estimate (MLE). It would be a somewhat more stable value than the MLE and would reward good experimentation. The approach would generally lower the water quality criteria by a factor of 2 to 3. However, an upper-limit approach to the conventional toxicologic method, on the grounds of consistency, might call for the adoption of other conservative approaches which in the aggregate could amount to a factor of 36.

2. The dose conversion could be done on a surface area rather than a weight basis. Although more scientifically defensible for some pharmacological effects, it is not clear that this would be true for carcinogens. The approach would result in lower water quality limits by a factor of 6 or 12 for studies done on rats or mice, respectively. Use of the surface area conversion is not the conventional toxicologic approach.

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3. A series of graded uncertainty factors could be established on the basis of strength of evidence for carcinogenicity and mutagenicity that would specify a single water quality criterion number rather than a range with guidance. This would avoid the possible difficulty that the proposed approach could result in different water quality criteria in different states. However, graded uncertainty factors associated with specified strength of evidence would be a somewhat arbitrary scheme.

4. The linear extrapolation could be extended down to risk levels of 10^{-6} or 10^{-7} instead of 10^{-5} to establish the lower range of the water quality criteria concentrations. This would conform to the currently promulgated water quality criteria, where such options are offered, and would be simple to include in the proposed approach. However, it might be widely regarded as excessively restrictive.

5. The midrange guidance could be done on an arithmetic basis rather than on a geometric basis; this would be a far less conservative approach since the resulting level would be relatively close to the upper concentration range, i.e., 1/2 the upper value. The midrange guidance is used where it is uncertain whether the dose-response is linear or curvilinear; i.e., where the risk drops very rapidly with decreasing dose. Where the concentration range is, for example, 100, the range of risk would also be 100 if the response were really linear. The use of the arithmetic average would thus represent a dose

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reduction from the upper-limit of only a factor of 2 and would thus constitute an endorsement of the curvilinear dose-response when the data do not support such a position. A geometric average, which in this example would be 10 times larger than the lower-limit and 10 times smaller than the upper-limit, would be a more balanced approach.

6. Limited carcinogenicity data with inadequate or inconclusive evidence for mutagenicity could call for a guidance toward the midrange instead of toward of the upper concentration limit. The double uncertainty about whether an agent is really carcinogenic and mutagenic is the basis for the proposed approach, but the midrange guidance might be more appropriate as one which errs on the side of protection.

Waterborne Carcinogens: A Regulator's View

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COST-BENEFIT analysis is useful in the regulatory decision process because it helps identify many anticipated effects of regulations and establish program priorities and the order in which potential regulatory actions should be followed. However, there are serious problems in quantifying various factors in such analysis, especially those related to health effects. The widespread use of cost-benefit analysis may not lead to improved decisionmaking.

Risk of death, injury, or serious illness is an ever-present aspect of daily living. Indeed, we all take measurable risks for benefits that are small or even frivolous. Every time we drive to another city instead of flying, or every time we get impatient while trying to make a left turn and do not wait until all oncoming cars have passed, we are showing that we place a finite value on our own lives. And though we may take those risks voluntarily, they are involuntary for the friends and family members riding with us.

In the broad range of societal activities called "regulation," implicit valuation of human life and the resulting limitation on what we as a society are willing to pay to save lives are commonplace. No regulatory agency would, for example, seriously consider requiring the installation of a technically perfect air bag system costing $10,000 for each car, even if the law explicitly required the maximum feasible protection. No government regulatory agency is trying to achieve a "zero-risk society." The

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The views expressed in this paper are those of the authors and do not necessarily represent the official position of the U.S. Environmental Protection Agency.

Delaney clause in the 1958 Food Additive Amendments, for example, is one of the few cases in which Congress itself balanced risk and benefit rather than delegating this judgment to a regulatory agency.[1] Congress concluded (whether correctly or not) that no food additive had sufficient benefits to justify any carcinogenic risk.

Such balancing is not new. When the Food and Drug Administration set allowable levels for mercury in fish, it had to make a whole series of assumptions involving such things as the level of fish consumption, the reliability of toxicity studies, the proportion of the mercury that is in the more toxic organic forms, and the magnitude of the safety factor. There would be a substantial difference in the final mercury level, for example, if it were based on the 90th percentile of fish consumption or on the 99.9th percentile. Thus, depending on these and other similar assumptions, the Food and Drug Administration could be more or less conservative, more or less stringent in the level eventually set. The point is that judgments about the costs and benefits of alternative mercury levels are buried in the choices that were made. So what is new is not that the balancing of risks and benefits is going on, but that it is becoming more explicit, particularly in the regulation of carcinogens. Since we assume that there is no safe level for a carcinogen, we can no longer pretend that our standards are safe levels—that is, levels at which no risk would occur.

As society grapples with the problems of regulation of carcinogens, regulatory agencies are being urged to quantify risks and benefits and to conduct cost-benefit analyses. President Carter's Executive Order 12044 encouraged the performance of such analyses, and President Reagan's executive order goes considerably further in this direction.[2] A variety of regulatory reform bills have been introduced in Congress that would require them with varying degrees of strictness. Some of these proposals seem to be based on the naive notion that the agencies do not balance risks and benefits and should, and that doing so would clearly show that many regulations are unjustified. A more sophisticated version of this argument maintains that the quality of decisionmaking would be vastly improved if agencies' implicit judgments about risks and benefits were made explicit and subjected to as much rigorous and quantitative analysis as possible. The regulatory agencies certainly have problems and should take advantage of whatever will enable them to make correct choices. But the

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[1] 72 Stat. 1785.

[2] E. O. 12044, "Improving Government Regulation," 3 C.F.R. 152 (1978); E. O. 12291, *Federal Register*, vol. 46 (February 19, 1981), pp. 13193–98.

Environmental Protection Agency's experience with the drinking water regulations leads to serious doubts that formal risk-benefit or cost-benefit analyses will actually improve decisionmaking.

To understand why, it is important to understand how carcinogens are regulated. All the federal regulatory agencies have made the policy judgment to treat carcinogens as though they have nonthreshold effects—that is, as if any exposure to such a substance, no matter how small, causes a small but nonzero probability of a cancer that could result in death. This is a policy judgment that has been developed over a long period, not a statement of scientifically proven fact. The basis of the judgment can be summarized briefly.

First, the existence of a threshold cannot be experimentally proven. Suppose that at some dose level, an experiment fails to show any positive response. Empirically the possibility cannot be ruled out that an experiment with a much larger sample size would have shown an effect. (Even if the larger experiment were then performed, the same argument could be applied.) Although this holds true for all toxicants, carcinogens and certain other categories of toxicants are treated differently.

For conventional toxicants a threshold is assumed, and the experimental data are used to estimate it, usually by taking the lowest no-effect dose and applying a series of safety factors that have been developed in the folklore of toxicology to allow for the genetic variability of the population and other factors. A threshold is assumed because the kinds of toxic effects in question are those in which a substantial number of cells must be functionally impaired to produce an adverse effect on the whole organism. For example, most of us could have a few liver cells damaged with no ill effects, since proliferation of new cells would compensate for the loss. It is only when a large portion of the cells in a particular organ are affected that the body is unable to compensate and a toxic effect occurs. In chemical carcinogenesis, on the other hand, a complex and poorly understood process occurs in a single cell and transforms that cell into a tumor cell that will multiply uncontrollably and eventually kill the host. However the chemical carcinogen participates in that process, it is likely that a single molecule or only a few molecules are involved. For that molecule to reach the cell's vulnerable point, another complex series of metabolic transformation and transport (pathways) must occur. That process may not be linear in dose, of course, and may include a large number of minor, virtually immeasurable, pathways along with the major pathways. In only a few cases is anything known about the major pathways, and it is impossible to reliably link knowledge about metabolism to the carcinogenic process.

For these reasons, the federal agencies involved in regulating carcinogens have reached the policy judgment to treat all carcinogens as though there were no threshold to their action. This means that when there is evidence that a substance causes cancer at high doses, it should be assumed that there is some risk at low doses as well. This assumption is consistent with what is known about carcinogenesis, but cannot be tested scientifically. This does not mean, however, that the substance should be banned or otherwise regulated. The decision about whether to regulate depends on a balancing of risks against benefits.

Evidence of Risk

Three kinds of evidence typically indicate that a substance is a carcinogen. First, in a few cases, there is clear-cut human evidence. This can be expected only when there are well-defined highly exposed and unexposed groups in the population, as with those who smoke and those who are exposed to some occupational chemicals, or when the tumors involved are of such a rare type that any clustering is strong evidence, as with liver angiosarcoma caused by vinyl chloride. While such evidence should obviously be exploited when it exists, it is likely to be available only in these special circumstances.

Second, there may be ambiguous human evidence in a few cases. When the exposures are low, as is usually the case with environmental contaminants, the magnitude of the effects is also likely to be small and therefore difficult to detect in an epidemiological study. When a positive association is observed, it is difficult to be certain whether it is caused by the substance or substances under consideration or by some other unrelated factor that happens to be correlated with the substance or by possible confounding factors. In the case of organic contaminants in drinking water, most of the dozen epidemiological studies that have been done demonstrate an association between cancer rates and some aspect of drinking water. Yet such association does not prove that chemicals in drinking water cause cancer but rather adds weight to the hypothesis of an effect on cancer rates.

Finally, in most cases, we will be forced to rely on animal experiments. Unlike the second type of epidemiological study, most animal studies generally show that the effects observed are in fact caused by the chemicals being studied. But these studies have their own problems. The two most obvious are extrapolation from animals to humans and from high to low doses, the latter problem being shared with most occupational epidemiology studies. In any case, the federal regulatory agencies have judged that substances that cause cancer in animals present a risk of cancer to humans at low doses. This consensus has been expressed in the National Academy of Sciences' *Drinking Water and Health,* a report produced under a mandate of the Safe Drinking Water Act.[3]

This consensus reflects a social decision that has been made in the face of scientific uncertainty. In most cases concerning environmental exposures to carcinogens, science can neither prove nor disprove that a risk exists, although the judgment that a risk does exist at low doses is consistent with what is known about carcinogenesis. Furthermore, since epidemiology is usually not sensitive enough to detect possible effects of carcinogens, it will also be too insensitive to verify that a reduction of cancer risk has in fact occurred after regulatory action. Thus, not only do we not know for sure whether regulatory action will actually reduce the incidence of cancer (and, of course, nobody knows for sure that they will not), but we will also probably never know from direct empirical evidence. The only hope of reaching greater certainty in this area is through greater understanding of the fundamental mechanisms of carcinogenesis and the metabolism and transport of chemicals in the body.

Risk Extrapolation

Until a few years ago substances that caused cancer in animals were regulated without attempts to measure the impact of the regulations. The balancing of risks and benefits required under the pesticide law, for example, was largely a matter of determining whether substitutes existed for particular uses: if a substitute was available, it was generally assumed that the benefits of use were smaller than the risks.[4] Air and water pollution control requirements were based on use of the best available control technology.

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[3] National Academy of Sciences, Safe Drinking Water Committee of the National Research Council, *Drinking Water and Health* (Washington, D.C.: NAS, 1977).

[4] Federal Insecticide, Fungicide, and Rodenticide Act of 1972 (86 Stat. 973).

Recently, however, interest has increased among regulatory agencies in the use of mathematical risk extrapolation models to derive quantitative estimates of the risks posed by exposure to carcinogens. This interest has evolved from several sources. Since the agencies' resources are limited and the regulatory process arduous, some way was needed to set priorities, to determine which of the thousands of potential regulatory targets should be high on the list. There was also concern that action might be taken in cases where the exposures were so small that the effects would be insignificant when evaluated. There was a feeling in some of the agencies that decisionmakers should have some estimate of the magnitude of the risks they were attempting to reduce, no matter how crude.

It is probably fair to say that the Environmental Protection Agency took the lead, among regulatory agencies, in applying risk extrapolation models to actual regulatory decisionmaking involving chemical carcinogens. Some of the models had been used for decades in the regulation of exposure to ionizing radiation, and the Food and Drug Administration had made limited use of them in regulating food contaminants. The process was tried out as part of the rulemaking on air emissions of vinyl chloride and has been institutionalized in the agency through establishment of the Carcinogen Assessment Group in 1976. Other agencies have adopted risk extrapolation models to varying degrees, and a recent decision of the Supreme Court, though ambiguous, appears to require some form of quantitative estimate of risk levels as a prerequisite for regulation under the Occupational Safety and Health Act of 1970.[5] The federal government's ambivalance toward these methods is indicated by the interdepartmental Regulatory Council's official government-wide policy statement, "The Regulation of Chemical Carcinogens," which says, in part, the following:

> After it has been determined that a chemical substance is likely to be carcinogenic, the next step in regulatory decision-making is to assess the risk that people face of developing cancer from their exposure to the substance. . . .
>
> The form, methodology, and elaborateness of the assessment may vary substantially. . . .
>
> Except where a statute, as in the case with the Clean Water Act, explicitly indicated which substances are to be controlled and how, every regulatory proposal will be accompanied by some form of risk assessment which includes,

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[5] *Industrial Union Department, AFL-CIO* v. *American Petroleum Institute*, 448 U.S. 607, 100 S.Ct. 2844, 48 U.S.L.W. 5022 (1980). The statutory citation for the Occupational Safety and Health Act of 1970 is 84 Stat. 1590.

> at a minimum, an analysis of the substance's carcinogenicity and a determination that people are likely to be exposed to the substance. . . .
>
> Where the available data are scientifically adequate to support them, quantitative risk estimates can provide useful information for proposed regulatory decisions. . . . However, quantitative risk estimates are not yet sufficiently developed to be regarded as more than rough indicators of the level of human risks.[6]

Thus, the Regulatory Council has taken the position of encouraging the agencies to use risk extrapolations but is not willing to require their use. One of the concerns about the use of extrapolation models is based on the limitations of the models themselves. Those that are usually used to assess risk fall into two groups. The first, of which the Bryan-Mantel procedure is the best known,[7] is based on the assumption that the fraction of the population responding at a range of doses follows a log-normal function; the underlying assumption is that the susceptibility of individuals in the population is log-normally distributed. This leads to a mathematical formulation that never reaches zero as dose approaches zero but that falls off much faster than linearly. The second group of models, which includes the one-hit, multiple-hit, and linear no-threshold models, all have linear dose-response curves at low doses.[8] These models are based on a conception of carcinogenesis in which infinitesimal increments of doses randomly cause changes in target cells (for example, molecules that bind to DNA), some portion of which will transform the cell into a tumor cell.

Both sets of models are rich enough mathematically to fit the experimental data as well as anyone could ask (at least when the data are monotonic). Thus, one cannot distinguish between them empirically on the basis of fit. And for both of them, doses are typically extrapolated by factors of perhaps one thousand to one million. With such a range of extrapolation, the scientific certainty of the results is unclear at best. These extrapolations fall far short of being predictions based on a chain of logic whose steps are empirically verified. Since present knowledge does not rule out the possibility that a dose threshold may in fact exist, particular environmental carcinogens may actually pose zero risk. It is also not difficult to develop a model in which the response would be higher than that predicted by the one-hit model.

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[6] Regulatory Council, "Regulation of Chemical Carcinogens" (September 1979), pp. 8–10.

[7] Nathan Mantel and W. Ray Bryan, "Safety Testing of Carcinogens," *Journal of the National Cancer Institute*, vol. 27 (August 1961), pp. 455–70.

[8] P. Armitage and R. Doll, "Stochastic Models of Carcinogenesis," in *Proceedings of the 4th Berkeley Symposium on Mathematical Statistics and Probability*, vol. 4 (University of California Press, 1961), p. 19; and K. S. Crump and others, "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," *Cancer Research*, vol. 36 (September 1976), pp. 2973–79.

In spite of the models' serious shortcomings, there is a need, perceived by both the agencies and their critics, for some kind of answer to the question, "What are we buying with this regulation?" In response to this need, there has been a tendency to choose as a working model the linear no-threshold model and its variants. It has a preferred place both in the Environmental Protection Agency guidelines issued in 1976[9] and in the Interagency Regulatory Liaison Group guidelines issued in 1979.[10] The reasons usually given for this choice are that the model is conservative and is consistent with what is known about the biological mechanism of cancer. By the term *conservative* people seem to mean that the model gives higher risk estimates than other models. So few usable data exist for low doses that no one really knows whether the linear model is in fact conservative. The linear or one-hit model is consistent with the theory of carcinogenesis as involving random binding to a cell's DNA, but it is not consistent with the other parts of scientific knowledge. In many cases, the substance that interacts with the DNA is not the same as what people swallow or breathe, but rather a metabolite of the contaminant found in the environment. Metabolites can be more or less toxic than the original compound. In fact, the way the body deals with a particular carcinogen is likely to consist of a complex sequence of metabolites and competing pathways, one or more of which may be involved in carcinogenesis. These various processes may be more or less active at different doses, so the effects may not be linear with dose.

Probably the best thing that can be said about the linear one-hit model is that it is the simplest index of carcinogenic risk scientists have been able to develop. If compound A produces tumors in 50 percent of the experimental animals at a given dose and compound B produces the same response at 1/100 of that dose, and if both are present in the environment at the same levels, then we should probably be one hundred times as worried about compound B as about A. This logic is basically what the linear model generates. Unfortunately, things are not quite that simple.

[9] U.S. Environmental Protection Agency, "Interim Procedures and Guidelines for Health Risk and Economic Impact Assessments of Suspected Carcinogens," *Federal Register,* vol. 41 (May 25, 1976), pp. 21402–03.

[10] Interagency Regulatory Liaison Group, "Scientific Bases for Identification of Potential Carcinogens and Estimation of Risks," *Federal Register,* vol. 44 (July 6, 1979), pt. 3, pp. 39859–79.

Many questions or methodological choices have to be resolved before applying the linear model or one of its variants. These questions include the following:

1. How can animal doses in a laboratory experiment be converted to equivalent human doses? The most popular solutions are to scale the conversion either on body weight or on surface area.

2. How can the data from different experiments and different species-sex-dose groups in a single experiment be combined? For example, the most sensitive single point in the data could be used or some method of curve fitting to all the data could be adopted.

3. Given the possibility that the observed effect may not be caused by the substance in question, should data from human epidemiological studies be used, and if so, how?

4. How can the statistical uncertainty in the animal tests resulting from the limited sample sizes used be accounted for?

5. How can intermittent dosing schedules—for example, doses given five rather than seven days a week—be accounted for?

6. How can data from experiments in which the dosing does not continue for the animals' lifetime be incorporated?

7. How can data from experiments in which animals are killed before their natural deaths be incorporated?

8. How can differing mortality in various experimental groups be incorporated?

9. How should benign tumors or other lesions that, in the opinion of some experts, would have been likely to develop into malignant tumors in the future be dealt with?

10. How can data on the time of tumor occurrence be incorporated?

11. How should situations in which a substance appears to cause tumors at more than one site be dealt with?

12. How can the fact that humans are frequently exposed to substances in utero and experimental animals usually are not be accounted for?

This list is probably not exhaustive, and legitimate differences of opinion exist on all or most of the points. The impact of these questions is significant. For example, the first on the list, conversion from animal to human equivalent doses, changes the result by a factor of thirteen when extrapolating from data on mice. The range of outcomes that would result from reasonable solutions to each of these problems could easily be greater than a factor of one thousand. This is not just an abstract possibility, as two examples will show.

The National Academy of Sciences produced two reports within a year of each other that use the same experimental data and contain risk extrapolations for chloroform using the one-hit model. In one report the lifetime cancer risk was estimated at 2 times 10^{-7} for each microgram of chloroform consumed per day.[11] In the second report the risk was estimated at 22 times 10^{-7} for every microgram of chloroform consumed per day.[12] These estimates differ by a factor of eleven. A second example is the results of a comparison the Environmental Protection Agency made recently between the risk extrapolations made by the National Academy of Sciences for a number of different carcinogens and those made independently by the agency's Carcinogen Assessment Group for some of the same substances. Some comparisons were very close, but others differed by factors as large as one hundred in both directions.

The magnitude of these ranges is important: it is hard to imagine many cost-benefit analyses whose conclusions would not be changed if one side of the balance were multiplied or divided by one hundred or one thousand. The situation is even more precarious if a true risk of zero is considered possible, since the possibility cannot be excluded that a threshold may actually exist, or that the risk could be larger than calculated if the linear model is not conservative enough or if synergistic effects occur with other environmental contaminants.

This range of uncertainty is not as serious if one requires less from the models—that is, not an absolute estimate of the number of cancer cases anticipated but only a comparative index for setting priorities, based on expected carcinogenic potency, and for getting a very rough idea of a problem's seriousness. For these purposes, it probably does not matter much which way the questions listed above are resolved. What is important is that a single methodology be generally accepted, so that comparisons can be made among cases.

The Trihalomethane Regulation

It is clear from this discussion that estimating the health benefits of carcinogen regulations is fraught with uncertainty. Further difficulties in applying cost-benefit or risk-benefit analysis can be illustrated by the example of the regulation of organic contaminants in drinking water.

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[11] National Academy of Sciences, Panel on Low Molecular Weight Halogenated Hydrocarbon of the Coordinating Committee for Scientific and Technical Assessments of Environmental Pollutants of the National Research Council, *Nonfluorinated Halomethanes in the Environment* (NAS, 1978), p. 197.

[12] National Academy of Sciences, *Drinking Water and Health*, p. 716.

Recently promulgated was a regulation for trihalomethanes, which set a maximum contaminant level of 100 micrograms per liter, applicable to all public water systems serving ten thousand or more people.[13] Trihalomethanes are a group of compounds, of which chloroform is the most common, which form during water treatment as a by-product of disinfection with chlorine. Varying amounts of these compounds, therefore, are found in all public water systems that chlorinate their water and are most concentrated in systems whose raw water contains relatively large amounts of naturally occurring organic matter.

The Environmental Protection Agency performed a cost-benefit analysis for this regulation. The agency had to assume that (1) the trihalomethane levels found in a series of nonrandom surveys were representative of the entire nation; (2) specific treatment technologies would be chosen by systems to comply with alternative standards under various conditions and with the trihalomethane standard set at different levels; (3) the trihalomethanes other than chloroform had the same carcinogenic potency as chloroform; and (4) other disinfection by-products pose no health risks. The agency also had to pick a risk extrapolation method.

There are problems with these assumptions, but the limitations in the available data forced the agency to make them. Under these assumptions, the agency made a point estimate of the number of cancer cases avoided as a result of the promulgated regulation—322 cases per year. The costs of compliance had been estimated at $18.7 million per year. The agency was thus able to derive an average cost per case avoided of about $60,000 and a marginal cost per case avoided (relative to the maximum contaminant level) of about $200,000. It is unclear what significance should be ascribed to these figures. It is our impression that the trihalomethane regulation compares favorably in terms of cost per life saved to other regulations for which similar computations have been made, but it is not known if this has any meaning without determining whether the assumptions and methodologies are consistent.

What is perhaps more interesting is that the information captured by this calculation does not include the considerations the agency wrestled with in its attempt to balance costs and benefits. For example, there was concern that the regulation of trihalomethanes not interfere with adequate disinfection of water, particularly in smaller systems where unsophisticated operators might try to reduce trihalomethane levels by reducing the amount of chlorination. The agency therefore proposed that the standard not be applied to systems serving populations below seventy-five thousand, a position modified in the final regulation to include phased implementation down to systems serving ten thousand people. Similarly, it was the fear of making too great a change in a process closely intertwined with disinfection that led the agency to set the standard as high as 100 micrograms per liter, although the agency believes that much lower levels ultimately will be attainable.

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[13] Environmental Protection Agency, "National Interim Primary Drinking Water Regulations: Control of Trihalomethanes in Drinking Water," *Federal Register*, vol. 44 (November 29, 1979), pt. 3, pp. 68624–707.

The agency also wrestled with another aspect of the trihalomethane problem. Little is known about the chemistry and toxicology of the byproducts of disinfectants other than chlorine whose use would be stimulated by a trihalomethane regulation. Since any disinfectant is a biologically active substance that kills bacteria, this is an area of serious concern. The agency has attempted to deal with the immediate problem by writing provisions into the regulation that limit the addition of other disinfectants to water with high disinfectant demand. The agency has also initiated a series of research programs to gather basic data in this area. Ultimately, a limitation on disinfectant demand may be implemented that would force systems to clean up the water before adding disinfectants. The important point is that these are considerations that do not easily fit into the cost-benefit analysis, yet they are the ones that are most bothersome. It is almost as though cost-benefit analysis, at least in this case, helps deal with the easy part of the problem but does not help with the hard part.

Because cost-benefit analysis requires the inclusion of *all* costs and benefits, the Environmental Protection Agency's analysis is incomplete. The agency has not been able to quantify some of the most important aspects of the trihalomethane regulation. However, we believe that this experience is typical of attempts to use methods of cost-benefit analysis on actual problems in the field of health and environmental regulation.

In the second part of the proposed organics regulation—a treatment technique requirement for granular activated carbon or an equivalent in large vulnerable water systems to remove synthetic organic chemicals—the agency was totally unsuccessful in capturing a significant part of the problem in the cost-benefit analysis framework. Many commentors on the regulation, including the U.S. Council on Wage and Price Stability, urged the agency to perform such an analysis.[14] The council presented its own attempt, which the agency does not think is valid; but the agency has not been able to come up with a valid approach. The problem is that no one knows what levels of which chemicals would be removed with granular activated carbon. There are many thousands of organic chemicals in drinking water. Already identified are over 700, which represent only 15 percent by weight of all organic matter found in drinking water.[15] Only a few have been tested for carcinogenicity in the usual animal tests. The agency has conducted several surveys of water from different systems and has found that the levels of measurable chemicals vary widely from system to system and even from day to day. It would therefore be difficult to derive a risk estimate based on this fragmentary occurrence data, even for those measurable chemicals that have been tested for carcinogenicity. The agency has not been able to think of any way to estimate the risk to public health posed by compounds that cannot be measured or that have not been tested for carcinogenicity. Nor do methodologies exist for dealing with the potential effects of human exposure to mixtures of carcinogens, including possible synergistic effects. Yet the value of granular activated carbon is that it removes a wide variety of these compounds from water. The agency has suggested that it makes sense to spend a nominal amount, less than ten dollars per family each year, to take advantage of a known technology that will remove a variety of organic contaminants. Yet neither the agency nor its critics have been able to develop a method of analysis that would help determine whether or not granular activated carbon is worth its costs.

Thus, the agency has not found cost-benefit analysis to be a powerful tool, at least for developing a regulatory strategy to deal with the presence of organic carcinogens in drinking water. The agency is concerned that little thought has been given to the results of inserting cost-benefit analysis into the adversary process of rulemaking. It is predictable that both (or all) sides will be able to introduce analyses that support their points of view. Each will then bring forward analysts, of equivalent credentials, who will argue among themselves about the superiority of their particular

[14] Ivy E. Broder, "Analysis of Proposed EPA Drinking Water Regulations" (U.S. Council on Wage and Price Stability, September 1978).

[15] National Academy of Sciences, *Drinking Water and Health*, pp. 489–92; and Environmental Protection Agency, Office of Water Supply, "Statement of Basis and Purpose for an Amendment to the National Interim Primary Drinking Water Regulation on a Treatment Technique for Synthetic Organic Chemicals" (January 1978), p. 1.

analyses in a language all their own. Incomprehensible analysis may then drive out understandable argument. This might be worth enduring if the issues being debated were central to the decision at hand. However, if the drinking water regulation's problems are at all typical (and we believe they are), the cost-benefit analyses are likely to capture only a small part of the problem and deflect attention from the rest. This will not contribute to better decisionmaking.

If cost-benefit analysis becomes a legal requirement, these difficulties will be multiplied. There will be a period of uncertainty until a body of case law is developed that will show what the courts consider an adequate analysis. This delay will benefit the opponents of any change in regulations. A great deal of energy and attention would probably then be focused on how well the analysis satisfies whatever criteria have developed. Given the limitations of cost-benefit analysis, this allocation of the limited resources of time and personnel does not seem to be warranted.

An alternative to cost-benefit analysis is the use of technology-based standards. This approach is most clearly exemplified in the provision of the Federal Water Pollution Control Act Amendments of 1972[16] that requires effluent limitations on water polluters based on the "best available technology." The use of the best available technology seems particularly attractive in the case of carcinogens. Since it is assumed that there is no safe level, it seems reasonable to reduce exposures as much as possible. But the technological approach does not preclude the necessity of balancing costs and benefits. It is always possible to spend more money to reduce exposures. Even if no more advanced technology is known, more of the old technology can always be used. So there is a considerable range of judgment as to what constitutes best available technology, and the degree of stringency chosen depends implicitly on the perceived magnitude of the risk.

Another approach to regulation is to continue balancing risks and benefits intuitively. With this approach, risk extrapolation models would be used when possible. Over time, regulators would develop a feeling for the ranges of risk that will have been judged socially acceptable in previous decisions and would aim for that range of risk unless there were reasons to be more or less stringent in a particular case. When possible, regulators could even calculate the marginal cost of each cancer case avoided and develop a feeling for what ranges have been judged socially acceptable in previous decisions. But we would be wary of overformalizing the approach. Ideally, each case would lead to a creative resolution of conflicting goals.

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[16] 86 Stat. 816.

Another problem is that of communicating regulatory activities to the public. Ultimately, the success of any social enterprise in a democratic society depends on the informed support of the people. Yet many discount the reports of carcinogens in their environment and do not believe, for example, that smoking causes cancer; and others go to ridiculous lengths to avoid minuscule risks, such as avoiding products containing polyvinyl chloride for fear of getting liver cancer. Because the adversary process tempts people to either exaggerate or minimize risks, almost nobody understands that although the risks may or may not exist, it is rational to treat them as real; that any reduction in cancer rates is likely to be small but still worth achieving; and that probably no one will ever know the true impact of preventive action. Sooner or later, regulatory agencies will have to face the consequences of their failure to communicate.

Appendix: Net Benefits of Several Trihalomethane Levels

The Environmental Protection Agency published a final regulation to control trihalomethane levels on November 29, 1979.[17] The regulation imposes a maximum contaminant level allowing no more than 100 micrograms of trihalomethanes per liter of finished drinking water. The requirement for granular activated carbon filtration systems to remove all organics was not promulgated. While the trihalomethane standard was being developed, other potential levels were suggested. There was a vigorous discussion of the cost estimates of control technology, and many issues related to risk estimation were raised. Within this highly charged atmosphere, the agency was challenged to analyze the economic efficiency of the proposed standards. As already discussed, there are several practical difficulties associated with refined cost-benefit analysis. The following case study presents the basic methodology used to determine the net benefits of alternative levels for the trihalomethane standard and discusses the dramatic effect produced by changes in basic assumptions.

Benefits associated with control of trihalomethanes essentially consist of a reduction in the number of cancer cases that could be attributed to the contaminant. The National Academy of Sciences presents the general formula for estimating cases avoided in *Drinking Water and Health:*[18] annual number of cases avoided equals RVT/E, where R equals risk per microgram per liter, V equals liters per day consumption, T equals total (national) reduction in exposure (in micrograms), and E equals life expectancy.

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[17] Environmental Protection Agency, "National Interim Primary Drinking Water Regulations."

Knowing the number of cases avoided allows for estimation of the dollar value of the benefits. Assuming that each case avoided is a life saved and that it is possible to ascribe a value to human life, the annual benefits are merely the cases avoided times the value per life. However, these benefits vary depending on selection of risk estimates, reduction in exposure, and value of life.

Estimation of the risk posed by each microgram per liter of drinking water is a function of many variables. The Environmental Protection Agency's Office of Drinking Water reviewed the methodology used to develop two different risk estimates, one presented by the National Academy of Sciences and the other by the agency's Carcinogen Assessment Group.[19] The estimates are essentially the same, although arrived at using slightly different assumptions. A value of 2.08 times 10^{-6} cases per microgram per liter was used. The estimate reached by the Carcinogen Assessment Group was used in the analysis presented here to maintain consistency within the agency. The estimate is based on data from several animal studies and derived from a linear no-threshold risk model.

The consumption of drinking water per day varies according to climate and individual habits. Although recent data suggest that average daily consumption may be as low as 1.2 liters, the National Academy of Sciences' estimate of 1.95 liters per day was used in this analysis. Also taken from the academy, and generally accepted by analysts, is a life expectancy of seventy years.

The remaining independent component is the total reduction in exposure to trihalomethanes. The agency developed an estimate of this parameter. In a perfect world, one would first ascertain the exposure of the public to trihalomethanes and then assess how that exposure would change through use of various engineering alternatives. Unfortunately, it was not possible to define the exact preregulation exposure. The only data available

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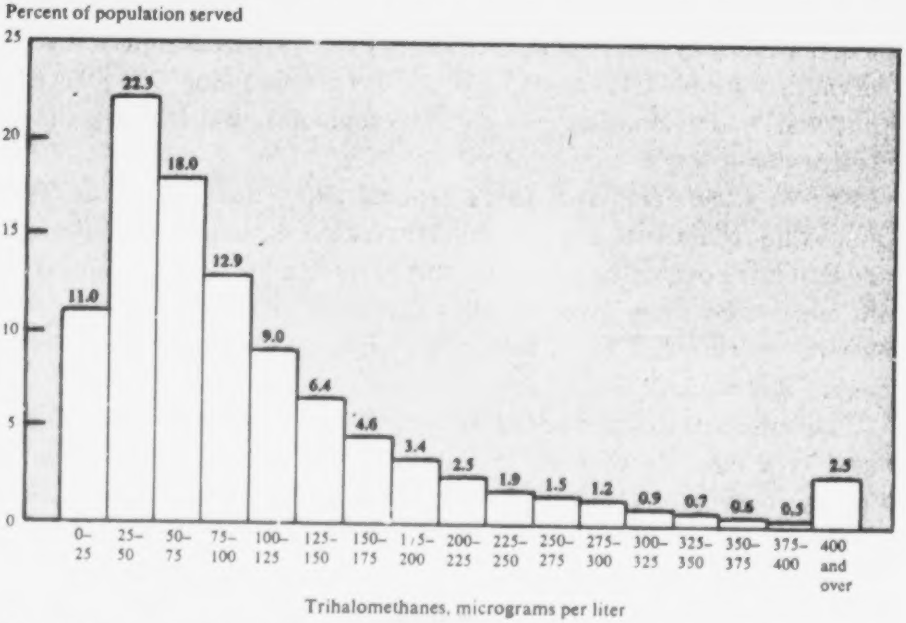
[18] National Academy of Sciences, *Drinking Water and Health*, pp. 489–92.

[19] National Academy of Sciences, *Drinking Water and Health;* and Environmental Protection Agency, "Interim Procedures and Guidelines for Health Risk and Economic Impact Assessments of Suspected Carcinogens."

Figure 1. Percentage of Population Served by Surface Water Systems according to Trihalomethane Level (Preregulation, 1977)[a]



Source: Memorandum to Arnold M. Kuzmack from John Clark and T. J. Glauthier of Temple, Barker and Sloane, Inc., February 25, 1977.

a. Includes only systems that chlorinate.

to assist in this estimate was the National Organic Monitoring Survey, which analyzed the water from 113 water supplies representing various types of sources and treatment processes.[20] About nineteen different contaminants were measured, including some of the trihalomethanes. These data were used to represent the preregulation distribution of trihalomethanes in water supplies. Although taken from a nonrandom sample, the data are the only basis upon which to estimate trihalomethane exposure. The number of people served by surface and ground water systems of various sizes was calculated from a statistical survey of the operating and financial characteristics of community water systems. Multiplying the percentages of the population affected by the population using drinking water, weighted by source of supply, leads to an estimate of the total preregulation national exposure. Figures 1 and 2 show the distribution of people exposed to trihalomethanes by concentration of the contaminant. Table 1 shows this exposure level for various population sizes.

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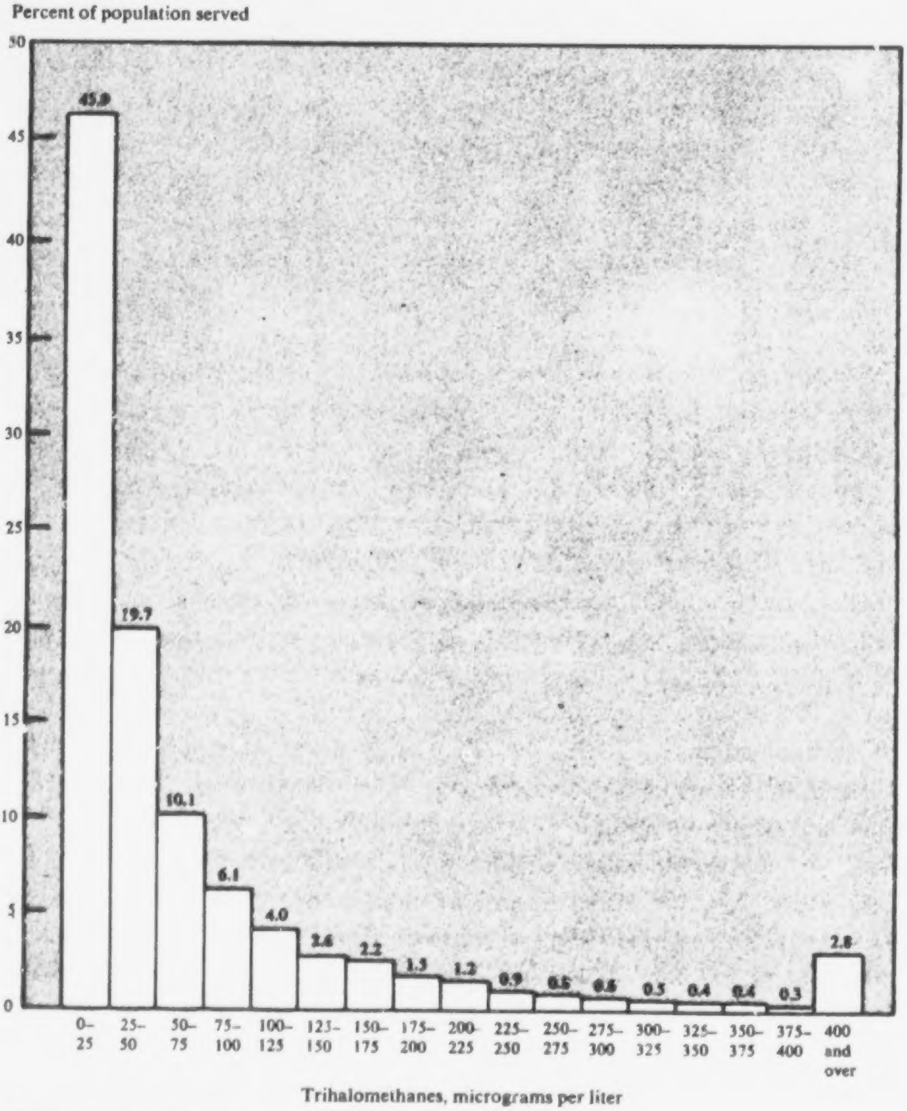
[20] Environmental Protection Agency, Office of Water Supply, Technical Support Division, "The National Organic Monitoring Survey," unpublished data.

Figure 2. Percentage of Population Served by Ground Water Systems according to Trihalomethane Level (Preregulation, 1977)[a]



Source: Same as figure 1.
a. Includes only systems that chlorinate.

The following net benefit analysis is for all systems serving more than ten thousand people—about 80 percent of the national population regulated under the safe drinking water legislation.[21]

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[21] Environmental Protection Agency, *Economic Impact Analysis of the Promulgated Regulation for Drinking Water*, EPA-520/9-79-022 (EPA, 1979), p. I-1.

Table 1. National Exposure to Trihalomethanes[a]

Level (micrograms per liter)	*Population*			
	Over 100,000	*Over 75,000*	*Over 50,000*	*Over 10,000*
Preregulation	6.1	7.1	8.6	11.4
150	4.1	4.7	5.7	7.4
100	3.1	3.6	4.3	5.8
50	2.0	2.4	2.7	4.0

Source: Calculated from U.S. Environmental Protection Agency, Office of Water Supply, Technical Support Division, "The National Organic Monitoring Survey," unpublished data.

a. The units of the table are in micrograms of trihalomethane levels multiplied by the number of people exposed, aggregated over the entire nation, or 10^6 gram persons.

To determine how the exposures presented in figures 1 and 2 would change after a regulation was implemented, a decision tree was constructed from information provided by water engineers asked to estimate the effectiveness of various water system treatments. The decision tree reflected assumptions about the combination of treatments that would be used, depending on the conditions within the different water supply plants. Further, the trihalomethane concentrations that will result from the treatments usually will be well below the maximum contaminant level standards. Only with the very expensive treatments (ozone and granular activated carbon) are final concentrations expected to be at the maximum contaminant level.

The national reduction in exposure to trihalomethanes is derived by subtracting the postregulation exposures from the preregulation exposures. These numbers, when combined with risk, consumption, and life expectancy, yield the number of cancer cases avoided each year by using various regulatory alternatives. To convert these cancer cases avoided to dollar benefits requires a simple multiplication with the value of life chosen by the analyst.

The national costs of the trihalomethane regulation are described in the economic impact analysis.[22] The particulars of the analysis are not unusual enough to warrant attention here. But it is important to recognize that even the cost analysis is subject to assumptions that can dramatically modify the national cost estimate. For example, the analysis included a contingency cost of up to 25 percent of capital to cover needs of the real world. If such contingencies are not really needed, as some environmental interest groups suggest, then costs would be significantly lower. On the other hand, certain water supply engineers, specifically

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[22] Ibid.

Table 2. Changes in Economic Efficiency with Various Risk Estimates[a]

Basis of risk estimate	*Maximum national net benefit (millions of dollars)*	*Economically efficient standard (micrograms per liter)*
Roe mouse	77	75
National Cancer Institute mouse	45	100
Roe rat	5	127

Source: Cost estimates were derived from cost data in Environmental Protection Agency, *Economic Impact Analysis of the Promulgated Regulation for Drinking Water*, EPA-520/9-79-022 (EPA, 1979).
a. A value of $200,000 per life was used in each calculation.

those now suing the Environmental Protection Agency over this regulation, believe that most systems would use the more expensive treatments, causing national costs to increase significantly.

To emphasize this point, consider the effects of a change in assumptions on risk. Table 2 represents the range of net benefits when the risk assumption is varied. The risks associated with three different animal tests were used to develop the net benefits shown.[23] The only parameter varied in the risk estimates is the set of animal data used. Models of risk estimation remain the same, and the value of life used is $200,000. Yet this relatively minor adjustment in risk results in greatly different net benefits. The maximum net benefits vary sufficiently to allow for selection of several different trihalomethane standards. This phenomenon can be reemphasized by the interested analyst through selection of different extrapolation models, which can provide risk estimates two orders of magnitude apart.

Variation in cost assumptions produces a similar picture, as table 3 indicates. The relatively modest changes in engineering assumptions described above result in not so modest changes in maximum contaminant levels, which are economically different. The point is made again in table 4, where net benefit is described for various values of life. Again, only a small range of values of life is shown. Some estimates have gone as high as $158 million per life, which was the value of life imputed from coke oven emission standards.[24] Some are as low as ten thousand dollars per

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[23] National Cancer Institute, *Report on the Carcinogenesis Bioassay of Chloroform* (Government Printing Office, 1976); F. J. C. Roe, "Preliminary Report of Long-Term Tests of Chloroform in Rats, Mice and Dogs" (unpublished paper, London, 1976).

[24] Talbot Page, Robert Harris, and Judith Bruser, "Removal of Carcinogens from Drinking Water: A Cost-Benefit Analysis," Social Science Working Paper 230 (California Institute of Technology, September 1978), app. D.

Table 3. Changes in Economic Efficiency with Various Cost Assumptions[a]

Basis of cost estimate	*Maximum national net benefit (millions of dollars)*	*Economically efficient standard (micrograms per liter)*
Public interest group	50	83
Environmental Protection Agency	45	100
Industry group	25	125

Source: Same as table 2. The public interest group and industry group cost estimates were derived from high- and low-range cost estimates.
a. A value of $200,000 per life was used in each calculation.

Table 4. Changes in Economic Efficiency Assuming Various Values of Life[a]

Value of life (thousands of dollars)	*Maximum national net benefit (millions of dollars)*	*Economically efficient standard (micrograms per liter)*
500	150	50
300	75	75
200	45	100
100	15	125
20	(−5)	150

Source: Same as table 2.
a. A value of $200,000 per life was used in each calculation.

life, which is the amount society is willing to pay to preserve the life of an unknown individual through public nursing homes.[25] Within that range, nearly any national standard could be justified.

There is no obvious way to resolve these differences of opinion about technical assumptions, assumptions about risk, or, for that matter, the value of life. It is the tenuousness of these assumptions that reduces the utility of careful cost-benefit analysis, even for the relatively straightforward trihalomethane regulation.

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[25] Ibid.

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August 22, 1982

Dr. John Hernandez, Deputy Administrator
A-101

Dr. Elizabeth Anderson, Director
Office of Health and Environmental Assessment
RD-689

U.S. Environmental Protection Agency
401 M. Street, S.W.
Washington, D.C. 20460

Dear Drs. Hernandez and Anderson,

Enclosed are the responses from the outside reviewers who were asked to comment on the document "Additional USEPA Guidance for the Health Assessment of Suspect Carcinogens with Specific Reference to Water Quality Criteria".

Also enclosed is the list of reviewers I sent the document to and my letter of response to them. This response is compatable with the comments received in-house and from scientists who participated in Jerry Starra's workshops and with discussions held in the CAG.

You will note that the letter is, of course, non-committal about what the EPA is going to do. However, the reviewers gave a clear signal to go ahead with adopting the IARC criteria for stratifying the evidence for carcinogenicity. There should be no trouble getting SAB approval for such a move and it could have a major impact on the EPA's regulatory posture for a number of carcinogens.

The quantitative issue of treating mutagenic and non-mutagenic carcinogens differently could not be adopted in its present form without provoking a strong negative reaction from an important component of the scientific community. However, the revised approach suggested in the enclosed letter to the reviewers would stand a much better chance of achieving a consensus.

I hope you will approve the initiation of steps to seek Agency approval of the IARC criteria as well as efforts to revise the quantitative approach to make it more generally acceptable.

Sincerely yours,

Roy

Roy E. Albert, M.D.
Professor and Deputy Director
Chairman, Carcinogen Assessment Group
U.S. Environmental Protection Agency

REA/mff

[illegible] of that mechanism. Further, at this [illegible] may be enough dose- [illegible] with carcinogens to support the [illegible] extrapolation model [illegible] mutagenesis as the sole basis for the [illegible]hanism.

[illegible], as noted in this document, [illegible] mathematical models [illegible] the experimental dose range, they [illegible]sed for low dose [illegible] because of wide discrepancies that appear [illegible]t low doses. Point [illegible]tes can not be obta[illegible] without knowing t[illegible] exact dose response [illegible]e the true model can [illegible]ot be obtained from [illegible]ssay data, all that [illegible]plished is to consider the uncertainties [illegible]isk assessments. If [illegible] response is curving upward, linear extrapol[illegible] below the experimental [illegible]rovides an upper limit on risk. This shou[illegible] be able to be [illegible] with bioassay data alone and develops [illegible]ument independent of, [illegible] mutagenicity data.

[illegible] it appears reasonable to use th[illegible] limits from the [illegible] model as one means for arri[illegible] [illegible]e" exposure levels [illegible]opulation, it might also be reasonable [illegible] include some reference [illegible] sensitivities of humans and test animals as a final step. [illegible]tional toxicological approach discussed [illegible] page 9 should be [illegible]d, delineated or referenced, as should the mathematical procedures [illegible] extrapolation.

[illegible] it may well be the case that this so-call[illegible] conventional toxico- [illegible] gives essentially the same answer as [illegible]linear multistage model if it is also tied to the same level of risk, [illegible] should be. The [illegible] of the so-called conventional toxicolog[illegible]oach is that it is very [illegible] akin to conventional procedures. It should be cited that [illegible] dose response level, e.g. the 10% re[illegible]se level, and ex- [illegible] early below that point has [illegible] by Van Ryzin

J. Occup. Med. 22, 1980) and by Farmer, Kodell, and Gaylor (Risk Analysis 2, 1982) except that they suggested using the 1% response level.

Also, can one really call this approach a conventional toxicologic approach, which is based on the no-observed-effect-level (NOEL). Even if the NOEL exists, it has no precise meaning because it depends on the number of animals tested and hence provides a different level of risk in each case. Since the proposed technique selects a given response level (10%) and then applies an uncertainty (safety) factor, would it not be more proper to call this approach a "safety factor procedure" or "low dose linear extrapolation", since the two are almost identical.

3. The argument in paragraph 2 on page 10 is not logical. In the previous paragraph, a factor of 10 was imposed because of the uncertainty of sensitivity of humans. If then the data are available in humans (and presumedly their sensitivity is then known), one can not justify the use of the same uncertainty factor because you now have a greater certainty. If one is uncomfortable with only a factor of 100 when the data are presumedly more reliable, then the safety factor in less certain cases should be reevaluated. Therefore, the two paragraphs at the top of page 10 could, if you like, be replaced by the following paragraph:

> If the dose response curves upward, reducing the dose by an uncertainty factor of F will reduce the risk by at least a factor of F. Thus, starting at the dose that gives a 10% response, dividing by an uncertainty factor of 10,000 should reduce the risk to below one in 100,000. If the extrapolation is based on animal rather than human data, an additional factor may be employed depending upon the relative sensitivity of humans to animals.

3. Page 8, paragraph 2, line 2. Change "to use" to read "a modification of"; line 6, change "conventional toxicological" to read "modified".

4. Page 9, paragraph 2, line 1. Change "the conventional" to read "a modified"; line 2, delete "with an added refinement of making", replace with "which makes"; line 9, after "...tumorigenic responses." add the following sentence, "Also, NOEL has no definition and is a statistical result which depends on the number of animals tested." Line 14, after "....is recommended", insert "to estimate the 10% response level"; line 16 after "....upper confidence limit.", delete period, insert "on the response."

5. Page 10, delete paragraphs 1 and 2. Please see General Comments, above.

6. Page 12, sub-paragraph D, line 6, delete "In order to incorporate evidence for mutagenicity.", begin new sentence, "It is....."; line 7, change "both" to read "either", change "and" to read "or", change "conventional" to read "modified"; line 8, delete "a range of"; line 9, change "conventional" to read "modified". The two approaches are nearly the same when a risk level of 10^{-5} is specified for both.

7. The modified exposure limit needs to be tied to risk. It is difficult to provide an exposure limit without any notion of the degree of protection that it might provide.

8. Page 13, first paragraph. Delete last sentence: "It is proposed...... ....toxicological method."; second paragraph, delete all of the paragraph down to last sentence, "Such guidance, together....". As was stated above, one could divorce the process from mutagenicity and use other reasons for low dose linearity.

9. Page 12, sub-paragraph E, line 1. Change "could" to "should". Reference last sentence of paragraph No. 1 - Why shouldn't they be employed? Can we be so comfortable with best estimates that we don't need to allow for sampling errors and experimental variability?

UNIVERSITIES ASSOCIATED
FOR RESEARCH AND EDUCATION IN PATHOLOGY, INC.

[illegible] ROCKVILLE PIKE • BETHESDA, MARYLAND [illegible] • PHONE [illegible]

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July 2, 1982

Dr. Roy Albert
Chairman, Carcinogen Assessment Group
Office of Environmental Assessment
Environmental Protection Agency
401 M Street S.W.
Washington, D.C. 20460

Dear Roy:

Thank you for your letter of June 18 and the attached draft of a proposed addendum to the U.S. Environmental Protection Agency's Guidelines for the Health Assessment of Suspect Carcinogens.

In general, I believe the document represents real progress in attempting to relate evaluation further to the scientific and biological data. I enclose some comments which you may find of some value. Please excuse typographical and grammatical errors as I have not had as much time for polishing as I would like.

Sincerely yours,

John

John Higginson, M.D.
Senior Scientist

JH/ls

Enclosure

CORPORATION

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Medicine and Environmental Health Department
Research and Environmental Health Division
Toxicology
RICHARD F. EGAN, Ph.D. - Director

July 1, 1982

82XR 1064

Roy E. Albert, M.D.
Chairman
Carcinogen Assessment Group
Office of Environmental Assessment
United States Environmental Protection Agency
Washington, DC 20460

Dear Roy:

I'm writing in reply to your June 21 letter to Vaun Newill. Vaun is out of the office, and in order to meet the rapid turnaround you requested, I have taken the liberty to respond in his absence.

Let me begin by saying that I am refreshed to see the Agency's interest in improving regulatory decision making procedures, particularly with respect to putative carcinogens. As your draft guidance document states, there is growing awareness of the need for flexibility in regulatory processes.

I note, however, a very one-sided view of the state of scientific knowledge of carcinogenicity. The EPA-modified IARC classification system considers only those data which support the conclusion that an agent under study is carcinogenic. This system of "stratification of evidence for carcinogenicity" is entirely blind to data from studies which show no carcinogenic effect, no matter how outstanding the effort or persuasive the results. "Adequate" evidence of non-carcinogenicity must be considered in a comprehensive and balanced assessment of cancer risk. The adequacy of negative findings from carcinogenicity studies can just as surely be judged as can the adequacy of negative findings from mutagenicity tests. Although I am prepared to accept that more rigorous criteria of adequacy will be required for negative findings than for positive findings, I feel that the acceptance of credible negative results (with some estimate of the "strength" of those results) is imperative. I suggest that the classification strata include a fourth set..."Adequate evidence against carcinogenicity".

3 of 7

The modification of the IARC classification system, as with the criteria for inclusion in Group 3, is particularly inappropriate. IARC recognized that some materials simply "cannot be classified" as to carcinogenicity. In the EPA draft document, this recognition has been restated as equivalent to "possibly carcinogenic". The terms "not classifiable" and "possibley carcinogenic" are simply not equivalent; whenever data are inadequate to support a conclusion, nothing more can be said. The inference the reader draws from EPA's modification of the IARC classification is that the Agency has presumed that all agents are suspect as carcinogens unless proven to be so...that is to say, all agents are classifiable as either possibly, probably or surely carcinogenic.

The notion that quantitative estimates of cancer risk may be legitimately made on the basis of mutagenic potency is neither theoretically nor empirically defensible. From the multi-stage model of the carcinogenic process, there is no reason to assume any direct relationship between the rate/frequency/incidence of the first stage event (mutation) and the rate/etc. of the ultimate event (clinical cancer). Even for those materials which have been shown to be both mutagenic and carcinogenic, very poor quantitative correlations between the two effects have been seen. To have assumed that the carcinogenic potencies of various agents were directly proportional to their mutagenic potencies would have required the following unsubstantiated and/or unjustified assumptions:

Don't understand

- That the mutation is the rate limiting step in the carcinogenic process.

- That the dose response functions for mutagenesis and carcinogenesis are parallel.

There are substantial empirical data to refute the assertion that carcinogenic potency (and associated risk) can be quantitatively estimated from the mutagenic potency of a particular material. In fact, work recently done by the American Petroleum Institute with highly-water-insoluble complex mixtures has failed to demonstrate even a qualitative relationship. This should not have been surprising; the results of mutagenicity bioassays (both bacterial and mammalian cell mutation tests) have proven to be highly method-sensitive and operator-sensitive. Results of carcinogenic bioassays are less method- and/or operator-sensitive but still vary sufficiently to support little hope for close correlation with the results of mutagenicity tests. For these reasons, the results of mutagenicity tests are totally useless for quantitative cancer risk assessment. At the very most, data from these studies may be used to qualitatively distinguish genotoxic from presumable non-genotoxic carcinogens. Even this application of the data must be done cautiously...with an eye to the method- and operator-sensitive nature of the results.

I take a rather simplistic view of the distinction between mutagenic (genotoxic) and non-mutagenic (non-genotoxic) carcinogens. A cancer is a cancer, by whatever mechanism may have lead to it. The only clinically important distinctions which have been revealed by animal experimentation are that non-genotoxic [illegible] tend (1) to be active only at higher doses (relative to genotoxic carcinogens) and (2) to display a "flatter" dose-response relationship. In either case (genotoxic or not), each increment of exposure is associated with some increment of risk, however small. All that separates the types of carcinogens, for practical purposes, are the vastly different rates at which the incremental risks accumulate...as functions of exposure.

The conventional toxicological approach has served the science and regulatory agencies well even when chronic, debilitating and irreversible health effects, such as chronic obstructive lung disease, were considered. My suggestion is that the "conventional toxicological approach", tempered by the judgment of trained, experienced and unbiased experts, be used in the standard setting process regardless of the health effect of concern. I feel a scientific policy decision will have to be made by our national policy makers as to what is a socially acceptable level of health risk. It then becomes the task of the toxicologist-oncologist-epidemiologist to estimate the relationship between exposure and risk, of the regulator to establish the target (or limit) for exposure, and of the environmental control technologist to devise the cost-effective means of achieving the target.

As for the passage entitled "Conventional Toxicological for Non-Mutagenic Carcinogens", I find it an odd mosaic of elements...ranging from relatively conservative to potentially quite lenient.. from practically reasonable to totally arbitrary. For instance, the use of the ED_{10} is attractive, and its use is well argued in the document. Its principal weakness, compared to the maximum NOEL, is that the ED_{10} can be (experimental circumstances determining) a much less conservative basis for extrapolation.

NOTE: As a parenthetical comment, I note an interesting conjunction in the document. This draft implies that the "NTP-type carcinogenesis bioassays" are "well conducted". In my opinion, they are certainly not well designed, at least, not for the generation of data suitable for use in extrapolative risk analyses. Their goal is to qualitatively segregate carcinogens from non-carcinogens...and the data are not suited to other ends.

The relative merits of dose determinations on the basis of maximum likelihood versus 95 percent upper confidence estimates are fairly and adequately covered. But the choice of the "uncertainty factor" is unnecessarily arbitrary. The ten-fold factors are apocryphal and enjoy no substantial basis. Recognizing the considerable heterogeneity within and among species, a ten-fold divisor for each is, in principle, not unreasonable. But, there is probably more variety both within and among species in their handling of non-mutagenic carcinogens than in their handling of mutagens, particulary if the mutagens are either direct-acting or require relatively little/non-specific metabolic activation. That is to say, the dose-response relationships for non-genotoxic carcinogens are "flatter" than those for genotox agents. In "conventional toxicologic" data analysis for standard setting, the principle is commonly applied that the flatter the dose-response relationship, the greater will be the required margin of safety over the NOEL.

The third factor of 10 is totally without scientific foundation and should not be mistaken for anything other than an arbitrary factor inserted for some non-scientific reason (humanistic sensitivity or political expedience).

The insistence that the first factor (to compensate for inter-species variation) is required, even when definitive data are available for man, seems illogical. If that ten-fold uncertainty factor is required for an adequate margin of safety, even when solid data are available, why wouldn't a larger uncertainty factor be in order when even less is known about the agent's effects in man?

The "criteria for evaluating...gene mutations" are reasonable.

I would like to have the "usual considerations that go into regulatory decision making" (see bottom page 13) listed and described in some detail.

Finally, I have no technical comments on the "options" as described in Section-E; however, a couple of philosophical observations are in order with respect to option 3. First, I feel have no objection to a

6 of 7

variety of standards (i.e., in different states) depending upon the environmental circumstances, the site, the water use, etc. Second, I feel that graded uncertainty factors are no more arbitrary than the safety factors suggested in the "conventional toxicological approach".

I hope these comments are helpful. I would be happy to answer any questions you might have. You may write me at the above address or call (201)273-6063.

Sincerely,

Steven C. Lewis, Ph.D.
Toxicology Associate

SCL:pai
Attachment

FROM: Doris Gillespie -755-3968
EPA WASH DC
FAX (755-7051)

7 of 7

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July 9, 1982

Dr. Roy E. Albert
[illegible] Carcinogen Assessment Group
Office [illegible] Environmental Assessment
Office of Research and Development
United States Environmental Protection Agency
Washington, D.C. 20460

Dear Roy:

Additional U.S. Environmental Protection Agency guidance for the health assessment of suspect carcinogens

Thank you for sending me the above document for review. I apologize for not responding more quickly. My comments will be confined to the first of the two issues addressed by the document, i.e., stratification of the weight-of-evidence for carcinogenicity. My experience in the utilization of mutagenicity data in the quantitative aspects of risk assessment is very limited as is my knowledge of the necessary mathematical manipulations.

I was very interested to find that EPA proposes to adopt the IARC methods for stratification of evidence for carcinogenicity. I have taken part in several meetings at IARC Lyon at which these methods were developed and used and I believe them to be the best that are currently available.

However, in reviewing Section B.1.3. of the EPA document I am not happy with the change from the IARC wording for Group 3 (Page 7, line 4). I prefer the original IARC version which clearly states that chemicals in this category cannot be classified as to their carcinogenicity in human beings. The EPA version seems to me to have a very different meaning by stating that these chemicals are "possibly carcinogenic for humans." In a sense the statement is a platitude because all chemicals for which the evidence is inadequate must be considered possibly carcinogenic but, in my opinion, to state it in this way could lead to misleading interpretation particularly in the law courts. I would recommend the retention of the original IARC wording.

I believe that the increasing evidence in support of the existence of genotoxic and non-genotoxic (or epigenetic) carcinogens warrants the introduction of this concept into approaches to the definition of exposure limits to certain carcinogens. This, of course, implies support for the "conventional toxicologic approach to defining exposure limits in terms of the no-observed-effect-level of response together with an uncertainty factor."

1of7

I am less happy with the selection of 1,000 for this factor. I suppose that one must recognize the arbitrary nature of the factor and I cannot make a very convincing case for an alternative. However, a factor of 1,000 does seem rather high to me if we really accept that some carcinogens do not act by the genotoxic mechanism and should not therefore be subjected to quantitative risk evaluations that are based on the assumed irreversibility of genotoxic (mutagenic) injury.

If I can be of any further assistance please let me know.

With best wishes.

Sincerely yours,

Peter

P.N. Magee

PNM:ls

2of7

INHALATION TOXICOLOGY RESEARCH INSTITUTE
LOVELACE BIOMEDICAL AND ENVIRONMENTAL RESEARCH INSTITUTE, INC.
P.O. Box 5890 Albuquerque, New Mexico 87185

July 2, 1982

Dr. Roy E. Albert
Chairman
Carcinogen Assessment Group
Office of [illegible] Environmental Assessment
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Roy:

I and several members of our staff have reviewed the proposed addendum to the U.S. Environmental Protection Agency's guidelines for the assessment of suspect carcinogens entitled:

> "Additional U.S. Environmental Protection Agency Guidance for the Health Assessment of Suspect Carcinogens with Specific Reference to Water Quality Criteria"

The following are our general concerns about the document.

A. Even well informed readers, including many toxicologists, are not likely to know what the multistage extrapolation model used to calculate the 10% response is. They will also not know how in vitro mutagenesis assay measurements can be used to calculate a cancer risk probability of 10^{-5} for people. These are two crucial pieces of information that are necessary for reviewing the appropriateness of this addendum. Clear explanations of these procedures should be included in the addendum or references should be made to other reports that can be easily obtained. As it now stands, it would be extremely difficult for anyone to independently calculate the risk factors given in Table 2 of the document.

B. Although one clearly stated purpose of this addendum is to stratify weight-of-evidence judgements about cancer causing agents, too many adjectives are used. The words, sufficient, limited, inadequate, strong, confounding, suggestive, almost sufficient, possibly, adequate, inconclusive, negative, are used to describe evidence of chemical carcinogenicity. None of these are defined very clearly. The number of these terms used should be limited and those used need clear definition.

C. Two concerns are likely to occur to readers of this addendum that should be discussed, even if they cannot be treated in a satisfactory way. These include exposures to mixtures of chemical carcinogens and to potential carcinogens for which no toxicological information is available. Exposures to mixtures of carcinogens and new chemicals is likely to occur for most people.

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D. Using a 10 cancer response level developed from studies in laboratory animals or people to estimate numerical exposure guidelines needs further definition. Does this criteria apply only to single types of tumors? Are these for life span studies only or for studies of shorter duration?

E At least two parts of this addendum attempt to provide detailed discussions of factors that remain very arbitrary. These should be simplified. [illegible] use of the safety factor of 1000 described on page 10 and use of aritnematic or geometric means of the calculated acceptable levels of exposure to carcinogens after page 12. These are arbitrary judgements and should be stated as such.

Overall, we feel that giving more attention to the above factors would improve the quality and usefulness of the addendum. If any of these concerns are not stated clearly in the above discussion, please feel free to contact me (FTS – 844-6835), Dr. Charles Hobbs (FTS – 844-2435) or Dr. Richard G. Cuddihy (FTS – 844-7544) for a further explanation.

Sincerely,

Roger O. McClellan

Roger O. McClellan, D.V.M.
Director

RGC:ROM:eb

xc: Dr. C. H. Hobbs
Dr. R. G. Cuddihy

McARDLE LABORATORY
FOR CANCER RESEARCH
DEPARTMENT OF ONCOLOGY
MEDICAL SCHOOL, UNIVERSITY OF WISCONSIN

June 28, 1982

Dr. Roy E. Albert
Chairman, Carcinogen Assessment Group
Office of Research and Development
United States Environmental Protection Agency
Washington, D.C. 20460

Dear Roy:

We have studied your draft of the proposed addendum to the U. S. EPA guidelines for the health assessment of suspect carcinogens with reference to water quality criteria.

We are pleased to see that the EPA is reconsidering its guidelines with a view to consideration of both the quality of the data and the biological properties (e.g., whether or not mutagenic) of the chemicals under consideration. We do not have sufficient time at present to study the draft intensively; thus we are not ready to approve or disapprove it. However, we do have the following comments for your consideration:

1. We do not find the classification of chemicals with regard to their carcinogenic risks to be adequate.

    a. Groups 1, 2A, and 2B are acceptable classifications as described on p. 6.

    b. Your definition for chemicals in Group 3 is inadequate and misleading. Especially among lay persons, but also among some scientists, the statement that a "group of chemicals is possibly carcinogenic for humans" infers that this is a fairly likely situation. This statement is quite different from the IARC statement that a "group of chemicals cannot be classified as to its carcinogenicity for humans." We strongly feel that the IARC statement should be used.

    c. Omission of groups 4 and 5 of the IARC from your classification (i.e. having no categories for chemicals for which the data are clearly negative and those for which there are no data) is very unfortunate. These categories should be included so that any chemical can be put in an appropriate class. In these cases, we assume that no permitted levels would be established.

[illegible]. The introduction of two methods of calculation of acceptable levels and presentation of some guidelines for the use of values obtained by both procedures in setting standards is a useful move toward individualizing decisions on chemicals with regard to their biological activities. Further modification of the procedure will doubtless be needed as further data become available.

Sincerely yours,

Jim and Betty

James A. and Elizabeth C. Miller
Van Rensselaer Potter Professors
of Oncology

JAM/ECM:mjg

Chemical Industry Institute of Toxicology

[illegible]
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July 2, 1982

Dr. Roy E. Albert, Chairman
Carcinogen Assessment Group
Office of Environmental Assessment
Environmental Protection Agency
Washington D.C. 20460

Dear Roy:

Thank you for this opportunity to comment on the draft of a proposed addendum to the EPA guidelines for the health assessment of suspect carcinogens with specific reference to water quality criteria. Our concerns with the draft center on three points.

First, although the IARC criteria for assessing the quality of evidence for carcinogenicity in experimental animals are to be used, the caveats attached by the IARC Working Group to the use of such data in quantitative assessments of human risk have not been included. Specifically, the IARC Monographs Preamble states, "Data that provide sufficient evidence of carcinogenicity in test animals may therefore be used in an approximate quantitative evaluation of the human risk at some given exposure level, provided that the nature of the chemical concerned and the physiological, pharmacological and toxicological differences between the test animals and humans are taken into account." We share the Working Group's concern that tumor incidence in a bioassay alone is an insufficient basis for quantitative assessment of human risk.

Second, in cases where the compound is mutagenic, we are concerned that the theoretical arguments which support the multi-stage model of carcinogenesis are being misinterpreted to support use of this model with administered dose as the independent variable. It is crucial to understand that all theories of carcinogenesis are framed in terms of the amount of active agent present in specific target tissue, i.e., delivered dose, rather than administered dose. The biologic plausibility of the linearized multi-stage model, or, indeed, any model of carcinogenesis, rests fundamentally on this delivered dose concept. Put simply, only what is present at a site can cause cancer there. Therefore, the mutagenicity of a chemical compound, by itself, is not sufficient to justify use of the linearized multi-stage model with administered dose as its independent variable. Indeed, it may be that in no case is it appropriate to use administered dose as the independent variable, since this would require strict linear proportionality between delivered and administered doses over the entire dose spectrum.

Careful consideration must be given to the structure and function of the whole animal systems which regulate how much of the administered dose actually reaches a specific target site. The quantitative relationship between delivered and administered dose is likely to be highly specific to the compound under investigation, its route of administration, and to the exposed species as well. Consequently, it seems unlikely that any single "formula" approach to quantitative risk assessment, even for mutagenic agents, will be universally applicable.

Third, we have a related concern with the proposal for a conventional toxicological approach for "non-mutagenic carcinogens". Although conventional practice is to convert doses across species on a body weight basis, there is no scientific basis for assuming this scaling rule to be universally applicable. Rather, professional scientific judgment on a case by case basis is required, and uncertainty regarding the conversion should somehow be incorporated into the assessment.

We appreciate the opportunity to provide these comments for your consideration. If you foresee a need for elaboration on any of these points, please feel free to contact us by letter or telephone.

Sincerely,

Bob
Robert A. Neal
President

RAN/ls

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[illegible]

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20 August 1982

Dr. Roy E. Albert
Chairman
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Washington, D.C. 20460

Dear Roy:

As you know, your proposals on risk assessment came to me while I was away from my office. I have been over them now and will respond.

The use of the IARC formula for stratifying the evidence is excellent. It has pretty well stood the test of repeated application and has the additional advantage of being reasonably familiar to most of the users of IARC documents.

I am not at all happy about the two-fold classification of genotoxic agents and non-genotoxic agents. Although we have come a long way in the last few years in understanding some of the details relating mutagenicity to carcinogenicity, I don't think we have the sharpness of methods required that all of these agents now deemed "negative" for genotoxicity will in the long run turn out to be negative; moreover, a grouping under the label of non-genotoxic is by its very nature a motley group of diversely acting agents; I see little advantage and some disadvantage in lumping all of these together in this way.

As you know, I have strongly supported the use of quantitative risk assessment as an important input into the evaluation and eventually the regulation of carcinogenic chemicals. I doubt that it can ever become the sole basis for our regulatory decisions considering all of the technical uncertainties in current procedures for quantitative risk assessment. (I refer here primarily to laboratory data rather than epidemiological data.)

I think that one should continue to use quantitative risk assessment along the lines that CAG has in the past. Obviously, where one has special information on any agent, genotoxic or "non-genotoxic," one

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should use such knowledge in estimating risk. With that provise, I am not at all troubled by using current mathematical procedures for the "non-genotoxic" chemicals. Always with the usual caveats plus the additional qualifications that may need to be added when applied to compounds negative for genotoxicity. I believe that such procedures are justified as a crude basis for estimating a probable outer limit of risk. Such estimates would, in any event, be used as only one input along with other information in estimating risk and reaching regulatory judgments.

This leads me to another reservation in the use of the safety factor approach which your document proposes. The use of the safety factor as proposed undertakes to establish an acceptable level of exposure. That is equivalent to overtly setting a number which could become a control or a regulatory statement. As you know, I have always felt that there should be two stages in regulation: the first is the technical assessment of risk, preferably as something equivalent to a crude dose response curve, and the second would be for the regulatory agency to take this information and make a regulatory decision in the light of socio-political and economic considerations. As you describe it, the safety factor approach jumps immediately from the first to second step and fails to take into account those balancing circumstances that in most cases are required.

If you were to use a straight line from the 10% incidence estimate down to zero and use that line as the dose response curve, that would overcome my objection of jumping from the first step to the second.

Finally, it may be desirable to consider a major review of quantitative risk assessment at the national level, perhaps through the National Academy of Sciences/National Research Council. I am sure that there will be differences of opinion scientifically and philosophically, but a responsible group faced with the recognition that some degree of quantification with appropriate estimates of uncertainty will be a useful ingredient in the standard setting process (wherever the data permits) will come out with constructive proposals.

Sincerely,

Norton

Norton Nelson
Professor

NN/j

Roy

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August 4, 1982

Dr. Roy Albert
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Dear Dr. Albert:

Thank you for sending me a copy of the "Additional U.S. Environmental Protection Agency Guidance for the Health Assessment of Suspect Carcinogens with Specific Reference to Water Quality Criteria." To get straight to the point, I do not agree with the fundamental premise of this paper -- that non-mutagenic carcinogens are of less concern than those that are mutagenic. I believe that the distinction is not based on sound scientific data and relies on certain questionable assumptions.

The draft states that for carcinogens that do not give evidence of the ability to induce gene mutations, the use of the linear non-threshold dose-response model is not plausible. It therefore proposes that for non-mutagenic carcinogens, the less conservative (NOEL) approach be used as a basis for setting standards.

My major concerns are as follows:

(1) There are inadequate scientific data to support this distinction between mechanisms. Somatic mutation is only one of the ways in which a chemical substance may cause cancer. Gene and chromosomal rearrangements, changes in chromosomal number or structure, disruption of cellular differentiation, activation of proto-oncogenes, DNA damage, and disruption of DNA repair systems are some of the other possibilities. Thus, both genotoxic (broadly defined as disturbance at the level of the DNA, the gene, or the chromosome) and/or non-genotoxic mechanisms may be involved. All must be considered of equal importance. Thus, to equate "mutagenicity" with "genotoxicity" with "the most serious carcinogenic mechanism" does not appear to be sound.

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(2) There is a lack of data establishing a threshold for non-mutagenic carcinogens or that the dose-response curve is different in the lower range from that for substances that cause gene mutations. On the contrary, the literature on ionizing radiation shows a linear/quadratic dose-response curve for chromosomal aberrations in which the curve intercepts the point of origin.[1]

In most cases, however, we do not know enough about the dose-response curve of non-mutagenic or even non-genotoxic carcinogens (as broadly defined above) to draw conclusions about its shape at low dose levels or about the existence of thresholds. With tumor promotors in general, the studies needed to understand the dose-response curve -- studies administering the test substance over a wide range of dose levels -- have not been carried out. Thus, as the draft acknowledges, "[w]e currently do not know enough about the mechanism of action of non-mutagenic carcinogens to plausibly extrapolate dose-response relationships for such agents to low levels of exposure." (p.8). To give an example, DES is probably one of the most studied of the hormones; yet the animal and human data do not provide a distinct dose-response curve.[2] Even a recent large-scale bioassay of DES[3] leaves questions about details of the lower range of the dose-response curve. Thus, I would agree that:

> [i]n general, it would seem that a chronic elevation above the level of endogenous estrogen production would qualify as a potential carcinogenic dose.[2]

At a recent workshop on EPA's ANPR on Volatile Organics in Philadelphia on July 21-22, Dr. William Lijinsky pointed out that we cannot yet design the experiments to determine whether there are thresholds at low doses (and thus cannot begin to set a threshold for carcinogenicity in humans). In particular, he emphasized that it is not now possible to establish a threshold for humans that takes into account differing organ sensitivities as well as other relevant factors. For example, in bioassays with diethylnitrosamine (DEN), Dr. Lijinsky has observed liver tumors at high doses while esophageal -- and not liver -- tumors appear at low doses. Had the experiments at lower doses not been carried out, one might have concluded the existence of a threshold for DEN based upon the induction of hyperplasia at high doses. Therefore, as IARC states:

> [T]he limited knowledge about the mechanisms of action of chemical carcinogens and about the multistep carcinogenic process preclude any rigid categorization of carcinogenic agents.[4]

(3) Beyond these uncertainties at the experimental level, the distinction between carcinogens on the basis of mechanism of action (where it were possible) appears to be of questionable practical value. This is because of the practical impossibility of identifying population thresholds given the lack of data at low dose levels, variations in individual human responses[5] (due to genetic factors, health status, and life history), and possible additive or synergistic effects of other agents to which individuals are exposed.

This is particularly true for halogenated organic water pollutants which show synergism with other environmental chemicals (e.g., MFO inducers) and which may themselves enhance the effect of other halogenated organics. Further, many of them act on the same organ, the liver, and are known to bioaccumulate. The fact that individuals have continuous lifetime exposure to the combination of organic contaminants in drinking water (some of which may be inhibitors, some promotors, some complete carcinogens) -- albeit generally at low doses -- argues against assumption of a safe level or threshold effect in the human population.

Additional complicating factors in carcinogenesis are time and duration of exposure. These factors may be as significant as dose itself; yet my impression is that we generally know as little about their specific role as about the lower range of the dose-response curve. Exceptions are DES and estrogen. The stage in embryonic development at which DES exposure occurs is established as a critical factor in vaginal cancer, as is duration of exposure to estrogen in the production of endometrial cancer.[6] It appears that with tumor promotors in general, frequency and duration of exposure -- as well as dose level -- are critical factors in the production of tumors.

(4) The policy resulting from the "double standard" approach in the draft will be considerably less protective than the present policy. Furthermore, according to Table 1 of the draft, absence of data or inconclusive data would cause EPA to use the less conservative NOEL approach or a midrange value rather than the linear extrapolation method. In Table 2, 10 of 14 compounds are characterized as having inconclusive or inadequate data on mutagenicity. As the table shows, the NOEL approach yields estimated standards that exceed those based on the CAG model by a factor of 10-369. Given the real problem of "false negatives" in mutagenicity assays arising from inappropriate metabolic activation systems etc., it seems unwise to use inconclusive or inadequate data for mutagenicity as a basis for a less protective standard.

(5) Somewhat hidden in the document is a change from the generally accepted procedure of giving guidance based on the range of risk values 10^{-5} - 10^{-7}. This is a significant policy change that must be extremely well documented before adoption; yet it is not supported by any discussion in the text.

* * *

In light of all of the above, I believe that EPA should solely use the conventional CAG model rather than alternate with the old-fashioned NOEL approach that has been modified to use MLE and body weight rather than surface area conversation. As you know, the conventional linear multi-stage model is by no means the most conservative model that could be used to extrapolate human risk.[7] At the Philadelphia conference mentioned earlier attended by Dr. Marvin Schneiderman, Dr. Robert Neal, Dr. William Lijinsky and others, the health effects working group concluded: "The multi-stage model is appropriate for both genotoxic and non-genotoxic compounds, thus distinctions between genotoxicity and non-genotoxicity are not relevant." Furthermore, in using the linear model, I believe that EPA should continue to give guidance based on the range of risk values 10^{-5} - 10^{-7}, rather than only 10^{-5}.

I hope these comments have been helpful and I would be happy to discuss these questions further. I hope your European trip was enjoyable. How are the crowds in Italy?

Sincerely,

Frederica P. Perera

Frederica P. Perera
Senior Staff Scientist

FPP:cp

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June 24, 1982

Dr. Roy E. Albert
Chairman, Carcinogen Assessment Group
Office of and Environmental Assessment
U. S. Protection Agency
Washington, 20460

Dear Roy,

With reference to your letter of June 18, I have had an opportunity to look over the proposed addendum to the EPA guidelines that you sent and would offer the following suggestions.

In general I am very happy to see that a federal regulatory agency such as the EPA is beginning to consider in a serious manner more of the available facts regarding the process of carcinogenesis and their relationship to possible human risk. As our knowledge of the natural history of neoplastic development increases I would hope that the Agency would continue to look at all the facts in relation to cancer risk assessments. Further I would hope that this approach to guidelines in this area would be extended to other environmental areas as well.

More specifically there are several points in the document for which I would suggest the following alternatives:

1. In the consideration of the stratification of the weight-of-evidence judgements, it would seem scientifically proper to consider the efficiency of repair of DNA damage caused by a specific agent as well as its pharmacokinetics. While I realize that these two variables take considerably more effort to determine, where such data is available I would feel that it would be important to include it in the weight-of-evidence judgments.

2. The term "tumour" which is used on the top of page 4 is taken directly from the IARC assessments. This term is not commonly used in the United States when speaking of neoplasms and it can be confusing since a tumor is not necessarily a neoplasm. In the final document I would hope that the term, neoplasm, is used throughout rather than a combined set of terms which can be confusing.

3. I do not understand the footnote at the bottom of page 3 which indicates that two IARC categories, negative and no data, were deleted. There certainly are instances where no data is available and this would be important to risk assessment. Negative studies are becoming more obvious with more and more compounds being investigated. I do not feel that the regulatory agencies should be afraid to call something negative if that's

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what all the available data shows. I am not one to argue that no matter how much negative data is available, one can never by absolutely certain that under some esoteric condition a material may be found to be positive in a specific test. If such a philosophy were to be accepted then in fact all materials in our environment are suspect carcinogens simply because we can never complete all possible tests on any single agent.

4. I do not think that the original IARC wording for group 3 (page [illegible]) should be changed as you have it. The term "possibly carcinogenic . . . humans" signifies to many if not the majority of lay individuals that such chemicals are likely to be carcinogenic. The IARC designation clarifies this point and is to be preferred.

5. Rather than arbitrarily taking an uncertainty factor of 1000 wouldn't it be reasonable to estimate such factors where some of the data is known such as with aflatoxin, DES, chlornaphazine and determine what the order of uncertainty would have been with these compounds. Furthermore one could guess at some of these factors with a number of other known human carcinogens based on more recent animal data, e.g. vinyl chloride, antineoplastic agents, etc. In doing this such estimations would have more basis than simple guesses.

6. The use of the term "chromosome mutations" is not clear to me. If translocations, transpositions, chromosomal deletions and polysomies are meant by this term than I feel that this should be indicared somewhere in the text. The sentence on page 11 (ll 17-22) is even more confusing when related to "primary DNA damage". Furthermore as you know there is more and more evidence accruing that chromosomal abnormalities may be the most critical factor involving the genome in relation to the initiation of neoplasia.

I hope that my questions and suggestions don't sound too naive. Obviously you look at such guidelines in a different light than I. However I hope these thoughts may be of help to you in your deliberations. If I can be of any other assistance, please don't hesitate to let me know.

With kindest regards,

Henry

Henry C. Pitot, M.D., Ph.D.
Director

HCP:mjg

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

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June 25, 1982

Dr. Roy Albert
Chairman
Carcinogen Assessment Group
Office of Research and Development
U. S. Environmental Protection Agency
Washington, D.C. 20460

Dear Roy,

I received your letter of June 21 requesting my comment on the draft EPA proposal on "Guidance for the health assessment of suspect carcinogens with specific reference to water quality criteria." I am leaving today (6/26/82) for Europe and in the last few days I had to finish our Annual Report and other pending documents. Therefore I have had no time to prepare a more detailed, annotated review of this proposal. The present comments strictly express my personal scientific views and are clearly not intended to represent any official position of the National Cancer Institute. I am only addressing the science aspects, and not the policy aspects which are outside my present scope.

The draft contains many assumptions and deductions that are, in my view, lacking scientific justification.

The cornerstone of the draft is the distinction of carcinogenic substances in the two categories of genotoxic and non-genotoxic. This distinction has been recently proposed in the scientific literature, e.g. by John Weisburger and Gary Williams, but the evidence for such a diverse pathway in the mechanism of action of carcinogens is far from clear. While we do know of many carcinogens as being genotoxic as shown by their mutagenic activity, the quantitative correlation of mutagenicity with carcinogenicity is poor, as is documented in the working paper by Ian Purchase of I.C.I. for the forthcoming SGOMSEC meeting on risk assessment to be held in Rome on July 12-16, 1982. The neoplastic transformation of cells in culture can be a much more frequent event than the induction of a gene mutation in the same cells. Carcinogenic, co-carcinogenic, promoting and cell growth-regulatory factors may concur in the induction of neoplastic expression. At the molecular level, the sequence of biological changes required to start a neoplastic process is not yet elucidated, although the rapid progress of molecular biology methods is now allowing us to probe it more closely. There are indeed some carcinogens for which no evidence so far has been gathered of a direct mutational mechanism.

Considerable interest has been generated by the possibility that a major mechanism of carcinogenesis may consist of a derepression of certain genes that function as suppressors of oncogenic information already present in the cell. Such functional gene regulation may not require a mutational event in order to be started, but the kinetics of such "toxic" mechanisms are still unknown. Many different concurrent factors may take part in the process of carcinogenesis and I know of no evidence that would exclude a linear type of dose-response relationship. The draft proposal embarks on such a hypothetical distinction as "genotoxic versus non-genotoxic" but rapidly runs out of steam when it has to come up with the criteria for such a classification (section c.2) and concludes with a nondefinition for non-genotoxicity (p.12), stating that the criteria may vary from chemical to chemical and will be left to professional judgment (not otherwise defined). Lack of detection of mutations in a few test systems is not sufficient to exclude a mechanism that may damage the genetic control of neoplastic expression in the human organism.

For practical purposes, nationally and internationally accepted scientific criteria for carcinogen identification have required precise definition of bioassay methods, and tests of their reproducibility and predictiveness. These are completely lacking in the proposed draft, and yet this determination represents its pivotal point.

On the point of "stratification of the weight of evidence", the adoption of a classification comparable to that used by the IARC monographs is intended here for a differential regulatory use, while the IARC classification is addressed mainly to distinguishing the amount of information available for a substance. A substance may be a highly effective carcinogen for humans in certain conditions but bioassay evidence may appear "limited" or "inadequate" if the bioassay system is biologically inappropriate or the test design inadequate. We know of several examples of substances that were known to be carcinogenic in humans and for which it took considerable time to find the adequate test conditions to reveal the effect in animals: examples include several aromatic amines, inhalation of polycyclic aromatic hydrocarbons, benzene, and the still pending example of arsenic.

If the IARC classification is considered, my major concern is to improve on the definition of "sufficient" and "limited" evidence: I have found that the current IARC definitions are not mutually separated by clear criteria, are confusing in their use of malignancy criteria, and inaccurate in the inclusion of "lung and liver tumors in mice" as examples of "neoplasms....that often occur spontaneously or are difficult to classify as malignant by histological criteria alone". There are strains of mice in which lung and liver tumors do not seem to occur often spontaneously and there are lung and liver tumors that are easily classified as malignant, even by histological criteria alone. If the IARC reference is used, one could at least specify: "lung adenomas and liver cell adenomas (hepatomas) in certain strains of mice". I have published some comments on this issue a couple of years ago and I refer you to those comments in the enclosed reprint (Saffiotti, U., J. Toxicol. Environ. Health, 6: 1029-1057, 1980, page 1035, footnote 1).

On p. 7, line 4, of the draft proposal, the definition of group 3 chemicals as "possibly carcinogenic for humans" is confusing; it is adequate if it refers to compounds for which there is limited evidence of carcinogenicity, but for compounds with inconclusive evidence only, the IARC wording is more accurate and these could constitute a separate group.

On page 7, the draft begins discussing the use of mutagenicity criteria. I have given you my general comments above. Here are some more specific comments.

- p. 7, line 9, etc.: the rationale for use of the linear non-threshold model is not heavily dependent on the mutagenicity of carcinogens, and the biological justification for its use does not lie in the correlation between carcinogenesis and mutagenesis. This is one of the most distorted arguments in the draft.

- p. 8, lines [illegible] ar model is just as "plausible" in the absence of demonstrated gene mutations by a given agent, unless there is very heavy direct evidence of another dose-response pattern at appropriately low doses. The selection of a particular model cannot now be based on the "required" acceptance of a mechanism of action, since the intimate molecular control mechanisms in carcinogenesis are still unknown. We know so little about the hypothetical non-genotoxic mechanisms for cancer causation that it seems highly premature to presume to treat them differently from those of other better known carcinogens, when the overall biology of the induced cancers is quite comparable.

- p. 8, line 14 etc, and p. 9, c.1: the draft proposes the use of a "conventional toxicologic approach for non-mutagenic carcinogens". What is "conventional" about an approach that does not fit the evidence? There is no such thing as "conventional" toxicology, if toxicology is to reflect the variety of chemical-biological interactions. The approach described as "conventional" was the approach developed during the Stone Age of toxicology, which I find best described by this statement I once heard: "Find a no-effect level in animals, divide by 100, and pray". This type of "conventional" toxicology has been rejected a long time ago as irrelevant to the evaluation of self-replicating toxic effects, no matter how induced. In cancer induction what is fundamentally different from toxic degenerative pathology is that the induced neoplastic state makes the target cell proliferate rather than die off; and therefore, the health effect keeps progressing when the toxic agent is no longer there. It is the neoplastic condition that is progressive, not the existence of a mutagenic mechanism; on the specific role of induced mutations in carcinogenesis, even for mutagenic carcinogens, there is still ample speculation.

I do not have time to comment on the tables and applied examples given in the draft, except for confirming my personal feeling that they are based on inappropriate premises, as discussed above. I believe that an excellent criticism of the attempt to regulate carcinogens on the basis of numerical and arbitrary risk estimates was given in the 1980 Report of the National Research Council's Committee on Prototype Explicit Analyses for Pesticides, entitled "Regulating Pesticides." As you know I am extremely skeptical of our present ability to arrive at any reliable numerical risk estimate of carcinogenic risk in the human population on the basis of bioassay data, while I think that well conducted bioassay results, critically analyzed and integrated with relevant research results, can give meaningful qualitative risk evaluations for use in public health.

These are my personal reactions to the draft you sent me. I will be glad to discuss them further when we meet in Rome.

Looking forward to seeing you soon, I remain.

Very cordially yours,

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Enclosure

TELEX FROM LORENZO TOMATIS, IARC, Lyon, France, July 6, 1982

TO: Roy E. Albert, M.D.
Chairman
Carcinogen Assessment Group Office of Environmental Assessment
U.S. Environmental Protection Agency
Washington, DC 20460

Having seen your letter 22 June, 1982 only today, and given the urgency you mention, I list below a few immediate comments:

Alt[illegible] chemicals may eventually receive different treatment according [illegible] mechanism by which they exert their carcinogenicity, there is at present considerable uncertainty on how categorization of chemicals could be established. At present, it appears that the basis for justifying any form of extrapolation down to levels of risk in the order of 10^{-5} is very weak.

Page 4: The definition of limited evidence has been slightly changed as point c) in this paragraph now reads: "The neoplasms produced often occur spontaneously and have in the past been difficult to classify", etc.

Page 5: The definition of limited evidence drafted by IARC says limited evidence of carcinogenicity which indicates that a causal interpretation is credible, but that alternative explanations, such as chance, bias or confounding, could not be adequately excluded. Inadequate evidence which indicates that one of three conditions prevailed:

(A) There were few pertinent data;

(B) The available studies, while showing evidence of an association, did not exclude chance, bias or confounding;

(C) Studies were available which do not show evidence of carcinogenicity.

Page 7: The definition of group 3 given by IARC seems much more appropriate. The new version would seem rather loose. It is important to keep in mind that for the chemicals in group 1, the categorization is final, but for chemicals in groups 2 and 3, the categorization may change when, and if, new data become available. To classify all group 3 chemicals as limited forcibly excludes the category inadequate.

Page 14: The grouping in Table 1 of the IARC groups 1, 2A and 2B under sufficient, and IARC group 3 as limited does not appear correct.

A copy of the introduction methods and results section of the new IARC supplement 4 updating IARC supplement 1 have been sent by airmail.

Lorenzo Tomatis, M.D.
Director, IARC
380023 Lyon, France

JUN 2[illegible] 1982

NEW YORK UNIVERSITY MEDICAL CENTER
Inter-office Communication

June 28, 1982

TO: Dr. Roy E. Albert

FROM: Dr. Arthur C. Upton

SUBJECT: Proposal addendum to EPA Guidance for the Health Assessment of Suspect Carcinogens

In response to your letter of June 18, I have reviewed the proposed addendum to the EPA Guidance for the Health Assessment of Suspect Carcinogens, with Specific Reference to Water Quality Criteria. In general, I find the proposed approach to be well conceived and appealing. I would suggest, however, that it be referred for comment to others who are more expert than I in assessing the risks of chemical carcinogens; eg, I. B. Weinstein, S. Weinhouse, U. Saffiotti, D. G. Hoel, H. Pitot, A. Whittemore.

A few minor editorial comments and corrections include the following:

page 5, line 15: "is" should replace "are";
page 7, line 4 et seq: how to deal with chemicals for which there is strong evidence that they are not carcinogenic?
page 8, line 10: the plausibility of the linear, non-threshold model is buttressed by data on *in vitro* cell transformation and by data on the carcinogenic effects of high-LET radiation in humans and laboratory animals;
page 10, line 7: "are" should replace "is";
page 10, line [illegible]: "uncertainty" is misspelled
page 12, lines 1-2: this statement seems redundant with the preceding
page 13, line 18: "criteria" is plural;
page 19, line 2: replace "criteria" with "criterion"

Many thanks for the privelege of reviewing the draft proposal. If there is anything further I can do to help you, please don't hesitate to let me know.

ACU:mtg

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June 29, 1982

Dr. Roy Alberts
Chairman
Carcinogen Assessment Group
Office of Environmental Assessment
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Roy:

I read your Guidelines for Health Assessment of Suspect Carcinogens with considerable interest and I am outlining my comments as follows.

Stratification is OK, but has to be done with great caution and with every effort to avoid undue substratification. I would go along with the IARC categorization and would suggest these to be as follows:

1. Sufficient evidence;
2. Limited evidence;
3. Inadequate evidence;

and possibly a fourth, negative evidence.

I would use exactly the same categories for human studies and for mutagenicity, thereby stratifying in the same manner each method of assessing carcinogenic activity. I don't think that other categories or substratification is warranted. One gets down to hair splitting, subjective judgments, and all of the personal viewpoints that lead to disputes and disagreements. I believe that regulatory agencies can feel comfortable with these four categories but they should be accompanied with proper textual elaboration, since each putative carcinogen has its own peculiar qualities and characteristics, and risks to specific populations.

As you know, I have decided views against mathematical quantitation of risk. At one time I favored such quantitation, but as I learned more about the pitfalls of interpretation and extrapolation from high doses to low doses and from animals to man I realized there is a limit to what can be done with data particularly when they are often fragmentary or contradictory or incomplete.

No matter how sophisticated the mathematical model, the results can have no greater significance than the original data from which they are derived.

This situation may not apply to ionizing radiation where there is a massive body of human as well as experimental data, much of which covers responses to graded doses even down to low levels. Still, the sharp and shrill disagreements that plague radiation carcinogenesis assessments should caution us to beware of quantitation in the field of less defined chemical carcinogenesis. Where there is so much room for individual judgment it is difficult or impossible to have experts agree on the significance or statistical errors of mathematical modeling, and in my judgment (which I realize may be somewhat biased), I believe that with all these defects of interpretation and the uncertainties of model building, the estimations of risks in quantitative terms are sterile academic exercises.

As I read your report, I think you give too much significance to mutagenicity data. It is true that there are some parallelisms. On the other hand there is a large body of evidence against mutation as the only mechanism of carcinogenesis and in fact there is a good deal of evidence against mutation. I am enclosing a recent article of my own which appeared in the JNCI in which I briefly discuss epigenetic versus genetic mechanisms and give a few references to thinking on this subject by other investigators. Another paper in this category is by Straus, JNCI 67, 233, 1981. Unless we know more about the mechanisms of cancer induction, I do not think that we can implicate theoretical conceptions such as mutation, initiation, promotion, etc. as bases for regulatory decisions affecting large populations. Too much is being made of mechanism, about which we still know little.

I will close by saying that I am strongly against the use of conventional toxicological approaches to estimate risks of chronic toxicants such as carcinogens. You use a factor of 1000 to assess carcinogenic risk. Your data of Table 2 indicate however, that there is an uncertainty factor in comparison with your linear extrapolation model of at least 100 or more. The conventional toxicologic approach is OK for acute, reversible toxicity but is a mockery for chronic, irreversible toxicity.

In summary, my own philosophical outlook turns me away from putting numbers on risk assessments. No matter how many caveats you place on your data, you may be sure that they will be used by regulatory agencies and their attorneys as graven in stone. I feel they are not only misleading but are subject to such a battery of objections and misinterpretations as to make them virtually useless for defensible regulations. It is far better I believe to say that the data are not of such nature to be subject to quantitation. In my judgment, I believe you are trying too hard to obtain figures which by their very nature are going to be provocative and extremely difficult to defend.

I hope you find these comments constructive and I hope that I have not been too critical. Believe me, I do commend your tireless efforts toward risk quantitation, even if I have to disagree with their value in establishing regulatory guidelines. I hope we have a chance to discuss these matters further.

With all good wishes and regards.

Sincerely,

Sidney

Sidney Weinhouse, Ph.D.

SW:ls

JUL 2 1982

College of Physicians & Surgeons of Columbia University | *New York, N.Y. 10032*

CANCER CENTER INSTITUTE OF CANCER RESEARCH
701 West 168th Street

June 28, 1982

Dr. Roy [illegible]
Dept. of Environmental Medicine
New York University Medical Center
New York, New York 10016

Dear Roy,

I have read the draft of the EPA guidelines related to water quality criteria and want to make the following comments.

First, I fully agree with the use of IARC criteria on pages 3-7 for "Stratification of the Weight of Evidence for Carcinogenicity". These criteria were revised somewhat at a February 1982 IARC Workshop and you will probably want to use the newer version, which you can obtain from Lorenzo Tomatis at the IARC.

Second, I seriously disagree with the premise that "negative evidence for mutagenicity would tend to call for the use of conventional toxicological method...". I am afraid that this is part of a misconception that is being perpetrated that carcinogenic agents that do not have demonstrable mutagenic activity are somehow safer than those that can be shown to be mutagenic. I believe that this logic is faulty for several reasons.

First, it assumes that conventional mutagenesis always plays a role in carcinogenesis, and yet currently the tide of research evidence is flowing the other way. Oncogene activation, disorders of differentiation, specific types of gene rearrangements, etc. are attractive alternatives that are more complex than conventional somatic mutation, and most of the current mutagenesis assays do not score for such effects. Second, negative mutagenesis assays may be "false negatives" due to failure of metabolic activation, tissue and species specificity, etc. Certain known carcinogens would probably be missed in conventional mutagenesis assay (certain nitrosamines, azo dyes, dinitrotoluene, cycasin) and yet when properly studied they are genotoxic. A new compound might be negative because it is not properly studied in terms of tissue or species metabolic activation. Third, mutagenesis assays may give false positives, or the mutagenic potency in bacterial systems may be much greater than the genotoxic effects in higher organisms. Witness the current controversey over mutagenicity and health hazhards of diesel engine emissions and nitroaromatics. Fourth, certain compounds are extremely potent carcinogens

(DES, TCDD) and yet are negative in most of the mutagenesis assays. To my knowledge there is no evidence that they show a threshold effect or a dose response curve which is qualitatively different from that of genotoxic carcinogens. Fifth, there is insufficient data to indicate that tumor promoters show threshold effects or dose-response curves that are different from complete carcinogens. Sixth, even conventional mutagens do not always show linear non-threshold dose response curves perhaps due to excision DNA repair, inducible repair, toxicity at high doses, pharmacokinetics, etc.

In general, the criteria in this draft fail to acknowledge the multistage nature of carcinogenesis and the problem of synergistic interactions between initiators, promoters and various cofactors. This considerably confounds efforts to do simple dose extrapolations. Table 2 is instructive in that it shows that mutagenicity as a practical criterion in risk extrapolation is often very irrelevant. For most of the compounds listed the evidence for mutagenicity is "inadequate" or "inconclusive"; therefore this parameter is irrelevant. Many of the compounds that pollute water sources are problematic since they are halogenated organics and we know very little about their mechanism of action. They also bioaccumulate and may show synergy with other compounds. Why is it more appropriate to use the "conventional toxicological method" for these compounds than the "linear non-threshold model"?

I trust that these comments are useful and I would be happy to discuss some of these issues with you. I can appreciate that this is a difficult area and that certain decisions must be made on the basis of existing knowledge. However, as indicated above, I disagree about some of the basic assumptions that are made in the draft of these guidelines.

Sincerely yours,

Bernie

I. Bernard Weinstein, M.D.
Professor of Medicine and
Environmental Sciences

IBW:ee

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service

National Institutes of Health
Bethesda, Maryland 20205
Building 31, Room 11A04

June 22, 1982

Dr. Roy E. Albert
Chairman
Carcinogen Assessment Group
United States Environmental
  Protection Agency
Waterside Mall
401 M Street, S.W.
Washington, D.C. 20460

Dear Dr. Albert:

I have read the document you provided. I consider it reasonable that EPA use the IARC guidelines on the weight-of-evidence for carcinogenicity. However, explicitly answering Part C, page 7, I am especially dubious about using mutagenicity data for quantitative cancer risk estimates as proposed on page 7. Most persons in the field would agree that the correlation between mutagenicity and carcinogenicity may vary between 60 and 90 percent depending on the structures of the compounds involved. Thus the statement that there is a correlation between carcinogenesis and mutagenesis should be motified. Furthermore, not all carcinogens interact with DNA which undermines the statement "the commonality of DNA damage for carcinogens and mutagens."

I am dubious about "the quantal nature of the interactions of chemicals with DNA" for risk analysis for certain reasons. (1) Although there may be a linear dose response between the level of DNA adducts for some specific carcinogens, for other carcinogens there are numerous DNA adducts. The significance of the various adducts is not known; thus dose-response relationships have not been determined. On Page 8 it is stated that the plausibility of the model depends heavily on the acceptance of the somatic mutation theory of cancer. I am distressed that this leaves no room for other theories of carcinogenesis and implies that one must accept this theory.

Page 10, 2nd paragraph: "the first uncertainty factor is considered to be a safety factor of 10 based on the greater certainty that the agent is carcinogenic for humans." Is it a fact that any one particular compound is more likely to be carcinogenic in humans?

On page 15, the last paragraph states: "The guidance toward the midrange with many of the compounds reflects the fact that few suspect carcinogens have been tested adequately for mutagenicity." I question this statement in view of many papers on testing large series of compounds for mutagenic activity as well as the NTP activity in this area.

Over all, therefore, I am dubious about the proposed scheme to use mutagenicity data for quantitative risk assessment.

Sincerely,

Elizabeth K. Weisburger, Ph.D.
Assistant Director for Chemical Carcinogenesis
Division of Cancer Cause and Prevention
National Cancer Institute

DOW CHEMICA[illegible]

[illegible] 28, 1982

[illegible]DLAND, MICHIGAN 48640

[illegible] . Albert, M.D.
[illegible]cinogen Assessment Group
[illegible]ice of Environmental Assessment
[illegible]PA
[illegible]hington, D.C. 20460

[illegible]r. Albert:

[illegible]hank you for the opportunity to comment on [illegible] raft proposed
dum to US-EPA guidelines for assessing carcin[illegible] in water.
encouraging to note that the guidelines are [illegible]mpting to
[illegible]der a broad spectrum of data including mechanistic information
[illegible]sk assessment analysis. We would continue to encourage the
[illegible] all appropriate data on any given che[illegible] [illegible]njunction
[illegible]ound scientific principles. Meeting th[illegible] [illegible]e dictates
[illegible]n even more scientifically flexible guideli[illegible] [illegible]n currently
[illegible]ed should be considered. Several individual[illegible] Park, R. Reitz,
[illegible] Watanabe) with background in biostatisti[illegible] [illegible]ing, toxicology/
[illegible]cology and biochemistry have commented on [illegible] [illegible]rent proposal.
[illegible]nce again thank you for the opportunity [illegible] the document
hope that the suggestions will be use[illegible]

[illegible]rely,

l. Gehring

Gehring
[illegible]or
[illegible] and Environmental Sciences
[illegible]ow Center
[illegible], MI 48640

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jh

- To add an additional safety factor of 10 for chemicals for which there are human data seems to cast doubt on the interpretation (for human disease) on those cases where there are not human data. I don't think this reasoning makes sense.

Page 13. The notion of bracketing the risk for determining water quality criteria,

10^{-15} > True risk > "conventional toxicological method"

appears to have merit - especially when combined with the product of some expert judgment in each case.

General comments

This paper proposes a rationale and a methodology for a semi-quantitative estimation of the liklihood that a given chemical species will contribute to human cancer burden. It conforms to the earlier EPA guidelines for estimating cancer risks which permits "quantitative estimation of incremental cancer risks..." The proposal implies that graded or differential responses to chemical exposures might be expected as a function of whether or not the chemical acts directly upon the genetic material of the target cells.

The proposal rests on the present understanding of biological mechanisms of cancer causation [illegible] empirical evidence and a theoretical framework. Specifically, it is proposed that the implications for human biology are substantially different (measured as probability of initiating neoplastic disease) for chemicals which induce gene mutations and those which do not and that it is possible to distinguish between these two classes.

The general idea expressed in this paper has merit. There is evidence for this biological distinction. Further, the specific proposal in this paper suggests a decision pattern to accomodate this distinction that includes a number of desirable features considering the present state of understanding:

1. It permits a substantial degree of flexibility.
2. It accomodates or even invites professional or expert judgment.
3. It remains essentially conservative - perhaps even too conservative (see below).
4. It proposes to display a range of estimates of risk in each case (extrapolation from a linear hypothesis model to that derived from "conventional toxicological method").
5. It would express the risk estimates in probabilistic terms.

Specific comments

Page 7. The proposed change in wording from the original IARC text amounts to a substantial change in meaning. I would suggest retaining the original text.

Page 9. I would argue that a scientifically better mode of conversion of results from animal to man would be on a surface area basis rather than a body weight basis.

Page 10. The author proposes that, in general, humans are more sensitive to chemical carcinogens than are other species. I am not aware that this is "generally" true.

DOW CHEMICAL U.S.A.

MIDLAND, MICHIGAN 48640

June 28, 1982

Roy E. Albert, M.D.
Carcinogen Assessment Group
Office of Environmental Assessment
US-EPA
Washington, D.C. 20460

Dear Dr. Albert:

Thank you for the opportunity to comment on the draft proposed addendum to US-EPA guidelines for assessing carcinogens in water. It is encouraging to note that the guidelines are attempting to consider a broad spectrum of data including mechanistic information for risk assessment analysis. We would continue to encourage the use of all appropriate data on any given chemical in conjunction with sound scientific principles. Meeting that objective dictates that an even more scientifically flexible guideline than currently proposed should be considered. Several individuals (C. Park, R. Reitz, and P. Watanabe) with background in biostatistics/modeling, toxicology/pharmacology and biochemistry have commented on the current proposal.

Once again thank you for the opportunity to review the document and we hope that the suggestions will be useful.

Sincerely,

P J Gehring

P. J. Gehring
Director
Health and Environmental Sciences
2020 Dow Center
Midland, MI 48640

Enclosure

jb

AN OPERATING UNIT OF THE DOW CHEMICAL COMPANY

GENERAL COMMENTS

While the document attempts to incorporate more flexibility in the standard setting process it is still too rigid and falls short of the goal of using all of the available scientific data to make the best scientific decision. The fact that none of the chemicals cited in Table 2 are characterized as nonmutagenic when considerable scientific evidence exist on some to support non-mutagenicity is a reflection of the use of inappropriate criteria. Furthermore, the use of inflexible presdesignated safety fact does not allow for scientific judgement based on the abunda or lack of available data. The selection of mathematical models should be based on scientific data of goodness of fit without disguised conservative factors such as 95% confidence limits on slope and assumed linearity at low doses. The best scientific estimate is seldom, if ever, accurately reflected by hidden conservative assumptions. The guidelines need modification such that the best science can be used and that better data is rewarded.

SPECIFIC COMMENTS

Criteria for genotoxicity (gene mutation)

The criteria for adequate or sufficient evidence for gene mutation being positive results in any two systems (one being eukaryotic) is inappropriate. More scientific flexibility is required for assessing sufficient evidence of gene mutational activity. The variable nature of results from the current *in vitro* mutagenicity tests do not support this rigid criteria. For example, chloroform was selected by the National Toxicology Program (NTP) as a "model" compound and was subjected to an extensive battery of mammalian and bacterial mutagenicity tests (Bridges et al., Progress in Mutation Research (1981), 1, 49-67; Brookes and Preston, loc. cit.) The conclusion from the NTP studies was that chloroform was not mutagenic. In spite of the extensive testing the current guidelines categorize the evidence for chloroform as "inconclusive".

Furthermore, none of the compounds listed in Table 2 are listed as "negative" although many of them have been extensively tested in short term tests with predominantly negative results. Another obvious inconsistency is characterizing benzene as unequivocally mutagenic. The mutagenicity data for benzene do not support this.

Creation of a new regulatory class of carcinogens without apparent mutagenic activity will have little consequence if it is impos[illegible]sh that any compounds actually belong in this class with state of the art experimentation. Those chemicals which are uniformly active in mutagenicity tests (certain nitrosamines, aromatic amines, PAH and alkyl epoxides) are examples of chemicals which should be classified as having sufficient evidence of mutagenicity. Those with predominantly negative results with weak or inconsistent positive tests are likely negative and should be characterized as such (exp. chloroform, DDT, benzene, carbon tetrachloride, etc.). Therefore it is suggested that a more flexible guideline and judicious scientific evaluation be given to the criteria.

Animal/Man Extrapolation

The animal to man conversion should be separated from the mechanism question. The proposed guidelines have mixed the two and made the procedure more conservative. We suggest calculating the 10^{-5} risk; the safety factor approach, and the midrange, then selecting the "best" number and then converting estimates from animal to man using whatever conversion is scientifically appropriate. The present midrange is the geometric mean of the two estimates; but one estimate is a mg/cm^2 conversion while the other is a mg/kg conversion.

The selection of the geometric mean as opposed to arithmetic mean is a policy decision favoring the conservative side rather than a scientific estimate. The arithmetic mean is preferred.

Statistical Models

The language is somewhat argumentative in places; particularly with respect to the linear model: the "plausiblity" of the linear model (p. 2); the implication that the linear model represents a reasonable biological mechanism (p. 8). The "scientific acceptance" of the linear model is certainly detabable. Furthermore, on page 7 (last line) ". . . linearity at low dose levels is a part of the model." This is very misleading. The multistage model does not have any assumption of linearity. Kenny Crump, under an EPA contract, developed a statistical method for incorporating an upper confidence on the linear term. His procedure forces a linear term regardless of the data, but this is not part of the multistage model. It was a policy decision, not a scientific one, to incorporate a linear component and modify the multistage model. It is important to separate policy from science and identify each accordingly.

Furthermore, the above comments on animal to man extrapolation after derivation of level/risk should be dictated by knowledge of the mechanism of toxic action (pharmacokinetics) of the chemical. Man will not always be more sensitive than rodents and this should be considered in the process of species extrapolation.

Selection of Safety Factors

The imposition of a rigid 1000x safety factor (to be applied to the extrapolated 10% effect level) does not allow the flexibility in scientific judgement needed when cancer is induced primarily as a result of nongentoxic mechanisms. In practice, use of this safety factor amounts to linear extrapolation to a risk of 10^{-4}. This is only 10 fold less than that proposed for use with DNA damaging agents (where a 10^{-5} risk level is used). When good evidence exists that a material is carcinogenic only at toxic levels, and non-toxic levels are well established, the selection of other safety factors should not be precluded by an arbitrary value of 1000.

Classification of Animal and Human Studies

The classification of negative studies into the "inadequate" category is misleading. A well conducted animal study characterizing effect and no-observed-effect doses is not "inadequate"; it is negative and should be characterized as such. Furthermore, well conducted epidemiology studies in conjunction with well conducted animal studies yields good evidence of the lack of any significant carcinogenic potential and should not be categorized as "possibly carcinogenic". A more acceptable compromising phrase would be "no evidence of potential carcinogenicity".

Table 2

The concentration estimate for TCDD should be rechecked. We could not duplicate this value. Other derived values are based on the absolutely worst [illegible] ose possible) regardless of other data. The most sensitive study should often be used but there should be considerable scientific judgement exercised in the selection process.

Groups of Chemicals (p. 6)

Groups of chemicals should be used only when data on all exist to support potential carcinogenic risk. The use of structural analogy is not supportable. Examples such as 2,4-toluenediamine (positive animal carcinogenicity) vs 2,6-toluenediamine (negative animal carcinogenicity) are too numerous to substantiate the use of structural similarity in regulatory decision making.

Policy Options

Number 3 on a single number versus a range with guidelines. This option appears worthy of much more thought as a viable alternative.

First of all, any evaluation of risk is complex and EPA should back and be responsible for the best judgement of risk. Either grided uncertainty factors, or case-by-case assessment of uncertainty factors on judgment are less arbitrary than the generic 1000 fold factor or the endpoint of a specified risk level.

Number 4 on the risk level. If this alternative is left in the document, emphasis should be placed on the fact that the linear multi-stage/model is an upper limit value, and that even a solely genotoxic substance is highly likely to present a much lower risk.

DEPARTMENT OF HEALTH & HUMAN SERVICES

Phone (501) 541 4000
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Public Health Service
Food and Drug Administration

National Center
For Toxicological Research
Jefferson AR 72079

Roy E. Albert, M.D.
Chairman, Carcinogen Assessment Group
Office of [illegible] Environmental Assessment
U. S. Environmental Protection Agency
401 M Street
Washington, D. C. 20460

Dear Roy:

As I mentioned to you last week, I am sorry about the delay in responding; however, things have been a bit tight recently. I appreciate the opportunity to review the document entitled, "Additional U.S. Environmental Protection Agency Guidance for the Health Assessment of Suspect Carcinogens with Specific Reference to Water Quality Criteria." My comments are attached for your consideration.

Sincerely,

Ronald W. Hart, Ph.D.
Director, NCTR

Attachment
as stated

ADDITIONAL U.S. ENVIRONMENTAL PROTECTION AGENCY GUIDANCE FOR THE HEALTH ASSESSMENT OF SUSPECT CARCINOGENS WITH SPECIFIC REFERENCE TO WATER QUALITY CRITERIA

Comments by:

Ronald W. Hart, Ph.D., Director, NCTR

A. General Comments

1. This document appears to base the plausibility of linear extrapolation on the mutagenicity of a carcinogen. This may represent an argument for low dose linearity, but it may not be necessary or even the major argument. Approximately one new cancer case occurs in the U.S. every minute. Thus, at any given instant there are a number of individuals who have a carcinogenic body burden insult which is almost adequate to initiate or promote a particular carcinogenic process. When a chemical carcinogen is introduced into an individual's environment, it may not enter as an increase above a zero dose but as an increase to an already existing carcinogenic exposure. If this already existing level of exposures is producing cancers, the detoxification/modulator processes are either already overloaded or circumvented. If the addition of a carcinogen adds to the existing level of exposure, then an additional increase in a particular cancer incidence may result. Regardless of the shape of the dose response curve, a short segment of this curve (corresponding to a small increase in dose) may be able to be approximated by a straight line (see Figure). This so-called theory of "background additivity" predicts low dose linearity of carcinogenic processes quite apart from mutagenicity. Thus, while the correlation between mutagenicity and carcinogenicity is sufficient to justify the use of mutagenicity data in assessing a potential carcinogen, it is not strong enough to imply that somatic mutation is the proven mechanism of carcinogenesis and that the linearity of carcinogenesis with respect to dose is dependent exclusively on

knowledge of that mechanism. Further, at this time there may be enough dose-response studies with [illegible] to support the linear extrapolation model without evoking mutagenesis as the sole basis for the mechanism.

Finally, as noted in [illegible], even though mathematical models may fit in the experimental dose range, they can not be used for low dose extrapolation because of wide discrepancies that appear at low doses. Point or best estimates can not be obtained without knowing the exact dose response model. Since the true model can not be obtained from bioassay data, all that can be accomplished is to consider the uncertainties in risk assessments. If the dose response is curving upward, linear extrapolation below the experimental dose range provides an upper limit on risk. This should be able to be accomplished with bioassay data alone and develops an argument independent of, but enhanced by mutagenicity data.

2. While it appears reasonable to use the confidence limits from the linear multistage model as one means for arriving at "safe" exposure levels for an animal population, it might also be reasonable to include some reference to the relative sensitivities of humans and test animals as a final step.

The conventional toxicological approach discussed on page 9 should be better defined, delineated or referenced, as should the mathematical procedures used for dose extrapolation.

Further, it may well be the case that this so-called conventional toxicologic approach gives essentially the same answer as the linear multistage model if it is also tied to the same level of risk, as it should be. The advantage of the so-called conventional toxicologic approach is that it is very simple and akin to conventional procedures. It should be cited that estimating a low dose response level, e.g. the 10% response level, and extrapolating linearly below that point has been proposed by Van Ryzin

J. Occup. Med. 22, 1980) and by Farmer, Kodell, and Gaylor (Risk Analysis 2, 1982) except that they suggested using the 1% response level.

Also, can one really call this approach a conventional toxicologic approach, which is based on the no-observed-effect-level (NOEL). Even if the NOEL exists, it has no precise meaning because it depends on the number of animals tested and hence provides a different level of risk in each case. Since the proposed technique selects a given response level (10%) and then applies an uncertainty (safety) factor, would it not be more proper to call this approach a "safety factor procedure" or "low dose linear extrapolation", since the two are almost identical.

3. The argument in paragraph 2 on page 10 is not logical. In the previous paragraph, a factor of 10 was imposed because of the uncertainty of sensitivity of humans. If then the data are available in humans (and presumedly their sensitivity is then known), one can not justify the use of the same uncertainty factor because you now have a greater certainty. If one is uncomfortable with only a factor of 100 when the data are presumedly more reliable, then the safety factor in less certain cases should be reevaluated. Therefore, the two paragraphs at the top of page 10 could, if you like, be replaced by the following paragraph:

> If the dose response curves upward, reducing the dose by an uncertainty factor of F will reduce the risk by at least a factor of F. Thus, starting at the dose that gives a 10% response, dividing by an uncertainty factor of 10,000 should reduce the risk to below one in 100,000. If the extrapolation is based on animal rather than human data, an additional factor may be employed depending upon the relative sensitivity of humans to animals.

3. Page 8, paragraph 2, line 2. Change "to use" to read "a modification of"; line 6, change "conventional toxicological" to read "modified".

4. Page 9, paragraph 2, line 1. Change "the conventional" to read "a modified"; line 2, delete "for an added refinement of making", replace with "which makes"; line 9, after "...tumorigenic responses." add the following sentence, "Also, NOEL has no definition and is a statistical result which depends on the number of animals tested." Line 14, after "....is recommended", insert "to estimate the 10% response level"; line 16 after "....upper confidence limit.", delete period, insert "on the response."

5. Page 10, delete paragraphs 1 and 2. Please see General Comments, above.

6. Page 12, sub-paragraph D, line 6, delete "In order to incorporate evidence for mutagenicity.", begin new sentence, "It is....."; line 7, change "both" to read "either", change "and" to read "or", change "conventional" to read "modified"; line 8, delete "a range of"; line 9, change "conventional" to read "modified". The two approaches are nearly the same when a risk level of 10^{-5} is specified for both.

7. The modified exposure limit needs to be tied to risk. It is difficult to provide an exposure limit without any notion of the degree of protection that it might provide.

8. Page 13, first paragraph. Delete last sentence: "It is proposed...... ....toxicological method."; second paragraph, delete all of the paragraph down to last sentence, "Such guidance, together....". As was stated above, one could divorce the process from mutagenicity and use other reasons for low dose linearity.

9. Page 13, sub-paragraph E, line 1. Change "could" to "should". Reference

last sentence of paragraph No. 1 - Why shouldn't they be employed? Can we be so comfortable with best estimates that we don't need to allow for sampling errors and experimental variability?

DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH

July 2, 1982

NATIONAL INSTITUTE OF
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P.O. BOX 12233
RESEARCH TRIANGLE PARK, N.C. 27709

Roy E. Albert, M.D.
Chairman
Carcinogen Assessment Group
Office of Health and Environmental
Assessment
U.S. Environmen Protection Agency
Washi 460

Dear Roy:

I read with interest your proposed risk estimation procedure. I am basically sympathetic with your concern over the various possible mechanisms of carcinogenesis and how they should be treated from the standpoint of low-dose estimation. Basically you are breaking out those compounds which are not gene mutagens and applying a type of safety-factor approach to them. In general, I find the use of safety-factors to be quite unreliable and very ad hoc as I tried to explain in the attached manuscript.

Specific problems I have with your approach are as follows:

(1) You assume the dose-response function for carcinogenesis is quite different (i.e. non-linear) when the chemical does not cause gene mutations. The philosophy that we took in Hoel et al. 1975 (enclosed) is that for these cases we assume that a linear function is a reasonable upper bound to the dose-response function. If we actually understand something for these cases we should then use the information. The question then is how different from linearity are your non-gene mutagens? I do not believe you have any real idea except possibly in a few specific cases.

(2) If a material is positive in one assay for gene mutation and the result is quite reproducible why do you require a second type of assay? This seems biased in the direction of minimizing your false positives at the expense of greatly increasing your false negatives. I am convinced that an Ames positive rodent carcinogen should be assumed to behave linearly at low dose after kinetic adjustments have been made.

(3) The safety factor of 1000 applied to an estimated 10% response is equivalent to extrapolating linearly to a risk level of 10^{-4} (with the confidence interval and species factor dropped). Since you usually

apply a confidence interval plus a species difference by surface area I would expect the two methods to differ by a factor of 100-500 (using 10^{-5} for the linear extrapolation) which is what your examples generally showed. Since you are using linear extrapolation and are, I believe, not assuming thresholds, why not simply state that you use 10^{-4} instead of clouding the issue with safety factor statements.

(4) You should be prepared to defend the possibility that 10 non-mutagens which are carcinogenic and linear could yield a total risk of $10 \times 10^{-4} \times 10 = 10^{-2}$ if a species factor of about 10 is reasonable (NAS Safe Drinking Water Report, Volume 3, page 49, for an argument for surface area).

From these comments you see that I am fairly negative with regard to "cookbook" approaches. What I would do is as follows. Take each compound on a case by case basis and argue away from linearity if the data warrants it. This would include pharmacokinetic considerations, short term test results, shape of the carcinogenesis dose-response curve and speculation on the mechanism. For this information, try to estimate the low-dose effect in man and use whatever level of risk the EPA is politically satisfied with.

We should sit down next week and discuss this issue further since I believe your objectives are correct.

Best wishes,

David G. Hoel

David G. Hoel, Ph.D.

Enclosures

COMMENTS ON U.S. ENVIRONMENTAL PROTECTION AGENCY GUIDELINES FOR THE HEALTH ASSESSMENT OF SUSPECT CARCINOGENS WITH SPECIFIC REFERENCE TO WATER QUALITY CRITERIA

by John Higginson, M.D.
Universities Associated for Research and
Education in Pathology, Inc.
9650 Rockville Pike
Bethesda, Maryland 20814

Introduction:

In general, I find this document represents considerable improvements on previous efforts to provide guidelines for the health assessment of suspected carcinogens. This is due partly to increased emphasis on the "weight of the evidence" approach, and partly to the recognition that modern concepts of the nature of carcinogenic mechanisms have significant implications for regulation.

General Comments:

The following points appear of major significance.

1. While it is widely accepted that there is a no-threshold for all genotoxic carcinogens, this concept lacks scientific proof. In fact, as the complexities of mechanisms become better understood, as does the nature of DNA repair [illegible] of certain steps in carcinogenesis, many scientists believe that an operational threshold probably exists at least for certain genotoxic agents. At least mentioning this possibility may avoid criticism that the report still implies that one molecule of a genotoxic agent may carry a potential risk of cancer. In this sense the term "potential" is meaningless.

2. Conversely, however, it has still not been shown conclusively that all non-genotoxic carcinogens or cancer inducing agents necessarily have a threshold, although this may be true. Both the epidemiological and experimental data are limited. Further, this group represents a wide range of agents, operating through a variety of different often unknown mechanisms, so that it is not possible to classify such agents generically. Thus, I would make a distinction between DES or conjugated estrogens, which are strong promoters, and for operational purposes, can be regarded as carcinogens and such non-specific cancer enhancing agents as sucrose, etc. While a number of these non-specific agents may be found in water, they are probably of little significance since the levels are too low for practical consideration. However, for consistency in guidelines, their existence should be noted. This would suggest that non-genotoxic cancer inducing agents be considered on an ad hoc rather than on a generic basis.

3. There is considerable evidence that mutagenic and carcinogenic potencies are not equivalent. False positives with mutagens have been reported. Damage to DNA is not the equivalent of carcinogenesis, and I think there is increasing support for the view that significant genetic damage is necessary for transformation as opposed to a single point mutation. Thus, the text should at least leave open the view that genotoxic agents may not necessarily act through simple somatic mutations, but that carcinogenesis may be triggered by carcinogens through other mechanisms, e.g. transposition, oncogenes, etc. Accordingly, it should be noted that there are scientific limitations to accepting mutagenic tests as invariably indicating genotoxicity in terms of carcinogenesis.

4. It is most important to avoid the impression that genotoxic agents are "bad" carcinogens, and non-genotoxiic agents "good" carcinogens. Thus, some of the latter may be strong cancer inducing agents in the sense of DES, estrogens and possibly asbestos, whereas certain other genotoxic carcinogens may be so weak as to be non-carcinogenic for practical purposes.

The above comments do not mean that I believe there are no differences in the nature of genotoxic and non-genotoxic agents from both a biological and operational viewpoint, nor that there are not important differences in considering thresholds, differing types of agents. From an operational viewpoint, I believe the distinction is desirable and justifiable. I also believe that the scientific differences require more elucidation and should not depend only on mutagenic testing.

5. Biomathematical modeling: I believe it is desirable to emphasize that nearly all biomathematical modeling is used for operational convenience or to stimulate research hypotheses due to the absence of biological data. They are never to be regarded as substitutes for data in the real world. Thus, whereas there may be considerable rationale for using linear, non-threshold models within a species, for extrapolation of dose responses, there are considerable scientific limitations regar[illegible] such models for extrapolation between species, whether [illegible] toxic or non-genotoxic agents, in view of the many unknown or unmeasured parameters involved. The data are simply not there. Further, the choice of the models still remains subjective and, as pointed out in the guidelines, is dependent on the individual's views of the mechanisms involved. The rationale, I believe, for using such models for non-genotoxic cancer inducing agents is even less strong than for genotoxic agents, in view of the many possible mechanisms that may be involved. The distinction between a simple arbitrary safety factor and a biomathematical model may falsely give the impression of a non-scientific versus a scientific approach that is not the case. The fact that models are extensively used adds nothing to their scientific validity as they have not been adequately tested.

Specific Comments:

Page 2, Section 1:

The term "formal approach": I think a tentative formal approach would be more desirable terminology, as implying uncertainty and indicating that approaches may change as new information becomes available.

Page 2, Section 2

In addition to taking mutagenecity into account in quantitative risk assessment, would it not be worthwhile to add a phrase such as "any other metabolic reaction that may be pertinent to initiation", i.e. comparative carcinogen metabolism between species, Thus, pivotal differences between activating and inactivating enzymes, for example, may be identified, which may be very pertinent in quantitation of risks. This approach may become increasingly important in the future, if not widely used at present.

Page 3:

I am in agreement on the value of the IARC approach, which is based on collective judgement of experts. However, as you have pointed out, it is possible that much of the IARC's value depends on the fact that Lyon is over 3000 miles from Washington and its staff and workshop participants are divorced from non-scientific pressures. The IARC does not exist to help regulatory agencies, but to provide, generate and evaluate scientific data.

I should point out that there has been one criticism of the IARC guidelines which your group might wish to consider. That is in relation to inadequate or negative data. Thus, there are some scientists who believe that the IARC criteria are weighed against negative studies to emphasize only the positive. Thus, many negative studies are never published or evaluated. There is also a tendency to ignore essentially negative epidemiological studies, since these can never completely prove the negative, a limitation incidentally which is also applied to the study of weak carcinogens in animals. A notable exception is the report on fluoride in water. Thus, under certain circumstances, the negative data in humans may be quite considerable and sufficient to exclude a substantial risk to man at levels of past exposures, although insufficient "to exclude a minimal or non-detectable risk". Further, the data may indicate the absence of a human risk, but it may again be insufficient to exclude a minimal risk. I believe this qualification, in practice, may be important especially when [illegible]luating non-gentoxic agents and providing backup data for mathemati[illegible]polation.

Page 7:

My comment here relates to quantitiative risk estimation with respect to mutagenicity. In general, it has been agreed that with ionizing radiation it may be possible to calculate approximately the "target cell" dose. Our lack of knowledge of the modifications that occur in chemical carcinogens as a result of digestion, absorption, deactivation and inactivation, etc. is such as not to permit any estimates of target cell doses in man. This was the conclusion of a group meeting in Stockholm under Cedelof in 1979. I believe that this distinction between ionizing radiation and chemical carcinogenesis should be mentioned when discussing linear non-threshold extrapolation models. This would leave open the possibility of developing better methods of estimating target cell doses (DNA adducts). If cell markers can be identified in the future they should be used rather than exposure levels for modeling.

Page 8:

I find this page useful in that it does emphasize the importance of carcinogenic theory in terms of evaluation of carcinogens and the attempt to classify them into "genotoxic" and "non-genotoxic". I still believe this to be desirable if scientifically imperfect. I would, however, like to suggest that a further paragraph be inserted on page 8 regarding the "non-mutagenic" or "non-genotoxic" carcinogens. There is some evidence that the latter may fall into two groups. The former includes promoters and other non-genotoxic agents, e.g. estrogens, which may have a specific effect and as such be identified for operational purposes as carcinogens, even if the mechanisms are unknown. However, there are a certain number of non-specific agents, such as sucrose, unsaturated fat, etc., which under certain experimental circumstances induce cancer and cannot be readily distinguished from classical promoters. These theoretically could be called "carcinogens", but I think this would give rise to confusion among the public. It may be advisable, therefore, to give further consideration to the definition of carcinogen as used in the addendum, and to distinguish such agents from non-specific cancer inducers such as sucrose, unsaturated fat, etc. This will avoid the danger of inconsistency in distinguishing between specific and non-specific cancer inducing agents.

Page 9:

In principle, I am in agreement with the approach on this page and on page 10 regarding the principle of uncertainty factors. However, I believe that the argument needs some clarification. Should one not consider the possibility now of having two levels of uncertainty, firstly an arbitrary scale where no data are available and a second scale where certain factors can be considered and given numerical values. Thus, in the case of vinyl chloride the difference between the metabolism in the human and the rat liver has been put forward as an explanation for the different sensitivity of the two strains. Would it not be worthwhile to emphasize the fact that this approach may be increasingly practical in the future? In dealing with "non-mutagenic" carcinogens, are we yet in a position to say that they cause irreversible lesions? Certainly not in the early stages if we include promoters as carcinogens. Thus, to justify an additional safety factor based on the grounds that cancer is irreversible without more knowledge of mechanisms of the particular agent accordingly appears somewhat illogical. If such a safety factor should be added, this should be on the grounds of cancer rather than on a biological principle.

Page 11.

Page 11 raises the old problem that any gene mutation is always regarded as indicative of carcinogenic potential. A wide range of gene mutations may be produced by mutagens but those essential for carcinogenesis are unknown. I also think it worthwhile at some point on this page to mention more recent views regarding mechanisms of carcinogenesis depending on the transposition, oncogenes, etc.

In Conclusion:

I believe the guidelines represent a reasonable attempt to improve quantitative risk assessment through better use of scientific knowledge. As one who is not personally concerned in regulation, I recognize the dangers of overemphasizing the lack of scientific data and its uncertainty as leading to excessively restricting caution or a "do nothing" attitude. It is, therefore, desirable to accept imperfect conclusions for operational purposes although not scientifically proven. However, the guideline should be sufficiently flexible to allow modification in the light of new data without needing to be rewritten.

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August 23, 1982

To the Individuals on the Attached List:

Thanks for your very worthwhile comments on the EPA draft document: "Additional U.S. Environmental Protection Agency Guidance for the Health Assessment of Suspect Carcinogens with Specific Reference to Water Quality Criteria".

The roster of people who commented is enclosed. I think that you will agree that the group is as balanced and competent as one could hope to get. Copies of the responses are also enclosed.

The use of the IARC criteria for stratifying the weight of evidence for carcinogens was strongly supported; there were no dissenters. Of the 12 commenters that addressed the issue directly, all were essentially in favor. However, there were provisos that more attention be paid to negative evidence and that Group 3 not be designated as "possible carcinogens" but as agents for which no decision on carcinogenicity could be made, i.e., the original IARC language.

It is probable that the EPA could adopt the IARC approach without serious scientific opposition. Such a move may have a significant regulatory impact since agents classified under Group 3 would probably have less regulatory impetus then those in Groups 1 and 2.

The quantitative issue of distinguishing between mutagenic and non-mutagenic carcinogens is more complicated and the reactions were varied. As you recall, the draft document, in general terms, used the linear non-threshold approach for mutagenic carcinogens and the more or less conventional NOEL - safety factor approach for non-mutagenic carcinogens. Nobody gave unqualified support the the approach. Half the responders (11 of 22) gave qualified encouragement: Gehring, Hoel, Higginson, Magee, J. Weisburger, Pitot, E. Miller, J. Miller, Ray (verbal), Burger, Upton. Six of the responders were negative: Saffiotti, Weinhouse, Nelson, Perera, Weinstein, Hart. Four of the responders were non-committal: E. Weisburger, Neal, Tomatis,

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McClellan. The negative responders felt that the tests for distinguishing mutagenic from non-mutagenic carcinogens were not certain and even if they were, there is insufficient biological basis to treat mutagenic and non-mutagenic carcinogens differently. The negative responders would generally prefer the linear non-threshold extrapolation model to be used for both mutagenic and non-mutagenic carcinogens. Weinhouse, alone, is flatly opposed to quantitation; he is also opposed to the conventional NOEL - safety factor approach. Lewis is for the NOEL - safety factor approach for both mutagenic and non-mutagenic carcinogens.

My own attitude toward the regulatory distinction between mutagenic and non-mutagenic carcinogens is guarded. However, since there has been so much talk about the matter, it seemed appropriate to give the proposition a trial. Although it elicited some resounding Bronx cheers, there were a number of eminent scientists who think there is enough substance to the distinction to warrant pursuing the matter further. The comments point out the way to do this; namely, to include mutagenicity as one of a number of factors in making regulatory distinctions amongst carcinogens. In this regard, there were many comments to the effect that the approach needed more flexibility in terms of pharmacokinetics, evidence for reversibility as with promoters, DNA repair, target dose, etc.

As a procedural matter, both Hoel and Hart pointed out that the NOEL approach of applying a safety factor to a specified level of carcinogenic response is the same as using the linear non-threshold extrapolation model to determine the dose which is equivalent to a specified target level of risk. For example, the application of a safety factor of 1000 to a response level of 10% is the same as a linear extrapolation to a target risk of 10^{-4}. Hence there is no need to use different methods since they are interchangable by adjusting safety factors and target risk levels.

One possible approach to a revision that might accommodate the above comments, in the case of the water quality criteria, would be to use the linear extrapolation model to identify the concentrations equivalent to various target risk levels, (e.g., 10^{-7}, 10^{-6}, 10^{-5}, 10^{-4}) and to present guidance as to which range might be given greatest weight in the formulation of water quality standards. Standard setting would involve such guidance together with a consideration of all the practical concerns that go into such judgements, such as technical and economic feasibility. The recommendation as to the appropriate target range of risk would be based not only on mutagenicity but all of the above mentioned factors as well as any other scientific considerations that are relevant at the time. The argument would be that the linear extrapolation model provides only an upper limit to the risk estimate and that at the recommended concentration range the risks are believed to be significantly less than the indicated levels.

Any further reactions you might have would be welcomed. We will let you known how things develope.

Thanks again for your help.

Sincerely yours,

Roy E. Albert, M.D.
Professor and Deputy Director
Chairman, Carcinogen Assessment Group
U.S. Environmental Protection Agency

Enc.

cc: John Hernandez, Deputy Administrator
U.S. Environmental Protection Agency

Elizabeth Anderson, Director
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency

REA/mff

ADDITIONAL U.S. ENVIRONMENTAL PROTECTION AGENCY GUIDANCE IN THE HEALTH ASSESSMENT OF SUSPECT CARCINOGENS (draft June 21, 1982)

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July 2, 1980

Elizabeth L. Anderson, Ph.D.
Director
Office of Health and Environmental
Assessment
Environmental Protection Agency
Washington, DC 20460

Dear Betty:

I have reviewed the paper sent to us on June 28 entitled "Additional U.S. Environmental Protection Agency Guidance for the Health Assessment of Suspect Carcinogens with Specific References to Water Quality Criteria" as your office requested. I have enclosed a series of comments which you may find useful.

Thank you for permitting us to review this interesting paper.

Sincerely,

Edward J. Burger, Jr., M.D., Sc.D.
Director

EJB:vh

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

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[illegible] Kovalick, Jr.

OFFICE OF
POLICY AND RESOURCE MANAGEMENT

SUBJECT: Toxics Integration Task Force

FROM: Richard D. Morgenstern, Director
Office of Policy Analysis

TO: Assistant Administrators
Associate Administrators
Acting Regional Administrator, Region III

Attached is an Agenda for the first meeting of the Toxics Integration Task Force, and a statement of the issues that I discussed with you this week.

The Agenda is self-explanatory; if you have any comments please let me know. The issues statement summarizes our understanding of what Al Alm wants the Task Force to address, and includes a few suggestions on how we might proceed from here. It basically expands on the issues that came out of a meeting we had with Alm last week; I have attached the talking points from that meeting for your reference.

During the first meeting next Wednesday (Room 1109, West Tower, 10:00 AM) I hope we can reach agreement on:

1. A limited number of topics on which we can promise useful products to the Administrator;

2. Subcommittee assignments: who shall lead on each topic, and who else shall participate; and

3. General modes of approach for each topic area.

Attachments

Received:
Director
Office of Toxic Substances

AGENDA

Toxics Integration Task Force
June 22, 1983
10:00 AM to 12:00 PM

Review of Agenda and Schedule — Dick Morgenstern

Discussion of Issues and Decisions on Assignments
(15 minutes each)

A.1 Consistency in risk assessment
  - Problem definition
  - Goals and products
  - Proposed approach
  - Assignments

A.2 Consistency in risk management
  - Problem definition
  - Goals and products
  - Proposed approach
  - Assignments

B.1 High-visibility chemicals
  - Problem definition
  - Goals and products
  - Proposed approach
  - Assignments

B.2 Intermedia inconsistencies
  - Problem definition
  - Goals and products
  - Proposed approach
  - Assignments

C. Interagency coordination
  - Problem definition
  - Goals and products
  - Proposed approach
  - Assignments

C. Options for inter-agency coordination

Historic paper exists on this area

Problem statement: In many instances, whether EPA is planning to regulate a chemical or an industry source, the Agency's mandate may cover only a fraction of the overall risk associated with the pollutants in question. Other agencies, notably OSHA, CPSC, and FDA, may have equal or larger responsibilities through their control of occupational exposure, consumer products, and foods. Without coordination among these agencies, it is not likely that the Federal government will propose either complete or efficient regulation.

Questions:

What is broken?
What needs to fixed?

(a) How serious is this problem? Is it confined to a few chemicals or industries, or is it pervasive?
(b) If a consortium of federal regulators to coordinate the activities of the various agencies were established, what agencies should participate?
(c) On what specific areas (information collection, risk assessment, risk management, regulatory development, etc.) should the group concentrate its efforts?
(d) How can the question of leadership be resolved?
(e) How much authority should this consortium have?

Possible approaches (sequential, not mutually exclusive):

Yes

(a) Study of the history, successes, and failures of the Interagency Regulatory Liaison Group.
(b) Design alternative institutional structures for an interagency consortium to coordinate toxics-related activities; analyze advantages and disadvantages of each.
(c) Recommend a specific plan for establishing the consortium:
    - propose members
    - draft charter

To be decided at first meeting:

Scope of issue: is the problem of interagency coordination serious enough to merit establishment of an interagency consortium on toxics?

Are there any plausible alternatives to an interagency group?

MOUs?

How should the Task Force deal with other Agencies, if at all, during its investigation of this issue?

Subcommittee assignment, as appropriate.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

JUL 01 1983

SUBJECT: Work Plan for the Toxics Integration Task Force, Sub-Ta[illegible] Group on Risk Assessment

FROM: Elizabeth L. Anderson
Director
Office of Health and Environmental Assessment (RD-689)

TO: Richard D. Morgenstern
Director
Office of Policy Analysis (PM-221)

The task group met on June 28, 1983, and agreed to the following work plan.

I. The task group identified four main areas which need addressing:

1. Guidelines development for health and exposure assessment.
2. Procedures for ensuring consistency and technical competence in the Agency's health and exposure assessments.
3. A process for updating health assessments on which the program offices rely in their rulemaking processes. (Currently, the Agency has a process for updating the Ambient Air Quality Criteria Documents, but this is not the case for any other health assessment documents.)
4. Approaches to ensure that testing protocols to generate data for health risk assessments are consistent within the EPA and with other agencies.

II. Problem identification, work assignments, and schedules:

1. First priority will be given to the topic of guidelines development. The task group identified areas for potential guidelines development and a lead person was assigned to each area:

Carcinogen Risk Assessment	Robert McGaughy
Mutagenicity Risk Assessment	Peter Voytek Jack Fowle

CONCURRENCES

SYMBOL								
SURNAME								
DATE								

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Reproductive Effects Risk Assessment	Peter Voytek Carol Sakai
Other Chronic Effects Guidelines	Jerry Stara Irv Baumel
Complete Mixture Methodology	Jerry Stara
Exposure Assessment Guidelines	Jim Falco

Each EPA office representative will submit issues of concern to their office in each of these guidelines areas to the lead representative. Cross-cutting issues will be submitted to Betty Anderson. These work products are due on July 16, 1983.

At the next task group meeting, which is scheduled for July 18, 1983, the lead person for each guidelines area will present a brief report to the task group on the following:

- status report on current guideline development in EPA, other U.S. agencies and committees, and the international community
- Agency needs for further guideline development
- state-of-science on which to base further guidance (i.e., is enough known to go forward?)
- recurring scientific and regulatory issues relevant to the particular guideline area
- a suggested process to follow, as appropriate, for further guideline development
- estimated time frames for work completion

After the deliberations of the task group on July 18, 1983, the group will recommend which areas need further guidelines development to ensure consistency in the Agency's approach to health risk assessment and exposure assessment. In addition, as time permits, the task group will set priorities for these efforts, recommend a process to carry out the development of guidelines, establish target time frames for these efforts, and recommend lead individuals to take responsibility for the process.

2. As a second priority, the working group will consider a process for ensuring consistency and technical competence in the Agency's risk assessment program. The lead person for this study is David Ziegele. Each EPA office representative will submit an issue paper on this topic to David by July 16. David will assemble the information he receives and make a report to the task group on the following issues:

    - current status

- current problems
- practical need for an organized process
- recommendations

This topic will be addressed, if time permits, at the July 18 working group meeting. Final recommendations will follow the next working group meeting to be scheduled no later than early August.

3. The topic of periodically updating important health assessments will be addressed by the working group in early August.

4. The task group believes that it is important for the Toxics Integration Task Force to address the issue of consistent protocols for collecting test data for risk assessment. The risk assessment task group, however, recommends that this topic be addressed by the Interagency Coordination Task group. Irv Baumel is preparing an issue paper on this topic.

In summary, the task group has established a work plan to define what work needs to be done to improve the Agency's risk assessment program. The task group also plans to recommend a process for completing this work in a timely fashion. It is the intent of the committee to make its final recommendations as soon as possible, and no later than the end of September, so that the necessary work can go forward.

cc: C. Riordan (RD-672) 7476
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M. Williams (TS-792 - 3815
W. Kovalick (TS-777) - ~~3275~~ 3436
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J. Stara (EPA/RTP)
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FILE

TOXICS INTEGRATION TASK FORCE

RISK MANAGEMENT SUBCOMMITTEE (TASK A.2)

INTERIM REPORT

1. Introduction

This report summarizes the findings of Phase I of the Risk Management Subcommittee's work plan, a retrospective study of how information about risks and cost--the elements of risk management--was used in 27 recent Agency regulatory actions. It also proposes some activities suggested by these findings that would comprise Phase II of the Subcommittee's task: the development of a uniform reporting format for risk management information and the formulation of a uniform policy for managing cancer risks.

The reader should note the preliminary nature of this work: a substantial amount of data was submitted by the program office members of the subcommittee, which appears to warrant more extensive analysis, and a number of points require clarification, especially those relating to the subtleties of why a particular decision fell out the way it did.

The submitted data has been summarized and is presented here in a master matrix [Exhibit 1]. The column headings represent types of information that the subcommittee considered to be essential to any definition of risk management. The master is a condensation of the matrix the programs used in submitting data [Exhibit 2].

Although all the programs used the same matrix to report on their regulations, it is clear that the risk management form of analysis is more suitable to some programs than to others. Risk management can be variously defined. In the narrowest sense, it means deciding what to do about the results of a risk assessment. (Risk assessment itself is subject to a variety of definitions; here we follow the recent NAS Report, defining it as the characterization of potential adverse health effects of human exposure to environmental pollutants. This includes the establishment of safe levels as well as the probabilistic expression of risk used where no safe level can be set, as with carcinogens.)

In our attempt to put all the Agency's regulatory activities under a single rubric, however, we have stretched this definition. Risk management in the broader sense means: the collection and presentation of information about the impact of a pollutant (or a mixture thereof) on human health or the environment;

consideration of the uncertainty surrounding the imputation of such impact; and, consideration of alternative ameliorative actions, what is gained from them and what they cost, cost being defined as all economic and social impacts. This definition implies a balancing of values, and while it is certain that such balancing occurs at various levels in all EPA programs, it is clear from the present work that it does not conform to the same rules in all programs, nor does it always occur in ways that can be presented simply, as we have tried to do. Where we seem to show blanks in important areas of our matrix, it is important for us to determine whether the program has truly not considered a particular aspect of risk management (perhaps for good reasons) or whether the gap is an artifact of our reporting format. We intend to do this over the next few weeks and revise the present paper accordingly.

2. Preliminary findings

o There are significant variations in risk management practice across EPA programs.

We can identify at least three major distinctions among programs in this area: (1) programs based on statutes that require explicit balancing of costs and benefits, such as TSCA and FIFRA; (2) programs that, while they may calculate risks or benefits, are essentially technology-driven, such as Effluent Guidelines and NAAQS; and (3) programs that attempt to incorporate balancing judgements in various ways, but which are essentially risk-driven, such as NESHAPS, NSPS, and Superfund. In fact, all programs incorporate balancing judgements of some kind, but some do it more explicitly than others. The RCRA and Superfund programs have the most difficulty doing this in practice because they deal with complex mixtures of substances. In Superfund, moreover, risk management occurs at the site level; the headquarters staff is developing guidelines for this process.

o Different programs accept different levels of health effects evidence as prompting regulatory action.

There are a number of different systems available for ranking strength of evidence (IARC, Squires, etc.). These are not used in a consistent way. In fact, given any particular body of experimental or epidemiological data, it would be hard to predict what the Agency would do. Some differences may be justified, as, for example, the PMN program using structure activity relationships to prevent a product entering the market. Others appear idiosyncratic in our study; we want to explore this area further. An associated problem is the use of "numbers" like CAG risk estimates or Water Quality Criteria. RCRA and Superfund are especially dependent on such numbers, but there is some danger that numbers used in this way will take on a "magical" authority unjustified by their scientific provenance. There does not appear to be any Agency-wide way of assuring that risk estimators of this type are being used correctly and are updated regularly wherever they are involved in program policy-making.

- Pre- and post-regulatory risk levels, where calculated, have extremely wide ranges, both across programs and within the same program.

Although no reported action where a risk assessment was involved acted on a risk of less than 10^{-5}, there were three order of magnitude between the lowest and the highest risk reported as remaining after regulation. The marginal cost-per-cancer avoided was calculated only for one regulation; we will do likewise for the others where we have risk and cost information. On inspection, however, it seems likely that these will vary considerably as well. Naturally, there would be nothing wrong with a range this wide if it had some rational justification: cost, for example. We have no evidence for this as yet, but it is an important problem and deserves further analysis.

In a subsidiary study, (Exhibit III) we investigated the handling of the risk assessments for three chemicals of concern (mercury, endrin, and TCE) across different programs. There appear to be substantial differences in the acceptable daily intakes established by different programs for the same chemical. We intend to expand this analysis to include both more substances and other aspects of risk management.

- The attention paid to intermedia transfer of pollutants varies among programs.

There is some evidence that intermedia transfer is being considered more seriously than in the past, when it was often not considered at all. Effluent guidelines adds the costs of disposing of hazardous sludges to its control cost estimates. RCRA is concerned with waste dumps as a source of air pollution. TSCA typically examines all media in which a chemical appears. Despite this interest, in most cases in this study where tranfer occurred it was considered "not relevant." These may have been reasonable judgements, but we cannot be sure of this given the data on hand. The Agency has no general rules or standards concerning the management of intermedia transfer against which we could measure the adequacy of such judgements.

- Costs-benefit and cost-effectiveness analysis are performed differently in different programs.

The NAAQS program may not consider costs, by statute, although they report that a cost-benefit study has been done for the particulates NAAQS. The RCRA and Superfund programs did not report cost calculations for the regulations covered, although Superfund did submit site-specific reports for three sites in which costs and risks were considered and compared, although differently at each site. There is no common way of expressing the benefit of particular regulatory programs, and thus no way to insure that the array of risks the Agency is managing at any particular time includes the most serious ones.

3. Phase II Products

If more uniform risk management is a desireable goal, these preliminary results suggest some ways of approaching it. It seems clear that whatever risk management techniques are appropriate to a particular program (and these may legitimately vary) there should be a uniform way of reporting and recording these decisions. We should use the experience of preparing the Phase I report to develop formats for uniform presentation of risk management information, including uniform rules for calculating and expressing exposure, intermedia transfer, benefits and costs of regulation, to Steering Committee and the Administrator.

Beyond reporting, there remains the question of unifying policy toward the endpoint that appears to be of most concern in this area, cancer. The Agency attempted to develop a cancer policy in 1981 and much of that work will still be useful. That study was carried out by a group at the division-director level, reporting to the Toxic Substances Policy Committee, an office-director level body. The cancer policy group attempted, as a first step to set some *de minimis* level for cancer risk and reached agreement on 10^{-6}. More difficult was the problem of how to handle maximum individual risks; the Agency has always had difficulty in situations where high risks that are expensive to control affect a small number of people. The group did not reach closure on this issue.

We intend to pick up on that work and attempt to delineate what combinations of risk, certainty, and cost ought to constrain particular Agency regulatory actions with respect to carcinogens.

EXPOSURE SUMMARY

Name and Type of Regulation/ Standard/Guideline	Route	Source	Population Considered	Intermedia Transfer
Air				
Arsenic NESHAPS	Inhalation	Copper smelters; Glass Manufacturers	All U.S. w/in 20 Km. of Source	Not Considered
Benzene NESHAPS	Inhalation	Maleic Anhydride; Ethyl Benzene/Styrene	All U.S. w/in 20 Km. of Source	Not Considered
Radionuclide NESHAPS	Inhalation	DOE/NRC Licensees Facilities; Underground Uranium Mines; Elemental Phosphorous plants	Max. Exposed Ind. within 80 Km. of Source	Solid Waste collected is buried at those low-level radioactive waste sites
Particulate NAAQS	Inhalation	Numerous & diverse sources	All U.S. population	None
Water				
Iron & Steel EG	Ingestion of Water and Organisms	Aquatic Life and Drinking Water	Population surrounding ind. pt. sources	Not relevant
Copper Forming EG	Ingestion of Water and Organisms	Aquatic Life and Drinking Water	Population surrounding ind. pt. sources	Not relevant
Benzene WQC	N/A	N/A	N/A	N/A
Toxaphene WQC	N/A	N/A	N/A	N/A
Endosulfon WQC	N/A	N/A	N/A	N/A
Drinking Water				
Trihalomethane MCL	Ingestion of Drinking Water	Drinking Water Treatment Process	U.S. Pop. served by pub. water sys (80%)	Not relevant

Exposure
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Name and Type of Regulation/ Standard/Guideline	Route	Source	Population Considered	Inter. Transfer
Solid Waste				
Criteria for Listing (§3001)	Ingestion of Ground Water; Inhalation; Dermal	RCRA regulation of hazardous waste management site	Hypothetical Population consumes water at maximum contamination point	Not relevant
RIA for Landfills	Ingestion of Ground & Surface Water	RCRA regulated land disposal sites	Defined by drinking water well use	
RIA for Incinerators	Inhalation; Ingestion & Dermal	RCRA reg. incinerators	Pop. ≤ 20 Km; 20-50 Km of source	Phase II will Address Dermal & Water
Superfund				
Remedial Action Hazard Ranking System	All Routes (Inhalation, Ingestion, Dermal)	Release from Uncontrolled Waste Sites	All affected Pop. Groundwater-3mi. radius; Air-4 mi. radius Surface Water-3mi. radius; Dermal-1 mi. radius Fire-2 mi. radius	N/A
Pesticides				
Amitraz RPAR	Dietary, Dermal, inhalation; Environmental	Residues in treated crops, meat & milk; mixing lodging & picking of crop	General U.S.; Fruit growers, applicators, pickers	Through spray drift
Chlorpyrifos Emerg. Exemption	Dietary	Treated grains & by-products	General U.S.	
Terbufos Registration Standard	Dietary; Dermal; inhalation Environmental	Residues in treated crops, meat & milk; pesticide application	General U.S.; Applicators; Aquatic Invertebrates & Vertebrates; Terrestrial org.	Fate considered for environment
Permethrin Tolerance Action	Dietary	Residues on Crop, Milk & meat	General U.S.	Not considered

Exposure
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Name and Type of Regulation/ Standard/Guideline	Route	Source	Population Considered	Intermedia Transfer
Toxics				
PMN #81-356	Ingestion of drinking water	Drinking water	General U.S.	N/A
PMN #81-357	Inhalation	Dust particles in textile plants	textile plant workers	N/A
PMN #82-251	Dermal	Spraying of paint stripper	Paint stripping applicators	N/A
Mesityl Oxide (Test Rule)	Inhalation	Industrial Fugitive Emissions	Industrial, Consumer & Gen-U.S, Pop.	N/A
C-9 Aromatic Hydrocarbons (Test Rule)	Inhalation	Refueling; transport truck; solvent use	Tank truck operators; service station attendant general U.S. Pop. (self-serve gas)	Water considered
PCB Electrical Transformer Use Rule	Inhalation, Dermal, Ingestion (food, water)	Leaks & Spills from Elec. Equipment	U.S. & World workers who serve equipment	qualitative treatment
MDA (§4f, decision to regulate	Inhalation and Dermal	Workplace Air	Workers at manufacturing & processing sites (Approx. 12,000)	Not relevant
NTA (§6, decision to regulate	Ingestion of Drinking Water	Prod. releases to surface water; consumer releases to surface & ground water	Entire U.S. pop.	None expected

COST/BENEFIT ANALYSES SUMMARY

Name and Type of Regulation/ Standard/Guideline	Regulatory Alternatives Presented	Cost-Effectiveness Analysis	Final Considerations Governing Decision
Air			
Arsenic NESHAPS	1. BAT Approach 2. Risk based	(1) $3,600/Mg (2) $85,500/Mg	Control all sources to at least BAT
Benzene NESHAPS	De-listing Benzene	(3) $158,000/Mg (4) $5/Mg (5) $350-940/Mg (6) $400/Mg (7) $990/Mg (8) $0/Mg	Decision not final
Radionuclide	Regulate - NRC/DOE facility licensees, Underground Uranium Mines, Elemental Phosphorous Plants Not Regulate - Coal Fired boilers, phosphate industry other extraction industry, and low energy accelerators	Costs were considered but no formal C/B analysis	1. Individual vs. Population Risks 2. Potential for Including Emissions 3. Available & Cost of controls 4. Effect of other Standards
Particulate NAAQS	Ranges of standards presented	Marginal benefits estimated; varies with alternative standards	Protection of health and welfare w/safety margin
Water			
Iron & Steel EG	8 Treatment levels presented	Est. Annual Cost $47.5 million Est. Annual Benefit $35.9--$727.7 million	Consent Decree/BAT
Copper Forming EG	4 Treatment levels presented	NA	" "
Benzene WQC	NA	NA	NA
Toxaphene WQC	NA	NA	NA
Endosulfan WQC	NA	NA	NA

Name and Type of Regulation/ Standard/Guideline	Regulatory Alternatives Presented	Cost-Effectiveness Analysis	Final Considerations Governing Decision
Drinking Water			
Trihalomethane MCL	Ranges of levels presented	$200,000/cancer case avoided	Feared tighter standard would lead smaller systems to stop chlorinating
Solid Waste			
Criteria for Listing (§3001)	Full spectrum of RCRA requirements	Not addressed	None
RIA for Landfills	1. Require ideal facility design 2. Spectrum of designs 3. Case-by-case guide	$/life saved	Unknown at this time
RIA for Incinerators	1. Permit Flex. 2. Performance Standard 3. Var. from 99.99% DRE on case-by-case risk analysis	Not addressed	Unknown at this time
Superfund			
Remedial Action	Determined site by site	Not finalized	Determined Site by site
Hazard Ranking System	NA	NA	NA
Pesticides			
Amitraz RPAR	1. Allow registration; 2. Use restriction 3. Conditional Registration data gathering; 4. Ban	Expressed in cases/yr. avoided; cost data on "Risks posed were minimal" for pears & apples	Need for pest control on apples not significant--was banned. Control of pests on pears was needed -- reduced exposure

Cost/Benefit
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Name and Type of Regulation/ Standard/Guideline	Regulatory Alternatives Presented	Cost-Effectiveness Analysis	Final Considerations Governing Decision
Chlorpyrifos Emergency Exemption	NA	Kansas claimed $750K loss prevention if use authorized--risks "minimal"	Risk-benefit analysis was driving force
Turbufos Registration Standard	NA	NA	Labeled prod. because of high acute toxicity Not demonstrated to cause unreasonable adverse affects
Permethrin Tolerance Action	NA	NA	No residues were detected on the raw commodity after exaggerated application rates were used.
Toxics			
PMN # 81-356	1. Section 5(e) order banning use pending further testing.	Voluntarily withdrawn	Manufacturer withdrew substances
PMN # 81-357	2. Labeling & use of respirators	"	"
PMN #82-251	"	"	"
Mesityl Oxide (test rule)	NA	Annualized costs of testing $116,510-$354,330 or $0.06-$0.19/lb.	NA
C-9 Aromatic Hydrocarbons (test rule)	NA	Annualized costs of testing $133,100-$398,500 or $.0004/lb.	NA

Cost/Benefit
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Name and Type of Regulation/ Standard/Guideline	Regulatory Alternatives Presented	Cost-Effectiveness Analysis	Final Considerations Governing Decision
PCB Electrical Transformer use Rule	1. Ban too costly 2. Equipment phase-out 3. Frequent inspections 4. containment	$237.00 per lb. not released	Court challenge of earlier regulation
MDA ($4F,decision to reg.)	Not completed	Not completed	Pre-regulatory decision allows more risk of error
NTA ($6, decision to drop)	NA	NA	NA

HAZARD ASSESSMENT SUMMARY

Name and Type of Regulation/ Standard/Guideline	Effect Endpoint	Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
Air				
Arsenic NESHAPS	Lung Cancer	Animal - Inadequate (IARC) Human - Sufficient (IARC)	(1) $2.3x10^{-1}$ or 1.1--17.4 cases/yr. (2) $4.3x10^{-3}$--$6.9x10^{-2}$ or 0.1--1.6 cases/yr. (3) $6.4x10^{-4}$--$1x10^{-2}$ or 0.07--1.2 cases/yr	(1) $5.8x10^{-3}$--$9.2x10^{-2}$ or 0.21--3.4 cases/yr. (2) $9.0x10^{-4}$--$1.5x10^{-2}$ or 0.04--0.64 case/yr. (3) $9.7x10^{-5}$--$1.6x10^{-3}$ or 0.01--0.21 case/yr.
Benzene NESHAPS	Leukemia	Human - Sufficient (IARC)	(4) $5.6x10^{-4}$--$3.8x10^{-3}$ or 1 case in 6 yrs. (5) $2.9x10^{-5}$--$2.0x10^{-4}$ or 1 case in 15-90 yrs. (6) $5.3x10^{-5}$--$3.7x10^{-4}$ or 1 case in 65-450 yrs. (7) $1.4x10^{-5}$--9.5x10-5 or 1 case in 9-60 yrs. (8) $3.1x10^{-3}$--$2.2x10^{-2}$ or 1 case in 0.1-1 yrs.	(4) $1.7x10^{-4}$--$1.2x10^{-3}$ or 1 case in 3-20 yr. (5) $4.2x10^{-6}$--$2.9x10^{-5}$ or 1 case in 15-110 yrs. (6) $3.5x10^{-6}$--$2.4x10^{-5}$ or 1 case in 380-2600 yrs. (7) $1.1x10^{-5}$--7.6x10--5 or 1 case in 10-70 yrs. (8) $1.3x10^{-4}$--$9.3x10^{-4}$ or 1 cases in 2-10 yrs.
Radionuclide NESHAPS	Fatal Cancer	Radiation is a proven carcinogen	(9) $5x10^{-3}$ (10) $1-2x10^{-2}$ (11) $1x10^{-4}$	(9) $2x10^{-4}$--$4x10^{-5}$ (10) $2x10^{-3}$ (11) $3x10^{-5}$
Particulate NAAQS	Reduced lung function, increased respiratory symptoms	NA	Not available	Not available
Water				
Iron & Steel Effluent Guideline	Chronic aquatic toxicity (fish); aesthetics	Not an issue	Cyanide 59% proj. exceedance of 3.5 ug/L WQC Phenol 65% proj. exceedance of 1.0 ug/L WQC	Cyanide 16% proj. exceedance 3.5ug/L WQC Phenol 7% proj. exceedance 1.0 ug/L WQC

Hazard Assessment
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Name and Type of Regulation/ Standard/Guideline	Effect Endpoint	Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
Copper Forming Effluent Guideline	" "	"	Copper 35% proj. excued of 5.6 ug/L WQC	Copper 12% proj. exceed of 5.6 ug/L WQC
Benzene WQC	Leukemia	Human - Sufficient (IARC)	Not a Regulation	6.6 ug/l = 1x10^{-5} risk
Toxaphene WQC	Ecological Effects	Not an issue	" "	Acute: 1.6 ug/l Chronic: .013 ug/l
Endosulfan WQC	Acute Toxicity	Not an issue	" "	75 ug/l = NOEL
Drinking Water				
Trihalomethane MCL	Liver and Kidney Tumor	Animal - Sufficient (IARC) Human - Limited (IARC)	2.0--2.4x10^{-3} or 256-743 cases/yr.	4x10^{-4} or 200-580 cases/yr.
Solid Waste				
Crit. for Listing (§3001)	All Chronic effects observed in animal population	Carcinog. pot. determined by CAG LOAELs;	Not addressed	Not addressed
RIA Landfills RIA Incinerators	" " " "	NOELs determined by ECAO; ADI calc. uses NAS wt. of evidence	Analysis not complete " "	Analysis not complete " "
Superfund				
Remedial Action	Varies from site to site	Varies	Varies from site to site	Not finalized

Hazard Assessment
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Name and Type of Regulation/ Standard/Guideline	Effect Endpoint	Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
Hazard Ranking System	Acute Toxicity	None	Risks among sites ranked	High score - incl. on list
Pesticides				
Amitraz RPAR	lympho-reticular tumors	Weakly positive evid. (CAG)	1.10^{-4} - apple use 1.2x10^{-5} - pear use	Zero risk for apple use Several orders of magnitude less for pear use
Chlorpyrifos Emerg. Exempt	Cholinesterase inhibition (more sensitive human)	Registration studies met guidelines requirements		
		NOEL - .25 ppm ocular lesions	NR	NA
	Cholinesterase inhibitor & exopthalmia (human)	Unreasonable adverse effects to estuarine org.	Not determined	Not determined
Terbufos Registration Standard	Mortality to fish; aquatic invertebrates and birds	IARC - limited; Squire - class V		
Permethrin Tolerance Action	Liver oncogenicity (animals)	IARC - limited; Squire class V Weisburger & Williams - epigenetic;	Tolerance levels set on chronic liver data	Tolerance levels estimated diet residue levels with an adequate margin of safety

Hazard Assessment
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Name and Type of Regulation/ Standard/Guideline	Effect Endpoint	Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
Toxics				
PMN # 81-356	Carcinogenicity	Speculation based on extrap. of analog	No significant data except structural similarity to another carcinogen	None - PMN withdrawn
PMN # 81-357	Carcinogenicity	" "	" "	" "
PMN # 82-251	Fetotoxicity; reprod. effects; ecotoxicity Carcinogenicity; Eye corrosivity; chloroacne	" "	" "	" "
Mesttityl Oxide (test rule)	Carcino-, Muta-, & Teratogenicity	NA	NA	NA
C-9 Aromatic Hydrocardon (test rule)	Muta-& carcino- genicity; Chronic terotogen	NA	NA	NA
PCB Elec. transformer Use Rule	Cancer; Reproductive Developmental Effects	NA	Not Reported	Not Reported

Name and Type of Regulation/ Standard/Guideline	Effect Endpoint	Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
MDA (§4f, dec. to reg.)	Cancer	Animal - Sufficient (CAG)	$1x10^{-4}$--$1x10^{-3}$	Not Reported
NTA (§6, dec. to drop)	Cancer	Animal - Sufficient (NCI)	(12) $7.7x10^{-8}$--$7.7x10^{-7}$ or $4.7x10^{-7}$--$4.7x10^{-6}$	Not Regulated
			(13) $1.6x10^{-9}$--$1.9x10^{-6}$ or $9.9x10^{-9}$--$1.2x10^{-5}$	None Calculated

Footnotes:

(1) High Arsenic Copper Smelters
(2) Low Arsenic Copper Smelters
(3) Glass Manufacturing
(4) Benzene Fugitive Emissions
(5) Maleic Anhydride
(6) Ethyl Benzene/Styrene
(7) Benzene Storage
(8) Coke By-Product Plants
(9) DOE/NRC Facility Licensees
(10) Underground Uranium Mines
(11) Elemental Phosphorus Plants
(12) Drinking Water from Surface Water
(13) Drinking Water from Ground Water

EXPOSURE SUMMARY

Superfund

Remedial Action	Route	Source	Population Considered	Intermedia Transfer
Eq. 1 Matthews Electroplating, Roanoke Co., VA	Ingestion of Groundwater	Release from uncontrolled waste site (chromium)	Populations exposed via drinking water from three separate wells	Ambient air and soil were considered and then discounted because of small contributions to total daily intake
Eq. 2 Lehigh Elec. & Eng. Co., Inc. Lackawanna Co., PA	Ingestion of soil; dermal contact with soil; and inhalation	Release from uncontrolled waste site (PCBs)	3 years (children); 7 years (youngsters); and adults were considered who lived within close proximity to site	Indirect exposure via surface and groundwater was also considered but was not significant
Hazard Ranking				
Eq. 1 Love Canal Niagra Falls, NY	Ingestion of drinking water from surface water; inhalation from ambient air in basements	Release from uncontrolled waste site (trichloroethene, tetrachloroethene, and Pentachlorobutadiene from air and dioxin and lindane from surface water)	Population served by surface water 77,000 people - Population within 4 mile radius by ambient air 1100 people	Groundwater was considered but was not significant
Eq. 2 Carolawn Co. Site Fort Lawn, S.C.	Ingestion of drinking water from ground water; recreation and fishing from surface water	Release from uncontrolled waste site (1,1,1-trichloroethane trichloroethene and 1,2 dichloroethene from groundwater; trichloroethane from surface water	1200 people served by groundwater 0 people served by surface water	Air and dermal routes were considered but were determined not to be significant

Exposure (Cont.d)

Superfund Hazard Ranking	Route	Source	Population Considered	Intermedia Transter
Eg. 3 Stringfellow Riverside, CA	Ingestion of drinking water from ground and surface water; inhalation of ambient air; irrigation of land from surface water	Release from uncontrolled waste site (ground water: chromium, nitrates, chlorides, sultates; surface water: DDT chronium & DDE; Air: Air: cyanides, acids, chlorides, nitrates, sulfides	Ground water - 16,724 people Surtace water - 16,724 people Air - 16,724 people within 0-4 mile radius	All media considered
Eg. 4 FMC Corp. Fridley, MN	Ingestion of drinking water from ground and surface water	Release from uncontrolled waste site (groundwater: acetone, dichloroethene mineral sprits trichloroethene; surtace water: 1,1,2-trichloroethene, trans 1,2 Dichloroethene, methylene chloride, tetrachloroethane, tetrachloroethene	Groundwater - 30,089 people Surface water - 466,415 people	Air considered but was not signiticant

HAZARD ASSESSMENT

Superfund Remedial Action	Effect Endpoint	Tox.	Persis.	Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
Eg. 1 Matthews Electroplating Roanoke Co., VA	Tubular necrosis of the kidney; and hemorrhaging of the gastro-intestinal tract			Acceptable daily intake developed by NAS (1980) 100 fold safety factor over maximum NOAEL	Three wells were contaminated approximately 2 to 4 times the 50 ug/l drinking water standard	No risk - Municipal drinking water supply was extended to users of the three wells
Eg. 2 Lehigh Elec. and Eng. Co., Inc. Lackawanna Co., PA	Carcinogenicity; Teratogenicity			Not mentioned	At max. conc.of 110,000 ppm: 1×10^{-1} - 8×10^{-1} At Av. Conc. of 3700 ppm 5×10^{-3} 2×10^{-2}	At 50 ppm 4×10^{-5} - 1×10^{-4}
Hazard Ranking						
Eg. 1 Love Canal Niagara Falls, NY	Lindane Dioxin Perchloroethene	3 3 3	3 3 -	None	Surface water score = 60 Air Score = 67.56 Total Score(Sm) = 32.04	This score represented inclusion on the superfund remedial list. (National Priority List)
	3 is maximun score available according to NFPA					
Eg. 2 Carolawn Co., Site Fort Lawn, SC	Trichloroethane	3	3	None	Groundwater Score = 55.10 Surface water score = 6.04 Total Score(Sm) = 32.04	"
Eg. 3 Springtellow Riverside, CA	DDT & DDE Chromium PCBs	3 3 3	3 3 3	None	Groundwater Score = 83.67 Surface water score = 65.45 Total Score (Sm) = 61.40	"

Hazard Assessment (Cont'd.)

Superfund Hazard Ranking	Effect Endpoint		Strength of Evidence	Pre-Regulatory Risk	Post-Regulatory Risk
Eg. 4	Trichloroethene			Surface water Score = 89.09	
FMC Corp.	Tetrachloroethene			Total Score(Sm) = 74.16	
Fridley,	Lead	3	3 None		
MN	Chromium				
	Cadmium				
	Methylene Chloride				

COST/BENEFIT ANALYSES SUMMARY

Superfund Remedial Action*	Regulatory Alternatives			Cost-Effectiveness Analysis	Final Considerations Governing Decision
	Alternative	Total Cost			
Eg.1 Matthews Electroplating Roanoke Co., VA	1. No on-site Action	$ 19,800		Est. cost of extending municipal drinking water supply - $954,000	Decision based on lowest cost alternative that is technologically feasible and reliable and which -- provides adequate protection of public health...
	2. Surface Mgt.	87,900			
	3. Site Capping	131,000			
	4. Contaminated Soil Removal	1,241,000			
	5. No off-site Action	40,800			
	6. Community Well Sys.	923,000			
	7. Extension of Municipal water Supply	$ 954,000			
	Alternative	Cap. Cost	O&M Cost		
Eg. 2 Lehigh Elec. and Eng. Co., Inc. Lackawanna Co., PA	1. No Action	$ 10,000	$ 135,000	Est. cost of excavation and off-site disposal; demolition of buildings; and backfilling, grading and vegetating of site and monitoring and main - $6,447,000	" "
	2. Removal to 10 ppm & site mgt	7,546,000	34K-54K		
	3. Removal to 10 ppm & site capping	7,725,000	"		
	4. Removal to 50 ppm & site mgmt.	6,140,000	"		
	5. Removal to 10 ppm & site capping	6,284,000	"		
	6. Removal to 10 ppm & site Encaps	6,448,000	"		
	7. Removal to 50 ppm with additional excavation on where cost-effective site mgmt.	6,401,000	"		

[*Note: Cost-Benefit considerations not included in Hazard Ranking System.]

Hazard Ranking			
Eg. 1 Love Canal Niagara Falls, NY	Not included in Hazard Ranking System		
Eg. 2 Carolawn Co. Site Fort Lawn, SC	"	"	"
Eg. 3 Stringfellow Riverside, CA.	"	"	"
Eg. 4 FMC Corp. Fridley, MN	"	"	"

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WASHINGTON, D.C. 20460

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OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

TO: Dick Morgenstern, Director
Office of Policy Analysis

SUBJECT: Interagency Coordination--Proposed Initiatives

This memorandum reports on the deliberations to date, as well as the initial recommendations, of the Interagency Work Group. As directed by the Deputy Administrator and your Task Force, we are proposing a portfolio of actionable initiatives--several of which could be developed into broad-scale, continuing mechanisms for interagency coordination, should they prove successful. By "portfolio", we mean that the choices to be made are not mutually exclusive, as the collection of initiatives offers some variety of subjects and mechanisms.

Thus, we have presented preliminary options--some or all of which may be selected by the Task Force for presentation to the Deputy Administrator. The proposals reflect issues of immediate concern to the Agency. The work group presents them with the understanding that the options for implementation, resource implications (both dollar and staff) description of the expected results, and time frames were not possible to develop in the time since your last meeting. We propose such work be done--probably by the lead offices for items selected for interagency activity.

Summary of Recommendations

Our portfolio of recommendations contains six initiatives for interagency coordination concerning both science and regulation. Four of the six deal with solving specific chemical assessment, regulatory process, or implementation problems. The implementation mechanisms developed for each can be considered as potential models for implementatation of other (generic or specific) problems that EPA shares with other agencies. These four initiatives are: Unified Assessment/Regulation (cadmium); Coordinated Risk Assessment; Regulatory Coordination (grain fumigants) and Coordinated Compliance.

A fifth initiative deals with another dimension of interagency relationships--expanding and enhancing EPA's connections with a single agency across all of our programs. The Centers for Disease Control (CDC) and the Occupational Safety and Health Administration (OSHA) are presented as two candidates.

The sixth initiative proposes early interagency coordination on an emerging high-technology issue of congressional and public interest. Bio-technology--the innovative combination of microorganisms as new products (with the assocated manufacture, production, use, release into the environmennt, and disposal issues)--is already confronting two of EPA's regulatory programs. Other Federal programs are already involved from a research viewpoint.

The work group views another issue--EPA's dioxin project--as a major, high priority, Agency activity with important interagency coordination aspects concerning risk assessment, regulatory coordination, and waste site clean up. We did not list it in our portfolic because the parallel activity underway on that project will contain interagency coordination plans.

Background for Recommendations

In order to develop our recommended portfolio, we conducted a census of problems or opportunities from the program offices. An abbreviated listing of these items and potentially affected EPA programs and Federal agencies appears in Attachment A. The variety in scope, degree of specificity, relative involvement of programs/agencies, elapsed time to develop solutions and/or to solve the problem is obvious from this list.

Many of these problems can be grouped under the generic functions of EPA programs:

- o unified assessments
- o regulatory coordination
- o implementation/compliance
- o communication/ information exchange

Attachment B groups most of the issues under functional headings, although some issues can easily fit in several classes.

All Federal agencies that either test or evaluate chemicals, regulate chemicals or industrial categories, or are major owners of Federal facilities are potential participants in EPA's interagency activities. Another way to view the problems and opportunities, is the frequency of other agency involvement. When the problems and opportunities are grouped under these functions, certain agencies are first tier of concern for EPA coordination. Thus, HHS(CDC), OSHA, FDA, and CPSC emerge as important players on most topics with DOD and DOE being important as owners of Federal facilities.

In addition to this census of program submissions, the work group reviewed materials describing the IRLG experience, a detailed evaluation of interagency coordination[1], recommendations of the Administrative Conference of the U.S. on these subjects, and two GAO Reports[2].

Criteria for Selection

We developed and applied five major criteria for the selection of our initiatives from the candidate list: 1) need or importance; 2) potential for success and benefit to the Agency; 3) focus on the areas of science and regulation; 4) results can be achieved within a short period of time; and 5) issues selected offer potential for a variety of mechanisms and approaches that can be used for resolution. Criteria three and four are derived explicitly from the Deputy Administrator's remarks and the ensuing discussion at your last task force meeting.

Primarily because of the regional and enforcement representation on the work group, the compliance initiative is proposed even though some work group members believe it falls outside a strict construction of criteria three. Similarly, although the bio-technology initiative seems to be a longer term (vs. criteria four) project, its unique character and the current public interest caused us to recommend it for your consideration.

Detailed Proposals

See Attachment C for Discussion of Issues and options for resolution.

I. UNIFIED ASSESSMENT

*Unified Assessment/Regulatory Coordination--Cadmium

A. Recommendation: An interagency work group should be established for the purpose of 1) developing a consensus on its health and environmental effects, estimates of cadmium's body burden (Task 1), and 2) coordinating the regualtions of cadmium across the Federal establishment (Task 2).

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[1] U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances: Improving Federal Integration and Coordination Efforts, January 1981.

[2] GAO Report, Gains and Shortcomings in Resolving Regulatory Conflicts and Overlaps; June, 1981
GAO Report, Grain Fumigation: A Multifaceted Issue Needing Coordinated Attention; September, 1981.

B. Lead Office: Task 1, ORD (OHEA); Task 2, OAQPS
Program Office Involvement: ORD, OAQPS, OSWER, OW, OTS
Other Agencies: OSHA, CPSC, FDA, USDA

C. Mechanism: Work Group

II. COORDINATED RISK ASSESSMENT:

°Risk Assessment

A. Recommendation: Federal agencies should sponsor a workshop, possibly hosted by NAS, to review and update the 1979 Scientific Basis for Identifying Potential Carcinogens and Estimating Their Risks. The Administrator should meet with the heads of other agencies dealing with cancer regulation and research, encourage support for this recommended approach, and develop a collective agenda for the workshop. The workshop has several optional focal points; all aspects of quantitative or qualitative hazard assessment for carcinogens expressed in the 1979 document or only portions of these earlier guidelines (e.g. with/without consideration of exposure assessment).

B. Lead Office: ORD (OHEA)
Program Office Involvement: ORD, OSWER, OPTS, OW, OANR
Other Agencies: OSHA, CPSC, HHS, ...

C. Mechanism: Workshop

°Test Guidelines and Good Laboratory Practices

A. Recommendation: An interagency work group should be established consisting of science policy managers and science experts, to establish, on a continuing basis, a process for developing baseline guidelines for government testing and good laboratory practices. Industry and public interest groups will be involved, as needed in the process.

B. Lead Office: OPTS
Program Office Involvement: OPTS, OSWER, OW, ORD, OANR
Other Agencies: FDA, NTP, NIOSH, OSHA, NIH

C. Mechanism: Work Group

III. REGULATORY COORDINATION

°Grain Fumigants

A. Recommendation: An interagency work group should be established to consider the scope of the problem and develop an approach for the coordination of regulatory actions and coverage.

B. Lead Office: CCS
Program Office Involvement: OPP, OAQPS, OSW
Other Agencies: OSHA, DOT, FDA, USDA,

C. Mechanism: Work Group

IV. COMPLIANCE/IMPLEMENTATION:

°Interagency Referral of Compliance Information

A. Recommendation: Informal discussion should be held, initially with FDA, OSHA, and USDA to discuss the idea of developing a referral/inspection process for the purpose of identifying easily detectible violations that may indicate a firm may be in violation of major standards enforced by another agency. This effort may be planned on a pilot regional or national basis.

B. Lead Office: Region III
Program Office Involvement: Regions, Enforcement Offices for each Program
Other Agencies: FDA, OSHA, USDA

C. Mechanism: Informal discussion

V. FOCUSED INTERAGENCY ACTIVITIES:

°Centers for Disease Control

A. Recommendation: An intragency work group should be established, composed of Headquarters and Regional representatives to discuss the coordination of activities with CDC. The goal of effort is the development of a generic MOU stating Agency interests concerning EPA/CDC relationships. Specific projects will be developed by the program/regional offices and funded, as necessary, by IAG's.

B. Lead Office: OPTS/CCS
Program Office Involvement: OSWER, OPTS, OW, ORD, OANR

C. Mechanism: Work Group/MOU

°Occupational Safety and Health Administration

A. Recommendation: The Administrator/Deputy Administrator should discuss area needing interagency coordination with the Administrator of OSHA. A lead EPA as well as OSHA office should be established.

B. Lead Office: OPTS
Program Office Involvement: OSWER, ORD, OW, OANR

C. Mechanism: Informal discussion

VI. INFORMATION EXCHANGE

°Bio-Technology

A. Recommendation: An interagency work group should be established to coordinate the evaluation of potential risks from bio-technology. An IAG to establish and support the expert group should be developed.

B. Lead Office: OTS
Program Office Involvement: OPP, ORD, OSW, OW, OAQPS

Other Agencies: NIOSH, FDA, CDC, NIH

C. Mechanism: Work Group

Action Items for Task Force Consideration

In order to proceed with these short term initiatives, the following action items are recommended:

1. Feedback from the Task Force and decisions by the Deputy Administrator concerning which issues should be pursued.

2. Once the issues are selected, Lead office representatives for each inititative need to be assigned to develop briefing materials for the Administrator--complete in 3 weeks of item #1.

3. The Administrator/Deputy Administrator should meet with each of the affected agencies and discuss areas EPA proposes for interagency coordination and obtain from the other agency heads their concurrence on the need to coordinate the specific items.

4. Detailed implementation plans need to be developed by the lead offices--complete within 4 weeks of item #3.

Two other items of importance need to be highlighted. First, for several of these initiatives, EPA's own activities may not be fully coordinated. Lead office recommendations for such internal coordination for each issue should be part of the detailed implementation plans (item #4 above). Second, a number of the topics and problems on our interagency list may merit further attention by the Agency, but were not included in the recommendations. You may wish to consider further reports or other inquiries of the relevant Assistant Administrators as to the seriousness of these issues, and recommended approaches for their resolution.

Walter W. Kovalick Jr

Walter W. Kovalick, Jr.
Chairman, Interagency Coordination
Work Group

Attachments

cc: Interagency Coordination Work Group Members
Marty Halper
Jake Mackenze

ATTACHMENT A ISSUES/PROBLEM MATRIX

Issue	What	Who															
		OSHA	CPSC	FDA	HHS	OANR	OSWER	OPTS	OW	ORD	NRC	DoE	DoD	DoI	DoT	DoL	S
Integrated exposure assessment	assess. & risk mgt.	x	x	x	x	x	x	x	x	x							
Testing protocols Math models for risk assessment Acceptance of studies	repro., mut., terat.; evidence of car., mut. chronic & acute effects	x	x	x	x	x	x	x	x	x							
Regulatory coordination																	
venting	OSHA venting std., ETO, Solvents	x				x											
cadmium	cadmium regulation	x		x		x				x							
formaldehyde	formaldehyde regulation	x	x			x		x		x							
Federal Radiation Guidance	policy level (plut. & occ. guidance)				x	x					x	x	x		x	x	
OECD Nuclear Energy Agency	lack of rep.					x					x	x					
Indoor-air (radon)	assessment of risk				x	x					x	x					
Workplace hazard regulation	existing & new chem. sharred priorities jurisdictional risk assessment	x						x							x		
Chemical labeling	regulatory coordination	x	x				x	x							x		x
Drug Precursors	info. coord. existing & new chem.	x		x				x									
DOD contract specs. procurement guidelines	existing chem.							x						(OFPM)			

Dioxin	coordiantion of clean up activities	x	x	x		x	x	x	x			
CDC relationship	coordiantion of activities			x	x	x	x	x	x			
Bio-technology	need for coordination	x	x	x	x	x	x	x	x	x		
Agricultural Non point sources	suface and groudwater pollution							x		(USDA)		
Acid mine drainage	clean up of sites										x	
Hazarous Waste Transport controls	jurisdictional					x						x

Testing protocols/ guidelines including good lab. practices	science and regs.	x	x	x	x	x	x	x	x	x	x	x
Interagency Assessment & Regulation	assessment and regs. benzene, formaldehyde, priorities	x	x	x	x	x	x	x	x	x		
Preserved wood/food	jurisdictional reg. coverage							x				
Worker exposure (pest)	jurisdictional	x						x			(USTR)	

Issue	What	OSHA	CPSC	FDA	HHS	OANR	OSWER	OPTS	OW	ORD	NRC	DoE	DoD	DoI	DoT	DL	S
Grain fumigants	jurisdictional risk mgt. grain poli	x		x				x					x		x		x
Animal damage	differing mandates							x						x			
Consideration of endangered spec.	RPAR review							x						x			
Uniform site risk analysis	analysis of haz waste sites				x		x										
On-site federal authority	one authorative voice				x		x								(FEMA)		
Federal Superfund sites (GOCO)	clean up						x					x	x	x	(GSA, NASA,USDA)		
National Resource Damage Assessment	damage format enforcement coordinati						x							x	(USDA)		
Federal Assistance to states CERCLA actions	mointoring, site evaluation						x							x	x		
Federal facility comp.	notification review omissions (privately owned) not joint with CERCLA						x					x (NASA)	x	x	x		
RCRA AEA facilties	regulatory interpre. jurisdictional disp.						x					x					
Acceptable levels in Food	need to set acceptable levels			x		x	x	x	x	x							

ATTACHMENT B   REGULATORY PROCESS MATRIX

Identification of Toxic Problems and Trends ========>	Multimedia Exposure Documents/Methods Development ========>	Assessments: Health/Environmental Risk ===================>
Interagency Chemical Assessment	Integrated Exposure Assessment	Math Models for Risk Assessment
CDC Relationship	Testing Protocols	Acceptance of Studies (Cd, Benzene, Formaldehyde)
Bio-technology	Good Lab Practices	Indoor Air (Radon)
	Interagency Chemical Assessment	Interagency Chemical Assessment
	Site Risk Analysis	Site Risk Analysis
	Federal Assistance to State CERCLA Actions	Dioxin
	Acceptable levels in Food	Acceptable levels in food
	Consumer Exposure to Solvents	CDC Relationship

Regulatory Coordination/ Consistency =======> Implementation/ Compliance

Venting
Cadmium
Formaldehyde
Workplace Hazard Regulation
Chemical Labeling
Interagency Chemical Regulation
Pesticide Worker Exposure
Grain Fumigants
Animal Damage
Acceptable levels in Food
Agricultural Non-point Sources
Federal Radiation Guidance

OECD Nuclear Energy Agency
Pesticide Worker Exposure
Federal Superfund Sites
Federal Facility Compliance
RCRA AEA Facilities
Acid Mine Drainage
Hazardous Waste Transportation

Information Exchange/Review

Drug Precursors
DOD Contract Specs.
Endangered Species
On-Site Federal Authority
National Resource Damage Assessment
Bio-technology

ATTACHMENT C

C 1 Unified Assessment/Regulatory Coordination--Cadmium

C 2 Risk Assessment

C 3 Risk Assessment: Test Guidelies and Good Laboratory Practices

C 4 Regulatory Coordination--Grain Fumigants

C 5 Impelementation/Compliance

C 6 Focused Interagency Activities--CDC

C 7 Focused Interagency Activities--OSHA

C 8 Information Exchange--Bio-technology

Attachment C 1

I. Item: Unified Assessment and Regulatory Coordination on Cadmium

Interagency Coordination on Assessment and Regulation of Cadmium Prior to the demise of the IRLG, both interagency and intra-Agency coordination of cadmium activities were achieved through staff-level participation in IRLG meetings. The potential health effect of primary concern was kidney dysfunction (cadmium accumulates in the kidneys). EPA, required by section 122 of the Clean Air Act to announce a decision, had determined that regulation of cadmium as an air pollutant was not necessary to protect the public from cadmium-induced kidney dysfunction. This determination and the attendant health and technical analyses were discussed with IRLG participants.

Subsequently, EPA deferred the annoncement of the decision not to regulate cadmium as an air pollutant in order to update the assessment of the carcinogenic and mutagenic potential of cadmium and incorporate the findings in the decision process.

Recently, EPA's Office of Health and Environmental Assessment (OHEA) completed its updated assessment and made the draft report available for review and comment. (See the Federal Register of July 17, 1983, pages 31297 and 31298.)

II. What is the issue and why is it important?

There are two issues, one related to science, the other regulation. The first issue is whether cadmium and/or cadmium compounds are to be considered human carcinogens. Several of the offices and other Federal agencies regulate cadmium and others do not, but none treats it as a human carcinogen. It is important that all be made aware of OHEA's updated report and consider its significance to their programs. The second issue is the agencies' regulatory response (i.e. risk management) and the coordination of those activities. EPA may be setting standards for the ambient air which indicate that the OSHA standard is not stringent enough.

III. What other agencies are involved?

The following is not necessarily a complete listing. Other agencies that were involved include:

1. OSHA - Had established workplace standards for air concentrations of cadmium fume and cadmium dust.

2. FDA - Had set "action levels" for the amount of cadmium allowed in ceramic holloware and flatware. Did not, nor intend to, limit the amount of cadmium in food.

3. Dept. of Agriculture - Was considering establishing a requirement for removal of the kidneys from fowl before mechanical de-boning.

EPA requirements for cadmium had been established for drinking water, water quality, industrial effluents, waste disposal, and the use of sludge on croplands.

IV. Mechanisms for resolution.

Each agency or office must decide for itself, based on available data and on its policy, what action is appropriate. It is important, however, to exchange information so that conflicting conclusions and actions, and glaring inconsistencies might be avoided. In order to develop a coordinated regulatory approach to cadmium activities concerning the assessment of cadmium can be undertaken either in parallel with on-going regulatory development or serially.

V. Recommended Option

It is recommended that an interagency Agency work group be established for the purposes of exchanging information and reporting on activities relative to cadmium. Only small additional EPA effort would be required to establish, and participate with such a group. Two groups should be established; one lead by ORD (OHEA) to examine the health effects and a second group lead by OAOPS to coordinate interagency risk management.

Attachment C 2

I. ITEM: INTERAGENCY RISK ASSESSMENT

During the period 1975 to 1980 the Interagency Regulatory Liaison Group Work Group on Risk Assessment developed a mutually satisfactory and acceptable position on the scientific basis for the identification of potential carcinogens and estimation of risks. The IRLG report on risk assessment was published in 1979 both in the Journal of the National Cancer Institute and the Federal Register. These documents became the basis for EPA's cancer policy and was also considered for the cancer policies of OSHA, CPSC, and FDA. The IRLG document focused on risks of cancer. The document described the basis for qualitative evaluations of whether a particular substance presented a carcinogenic hazard to man. The document presented how the results of epidemiological studies, animal bioassays along with other types of information were to be used making evaluations that a particular substance was carcinogenic. The second thing the document did was to discuss and present methods for quantifying estimates of carcinogenic risk posed by a particular type substance, if such risk estimates were appropriate or required. The IRLG report represented the first time Federal agencies had taken a unified stand on risk issues and issues of uncertainty which greatly affected their regulatory activities. Since the IRLG report, much new information has become available on risk of cancer. The work has been discussed by institutions such as the National Academy of Sciences and other scientific communities, especially with regard to estimation of risk posed to the public by potential carcinogens.

II. WHAT IS THE ISSUE AND WHY IS IT IMPORTANT

With regard to interagency coordination, the major concern centering around the IRLG report is the currentness of the material which was published in 1979. Since 1981 the IRLG has failed to function as an interagency group. However, in the last three years, much new information has become available throughout the scientific community which directly affects the contents of the IRLG report.

The IRLG report is often cited as the basis for regulatory actions both in EPA as well as in other agencies. The issue posed at this time is the update of the report and how Federal agencies involved with the assessment of carcinogens and risk of potential carcinogens should go about updating the work.

III. WHAT OTHER AGENCIES ARE INVOLVED

The IRLG report of 1979 involved EPA, the Consumer Product and Safety Commission, the Food and Drug Administration, Occupational Safety and Health Administration. All these agencies involve themselves with the regulation of potential carcinogens. In addition to these agencies' interest in risk assessment, the Center for Disease Control, NIEHS, and the National Cancer Research Institute are other Federal agencies which are deeply involved in the national issues surrounding cancer and risk assessments. For the purpose of coordination, all agencies involved in risk assessment, especially those named above, should be encouraged to become involved in interagency efforts to review and revise the IRLG's scientific basis for the identification of potential carcinogens and the estimation of risk.

IV. MECHANISMS FOR RESOLUTION

Essentially there are two possible mechanisms for reviewing and revising the IRLG report of 1979. The first mechanism could be that used in the period 1975 to 1980 by resurrecting the IRLG and revisiting the work of that group using the same procedure and format. The second mechanism, could be to encourage the Federal agencies to focus on the National Academy of Sciences to hold an international forum or symposium. The topic would be "The scientific Basis for the Identification of Potential Carcinogens and Estimation of Risk." The symposium sponsored by the Federal agency could be established specifically for updating, reviewing and revising the position on carcinogenesis and estimation of risk which was presented some four years ago.

V. RECOMMENDED OPTIONS

The option recommended for the update of the report on risk assessment is for the Federal agencies to sponsor a workshop which may be conducted by the National Academy of Sciences. The lead office in EPA to coordinate the activity should be the Office of Research and Development. Specifically in that office, the Office of Health and Environmental Assessment should be given the responsibility to conduct this workshop. The mechanism recommended is for the Administrator of EPA to officially contact the heads of other agencies dealing with cancer regulation and research and encourage the above option. Main resources required are senior EPA staff to coordinate. Sums of money required for such a workshop and update are estimated to be about $200,000 for the conduct of the workshop. Low estimate for such a workshop would be $100,000 and a high estimate would be $300,000. These amounts are those which are not already committed by the Agency.

Attachment C 3

Interagency Coordination
Test Guidelines
and
Good Laboratory Practices

I. Test Guidelines and Good Laboratory Practices

This need is for interagency agreement on the acceptable, baseline methodologies for conducting toxic effects and other tests. Since the data produced by testing are the decisional base for risk assessment, agencies must begin with consistency in the data they use if they are to have consistency in risk assessment.

A successful effort was carried out at the international level, through the OECD, to arrive at consensus guidelines (protocols) for some tests. The FDA and EPA were participants.

This project would be a continuing one, since improvement in the scientific state-of-the-art requires methods to be updated frequently. However, there would also be short-term goals for the work.

II. Issues and Importance

Various regulatory and science agencies either perform testing or require it from regulated industries. It is essential not only that the agencies agree on acceptable baseline protocols for government testing, but also that they are consistent in what they require of the regulated industries.

III. Other Agencies Involved

Agencies include FDA, EPA, NTP, NIOSH, OSHA, and NIH.

IV. Mechanism

Option 1: The model would be the successful OECD effort. In that case there was a three-tiered structure. At the top were the policymakers (here it would be the Administrator and AA's and their peers). The next tier was the science polity managers (Office Director/Division Director). The last tier was science experts. Experts were drawn together as a work group for each protocol (or several related ones). Their recommendation was reported to the science policy managers who in turn reported recommendations to the policy level for adoption.

The objective is not to achieve perfect standards for testing. This would not permit the flexibility that is needed to accomodate interests of particular programs or the improvements that science can achieve over time.

Option 2: Option 1, but with requirements that make the agreed-upon baseline guidelines mandatory rather than advisory.

Option 3: Option1, and include industry and public interest group representatives on the expert groups.

V. Recommended Option

° Options 1 and 3 are recommended. Option 2 which would make guidelines mandatory would be less likely to succeed since it would require agencies to give up flexibility.

° The lead office could be OPTS or ORD. OPTS is recommended because it contains two programs that have statutory requirements for industry testing and, under TSCA, statutory requirements for producing test guidelines. Either way, both offices would have to participate fully.

° The resources required would include;

- planning and setting up an interagency group with an agreed charter.
- personnel to work in expert groups and policy level groups
- funds to support validation of some (not all) protocols

Attachment C 4

Grain Fumigants

I. Item: Coordination on Grain Fumigants

II. Issue

Fumigants to control pests in grain are of interest to several Federal agencies. Approximately 10 fumigants are used to protect grain from insect damage. EPA regulates the use of fumigants as pesticides; OSHA has authority in the workplace of those who handle or inspect grain as opposed to those who apply the pesticides; FDA monitors levels of fumigant residues in processed grain products; DOT defines the manner in which grain may be safely transported; and USDA helps shape the market trends and affects the marketability of grain. GAO did a report several years ago noting the lack of coordination between Federal agencies addressing policy issues or regulations affecting grain fumigation. In fact, there may be conflicting activities between agencies. For example, while the FDA may take legal action against processed grain products containing an excessive number of insect parts, USDA grades grain on the basis of live insects only. Policies of USDA which tend to lead farmers to store more grain also encourage increased fumigation activity. EPA is restricting the use of several grain fumigants, but still needs to pursue the interagency issue. The issue is important because of the GAO attention, and the legitimate issues of inter-agency jurisdiction and grain policy raised.

III. Other Agencies involved

OSHA, DOT, USDA, FDA

IV. Recommended Option

a. Lead office: Chemical Coordination Staff, OPTS; optional lead: Office of Pesticide Programs.

b. Mechanism: Formal interagency Work Group or Task Force should be formed to consider scope of problem and discuss course of coordinated action which can be taken by all agencies involved. Formal agreements, e.g. MOU's, could result from the efforts of the Work Group. Participation on the Work Group should be Branch Chief and/or Division Director level.

c. Resources required: Medium.

Attachment C 5

INTER-AGENCY COORDINATION COMMITTEE
IMPLEMENTATION AND COMPLIANCE PROJECT

I. ITEM: Implementation of Mechanism for the Interagency Referral of Compliance Information For Use in Targeting Use of Inspectional Resources.

A. Background.

A large number of firms subject to EPA regulation are also subject to regulation by other Federal agencies. A pharmaceutical 4irm which operates pursuant to EPA-issued water, air and waste management permits is also subject to FDA good laboratory practice standards and OSHA worker safety requirements. A meat or poultry processing house must operate in compliance with USDA standards for pesticide residues, OSHA workplace requirements, as well as EPA standards for pest management.

Each regulatory agency monitors compliance with its standards based upon its own program priorities and enforcement strategies. Each conducts periodic inspections of regulated firms as resources allow. In the absence of rigorous interagency planning, each agency would inspect a given firm to assure compliance with its standards. Flagrant violations of sister Agency's standards would go unnoticed and unreported. Furthermore, the operations of the firm would be interrupted as inspectors from each Agency appear in turn to conduct inspections.

B. Referral Inspection Process.

With a limited amount of orientation training, the field staff of each of the principle regulatory agencies could become competent to observe easily detectible (clearly visible) indications that the firm may be operating in violation of major standards enforced by a sister agency. Using a standard format, the inspector would notify a predetermined contact in the sister agency of what he has observed This information would be used by the receiving agency in determining its inspection/monitoring priorities. Where the receiving agency determines that the information warrants follow-up, it will inspect the firm and initiate appropriate corrective action.

To assure maximum value to the program, it should include the following design:

-- Identification of Referral Indicators: The standards targeted for orientation training and referral must be both (1) easily identifiable, and (2) of sufficient importance to the receiving agency that the receiving agency will want to follow-up. This will require careful selection of agency-by-agency referal indicators. As examples, the agency identified below would be interested in observations of the followings compliance problems

oo EPA: Foamy buildup around permited outfalls; leaking pesticide/waste drums; workers in the field during pesticide application; black plumes from smoke stacks; stagnant, non-maintained pits, ponds and lagoons at industrial sites; leaking PCB transformers in food processing establishments.

oo FDA: Damaged food containers, rodent droppings/insect infestations in food processing plant.

oo OSHA: Slippery floors, open vats or toxic fumes in chemcal/pesticide plants; absence of protective clothing/equipment; farm equipment without precautionary signs;

-- Training of Field Staff. The inspectors employed by each participating agency must learn to identify the key referral indicators. This training may be done by classroom or video instruction, augmented by a training manual containing all relevant training data for periodic self-instruction.

-- Transmittal of Referrals. A simple one page Inspection Checklist of referral indicators should be utilized by the field staff of the interested agencies to remind inspectors of the types of items they should try to notice. Another one page tear-off Referral Form will be used to transmit referrals to sister agencies.

--Follow-up By Receiving Agency. Each agency will determine whether a referral warrants a follow-up inspection. In some instances the receiving agency may already be aware of the problem referred, or may determine that the issue does not pose a serious problem and competing program demands will predominate.

II. Benefits To Be Achieved By Implementing An Inspection Referral Program.

Implementation of an interagency referral mechanism would effectively expand each Agency's field presence. It would provide valuable intellegence regarding instances of probable violations which the receiving agency can utilize in estbilishing compliance monitoring priorities. Since the indicators would focus on violations of concern to the receiving agency, it would minimize the referral of information of no programmatic concern.

Finally, the implementation of a formal interagency referral mechanism would enhance the credibility of each agency's compliance programs. Inspected firms would realize that an inspection by representatives of any participating agency could yield a referral to other agencies. It would have a "multiplier" effect on the overall regulatory impact of each inspection conducted by each agency.

III. Participating Agencies.

The agencies whose participation would be most useful are: EPA, FDA, OSHA, USDA.

IV. Mechanism for Implementation.

A. Formal Implementation.

A formal mechanism of interagency referral would best be implemented by the execution of appropriate MOUs or IAGs. To magnify its importance with the regulated community, the execution of the formal agreement could coincide with a campaign announcing the initiation of the program.

This campaign would need to be preceeded by discussions with large trade associations of those firms most likely to be impacted by interagency referrals to assure that the intent and process of the program is not misunderstood. Unwarranted objections to the program (regarding, for example, right of entry, cross-agency delegation of enforcement authority) must be ancitipated and directly addressed.

B. Informal Implementation.

A considerable number of interagency referrals occur already. If the execution of an MOU or IAG becomes a cumbersome process, a less formal approach could be undertaken. Interagency contacts could be made at the Office Director level among interested program managers. These individuals could establish Headquarters and Regional contact persons, agree to train their respective staffs, and agree to utilize the referral process without a formal MOU or IAG.

V. Recommended Action.

It is suggested that Region III develop implementation options for both a pilot regional or a national effort in consultation with EPA headquarters enforcement programs.

Attachment C 6

I. Item: Coordination of Activities with the Occupational Safety and Health Administration (OSHA).

The Occupational Safety and Health Administration (OSHA) within the Department of Labor is responsible for administering and enforcing the Occupational Safety and Health Act. The Directorates of Health Standards Programs and Safety Standards Programs are responsible for the development and continued review of mandatory health and safety standards concerning worker exposure to toxic chemicals and general "physical" hazards in the work place.

Under section 6 (a) of OSH Act, OSHA has designated over 400 chemical substances as air contaminants and promulgated exposure standards, threshold limit values (TLV's) for them. Under section 6 (b), OSHA has developed health standards for several chemicals to include: Asbestos, Benzene, Inorganic Arsenic, and Lead.

II. What is the issue and why is it important:

Each of EPA's program activities potentially impact and may be impacted by the activities of OSHA. A critical aspect concerns the need for interagency coordination for the evaluation of the health effects of various toxic chemicals. EPA as well as OSHA each assess chemicals to determine their acute and chronic (carcinogenic and non-carcinogenic) hazards. Each EPA Office and OSHA then as a result of these assessments make an appropriate regualtory response (based upon policy and statutory authority). Indenpendent assessment and regulatory development may result in conflicting, inconsistent and duplicative activities. Past regulatory or judical actions by OSHA (e.g Benzene) have affected the regulatory activities of EPA. There is a critical need to assure that the actions of each of the agencies are connected especially since regulations developed under authority of RCRA, TSCA, FIFRA, and CAA directly or indirectly impact worker exposure. Conversely, regulations developed by OSHA (e.g. requiring or permitting work place ventilation) can affect EPA programs.

III. What EPA Offices are Involved:

Potentially each of the EPA program offices may, at one point in time, have a need to coordinate with OSHA. The Office of Toxic Substances as a result of the new and existing chemicals programs may find the need to coordinate the regulation of a chemical through record keeping and reporting regulations or explicit control (use or exposure). The Office of Pesticides Programs regulates worker (applicator) exposures to pesticides and is concerned about worker exposure to pesticides in the non-field agricultural setting which is not subject to EPA regulation under FIFRA. The Office of Solid Waste and Emergency Response is constantally involved in worker exposure problems concerning developing regualitons for hazardous waste facilities and the clean up of hazardous waste sites. The Office of Air Quality Planning and Standards through the assessment of the need to regulate potentially toxic air pollutants becomes involved in worker exposure issues and the effects of air contaminants on health. The Office of Reasearch and Development, because of development of health and environmental assessment and risk assessment documents, is concerned about the need to have consistent assessments on chemicals of concern and an appropriate response by the agencies (e.g. cadimum).

Current activities of OSHA may also have a regulatory impact on on-going EPA activities. OSHA is currently reviewing its benzene, asbestos, and standards and working on EDB, ETO, and MDA. In addition to these specific chemicals, OSHA is also developing a chemical labeling standard, standards for labortory chemicals, a cancer policy, standards for non-ionizing radiation, and is reevaluationg its respirator standards. Below is an over-view of the potential EPA offices impacted by these efforts.

IV. Mechanisms for Issue Resolution

Due to the multitude of issues and needs concerning interagency coordination with OSHA a variety of mechanisms can be used to address each agencies neeeds. In order to simplify the approach for EPA/OSHA coordination the following three options are presented:

1. Development of a Memorandum of Understanding (MOU)--Generic

In order to address all issues both current and future, a MOU could be developed laying out the policy of each agency concerning coordination and cooperation. Specific details or issues would not be addressed in the MOU, rather areas of mutual concern (e.g. health assessment) would be addressed and, as identified, issues (e.g. benzene, cadmium) would be brought to the attention of the Agencies by contact to a lead Agency Office. Upon contact to the agency a formal/informal effort would be initiated.

Pros:

a. Presents the position of EPA and OSHA concerning what will and will not be coordinated.
b. Lays the foundation for continued interagency coordination and cooperation by allowing flexibility.
c. Assigns lead responsibility in each Agency to assure continued interagency cooperation and coordination.

Cons:

a. MOU may take a long time to initiate, while both Agencies have immediate needs for coordination.
b. Specific items needing coordination may not be identified,

or identified on an ad-hoc basis. Only program offices wanting or knowing of the interagency connection would become involved.

c. Because of the ad-hoc nature of events, difficulties would be encountered in the planning and scheduling of events.

2. Development of MOU--item specific

Another approach to interagency issue resoultion would be the development of item specific MOU Each MOU would address a specific topic and outline the responsibilities of each Agency to include resources and lead office responsiblity.

Pros:

a. Formally presents the position of each agency concerning inteagency coordination and cooperation on a specific issue.
b. Limits the scope of interagency efforts to only items listed in each MOU.
c. Allows each Agency to plan interagency activities.

Cons:

a. Limited to only items listed in MOU
b. Time consuming. A MOU would have to be developed to address each issues of concern as it develops. Unnecessary delays may occur concering problems that need immediate interagency action.

3. Open lines of Communication

As a first step to breaking down the barriers to interagency coordination would be contact by the Administrator with OSHA. A letter requesting interagency coordination and cooperation could be developed either trageting generic issues or specific issues needing interagency work. Once the lines of communication were open, EPA and OSHA offices could then on an ad-hoc basis being to work together. Lead offices could be designated to act as funnels to the appropriate offices.

Pros:

a. Relatively easy to initiate.
b. Does not requres alot of EPA resources (management/staff).
c. Because of its open ended nature, could apply to almost any issue facing EPA.

Cons:

a. Presents only a one-way approach to coordination. EPA initiating action and OSHA responding ad-hoc.
b. Agencies are not accountable to each other. Informal mechanism with no resources or committment.
c. With no formal mechanism established, potential to loose focus of effort.

V. RECOMMENDED APPROACH

In order to facilitate the coordination of activities between EPA and OSHA, the Work Group is recommending that lines of communication be opened between the two agencies. A lead office within each agency should be assigned; it will have the responsibility of assuring that appropriate interagency issues are identified and efforts are made to resolve them. Within EPA, the Office of Pesticides and Toxic Substances should have the responsiblity for coordinating Agency-wide activities for each of the program offices.

Attachment C 7

I. ITEM: Interagency Coordination with the Centers for Disease Control (CDC)

The Centers for Disease Control are responsible for the protecting of public health from disease, disability and death and are charged with enhancing the health of the American people. Within CDC, the Center for Environmental Health, the Agency for Toxic Substances and Disease Registries, and the National Institute for Occupational Safety and Health are the key organizations for targeted interagency coordination efforts.

The goal of the Center for Environmental Health (CEH) is to prevent or control environmentally related health problems occurring outside the work place. CEH conducts programs designed to assist the public health community in the surveillance, investigation, analysis, prevention and control of environmentally-induced health problems such as cancer, birth defects, injuries, environmental hazards and related chronic diseases. The Center is authorized to act on behalf of the Department of Health and Human Services for health activities to be conducted under the Comprehensive Environmental Response, Compensation and Liability Act. The Center provides technical advice and assistance through a staff of Public Health Advisors assigned to EPA regional offices, to assist EPA, State, and local officials in their efforts to address toxic chemical spills and waste dumps hazards. The Center also serves as the coordinating point in the Public Health Service for reviewing Environmental Impact Statements and for radiation emergency planning.

The Agency for Toxic Substances and Disease Registry (ATSDR), is required by law (CERCLA) to carry out the health-related aspects of CERCLA. ATSDR is required to: 1) establish and maintain a national registry of serious diseases and illness and a national registry of persons exposed to toxic substances; 2) establish and maintain an inventory of literature, research and studies on the health effects of toxic substances; 3) list areas restricted to the public because of toxic substance contamination; 4) provide medical care and testing to exposed individuals, including tissue sampling, chromosomal testing and epidemiological studies; and, 5) conduct periodic surveys and screening programs to determine relationships between exposure to toxic substances and illness.

The National Institute for Occupational Safety and Health (NIOSH) is responsible for developing recommended occupational safety and health standards. This is achieved principally through developing criteria documents (by industry and chemical), conducting epidemiological investigations, laboratory research to determine the acute and subchronic toxicity of industrial chemicals, and developing and evaluating performance criteria for environmental monitoring and engineering control technology.

II. What is the issue and why is it important:

There are several reasons for bringing together the health and environmental agencies, it is a requirement under CERCLA, the need to assure the best scientific evaluation of various chemicals, and the need to obtain a government-wide consensus on a chemical's health and environmental effects. In addition, because of recent budget cuts, EPA's Office of Research and Development has a reduced capability to respond to the epidemiologic needs of the various program offices--especially those of Superfund concerning hazrdous waste sites. Because of the expertise and capabilities of CDC, coordination and the development of specific joint porjects will materially enchance EPA's ability to respond to health/environmental concerns.

Currently, EPA and CDC coordinate on an ad hoc basis. The major impact of continuing this type of coordination will be in the areas of hazardous waste sites and identifying and evaluating potentially hazardous chemicals. Ad hoc coordination may also limit the scientific evaluation of various chemicals and prohibit the development of a procedure to systematically obtain a government-wide consensus on their health and environmental effects.

III. What EPA Programs are Involved:

Because of the nature of CDC's area of expertise, the entire Agency potentially will be impacted by EPA/CDC coordination. The Offices of Pesticides and Toxic Substances, Emergency and Remedial Response and Research and Development, as well as the EPA Regional Offices all potentially play an important role in EPA's need for coordination with the Centers for Disease Control.

IV. Mechanisms for Resolution:

Due to the variety of activities needing coordination on both the Regional and Headquarters levels, many coordinating mechanisms can be used. Such activities could include: 1) information exchange concerning research activities; 2) waste site-specific analyses; 3) peer review of health studies; 4) development of joint program evaluation techniques; 5) health data base development and data sharing; 6) development of monitoring methods, model validation and quality assurance protocols; 7) cross agency cooperation in NIOSH Criteria Document review and EPA risk assessment analysis; and, 8) development of joint broad-brush health studies.

Mechanisms could be developed that are either item specific or generic and could take the form of MOU's, IAG's, or formal or informal working groups on the Regional or Headquarters level.

Because of the complexity of issues and the large number of participants in the effort, initial efforts should be focused on scoping the problem and issues that need to be addressed.

V. Recommended Option.

In order to identify all the issues/problems and players a meeting of all program offices potentially involved should be held to review the current extent of EPA's relationship with CDC and to evaluate that relationship within the context of the Agency's overall needs for the kind of support CDC can offer. In addition, a lead office (ORD/CCS/OPRM/OPTS) should be assigned to open discussions with CDC concerning identified areas of mutual interest. Based upon agreements reached, the Agency should consider developing a formal MOU. The MOU should incorporate the basic principles that will govern cooperative activities and set forth broad areas within which cooperation will occur. The MOU would then form the basis for Agency-wide or program-specific interagency tasks to implement particular joint projects.

Lead Agency Office responsibility needs to be assigned as well as program office leads. It is recommended that the Chemical Coordination Staff be assigned the Agency lead and that, as specific items arise, program office leads be designated. Due to the complexity of the developmental work and the need for major planning and negotiations efforts, moderate to high resources would be required for CCS during the next year, with moderate resources required for the program offices. Following the first year, CCS resource needs will be reduced somewhat while program office resources will be project(s) dependent.

Attachment C 8

I. ITEM: Regulatory Coordination/Information Exchange--Biotechnology

In its broadest meaning, biotechnology is the application of biological science towards technological ends, towards the production of chemicals or life forms which have potential for commercial uses. It includes a diverse spectrum of activities, ranging from simple and clearly safe techniques such as the use of yeast to make alcoholic beverages and bread, to the complex recent technologies of gene- splicing to create modified life forms and biochemicals. For regulatory purposes, primary attention is turned to a more focussed definition--applications of genetic engineering which involve modified life forms and substances which do not occur in nature. This definition accounts for the primary risk which such substances pose, namely, that they are "exotic" and, therefore, are less likely to find natural enemies in human bodies or the environment.

II. What is the issue and why is it important?

Scientists and government regulators have serious concerns over the prospect of modified life forms being sold, distributed and used in the open environment, where they would be particularly difficult to control. Scientific uncertainties support the need for government review and evaluation prior to commercialization. For example, one unknown is whether genetically engineered organisms, released to the environment, can be infective or toxic to humans or other organisms. Another unknown is the possibility that a genetically modified organism could disrupt existing ecosystems. At this time, we have no clues as to the probability of such events with genetically engineered microbes, nor how to estimate or minimize the risk if it does exist. Of primary importance is the fact that there is uncertainty with regard to which if any areas of recombinant DNA technology will require regulatory controls.

Because EPA's role, principally through FIFRA, TSCA and ORD, will be complementing the roles of other regulatory and research programs, significant effort towards intergovernment coordination and planning will be required in order to avoid overlapping responsibilities and to ensure scientific data are available and consistent evaluations occur.

A serious technical problem, shared with other government agencies which are overseeing this area, is that there are almost no accepted methodologies for evaluating the safety of genetically engineered products. The risk assessment tools and data used for inanimate chemical substances will not apply in the case of organisms. It may take several years to have adequate testing methods and risk analysis techniques in any of these areas. At the same time, while we expect broad commericalization of these products to be a couple years in the future, we have begun to see the first of the potential biotech organisms in the TSCA premanufacture review program.

III. What other Agencies are involved:

EPA: OPTS (OTS, OPP), ORD, ODW, OWRS, OSW, OGC

Other federal agencies working on biotechnology issues include FDA, USDA, NIH, OSHA (regulations); NIOSH, NAS, OTA (assessments, legal and economic issues).

IV. Mechanisms for Resolution:

a. One option would be a broad umbrella group established for coordination of assessment, guidelines and regulatory coordination be established through a Memorandum of Understanding, special charter, or Executive Order.

An IAG to establish and support the expert group may be needed to funnel special funds for research needs such as occasional characterization of various segments of biotech. e.g., viruses, development of testing and assessment methodologies.

b. Separate MOUs or cooperative agreements could be established to separate the needed tasks, especially one for regulatory initiatives and one for scientific research, and one for risk assessment. These generally involve different agencies.

V. Recommended options:

Recommend that an umbrella expert group be established to speak with one voice for the Federal government.

EXTERNAL STAFF DRAFT
03-23-83

Proposed Process for Evaluation and Control of Toxic Air Pollutants

DRAFT

This is an EPA staff draft that is being circulated for external comment. It has not been approved by Agency management.

Strategies and Air Standards Division
Office of Air Quality Planning and Standards

PROPOSED PROCESS FOR EVALUATION AND CONTROL OF TOXIC AIR POLLUTANTS

I. INTRODUCTION

~~The purpose of this paper is to describe a process which will allow EPA to discharge more effectively its responsibilities under the Clean Air Act (CAA) for the evaluation and control of toxic air pollutants.~~

The basic goal of the Clean Air Act is to protect and enhance the quality of the Nation's air resources so as to promote the public health and welfare. Significant progress toward this goal has been made in developing scientific criteria and setting, reviewing, and implementing national ambient air quality standards. Pollutants for which ambient air standards are set* are those which may reasonably be anticipated to endanger public health or welfare and whose presence results from numerous and diverse emission sources. These often are called "criteria" pollutants because of the requirement under the CAA (section 108) to develop comprehensive criteria documents for each pollutant.

The CAA also provides for the control of non-criteria air pollutants which may adversely affect public health but for which an ambient air standard is not appropriate. These pollutants are referred to in this paper as toxic air pollutants. The principal authority under the CAA for control of toxic air pollutants is section 112, entitled National Emission Standards for Hazardous Air Pollutants. Section 112 defines "hazardous pollutant" as an "air pollutant to which no ambient air quality standard is applicable and which in the judgment of the Administrator causes, or contributes to, air pollution which may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." Section 111 (New Source Performance Standards) also provides for control of pollutants which adversely affect human health or welfare ~~but are not "hazardous" as defined under section 112~~.

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*Particulate matter, sulfur oxides, nitrogen dioxide, carbon monoxide, ozone, lead

A number of substances have been identified which have a potential for adversely affecting human health and therefore are candidates for control. To date, the Agency has focused primarily on the use of section 112 to effect this control. Seven pollutants* have been listed as hazardous under section 112 and to date emission regulations have been promulgated to control four. Regulations for the remaining three hazardous air pollutants are in preparation. One additional non-criteria pollutant (sulfuric acid mist) has been regulated under section 111 for health reasons.

II. BACKGROUND

A. Extent of the Toxic Air Pollutant Problem

The general reduction in emissions of criteria pollutants as a result of State Implementation Plans and the New Source Performance Standards has significantly reduced emissions of toxic air pollutants. This results because many toxic air pollutants are emitted either as particulate matter or volatile organic compounds (precursors to ozone), both of which are controlled to meet ambient air quality standards. Regulations promulgated under the Solid Waste Disposal Act for hazardous waste incinerators also significantly reduce emissions of toxic air pollutants.

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*Asbestos, beryllium, mercury, vinyl chloride, benzene, radionuclides, inorganic arsenic.

In evaluating the extent of the toxic air pollutant problem, the Agency must determine whether pollutants emitted by individual sources or combinations of sources pose unreasonable risks to public health. Available exposure data and health effects information and the scientific interpretation of this information do not allow a clear-cut, absolute determination of the extent of the health risks associated with toxic air pollutants for the nation as a whole. While there do not seem to be significant widespread risks, clearly there are individuals who may be exposed to potentially toxic air pollutants emitted from uncontrolled or partially controlled sources. As with other toxic chemicals, there is always the concern that exposures even at low levels may result in chronic adverse effects which may not become evident for years.

The magnitude of the toxic air pollutant problem in terms of number of pollutants and sources of emissions is difficult to determine with precision. A 1976 survey of the organic chemical industry identified over six hundred commercially important chemicals, of which about 50 were identified as potential toxic air pollutants requiring a more detailed assessment. Many source categories other than those in the organic chemical industry also may be potentially significant emitters of toxic air pollutants. These sources include mining, smelting, refining, manufacture and end-use of minerals and other inorganic chemicals; combustion; petroleum refining, distribution, and storage; solvent usage and waste disposal; mining, processing, use and disposal of radioactive substances and radioactive by-products; and various sources of non-toxic emissions which are chemically transformed into toxic air pollutants in the atmosphere.

The Agency is concerned with all human health effects, but cancer is of special concern because of the high incidence of mortality associated with it. The total number of cancer deaths is well known,* but the contribution of air pollution to this total is uncertain. Ambient air pollutants generally are thought to rank well below smoking, occupational exposure, and diet as an incremental cause of cancer.

B. Statutes Applicable to Air Toxics Control

1. The Clean Air Act

The Clean Air Act requires EPA to identify and regulate air pollutants that adversely affect public health. Three alternatives are provided for dealing with pollutants from stationary sources:

a. List the pollutant under section 108, set national ambient air quality standards under section 109, and require States to control sources through State Implementation Plans under section 110. Pollutants regulated through this mechanism are those which may reasonably be anticipated to endanger public health or welfare and which are emitted by numerous and diverse mobile or stationary sources.

b. Establish new source performance standards (NSPS) under section 111 for specific new or modified sources which "cause or contribute significantly to air pollution which may reasonably be anticipated to endanger public health and welfare." Most, but not all, NSPS are for pollutants subject to ambient air standards. In cases where an NSPS affects a pollutant not subject to an ambient air standard or not designated as a hazardous pollutant, States must regulate existing sources (of the type covered by the NSPS) as required by section 111(d).

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*National Center for Health Statistics, Monthly Vital Statistics Reports.

c. List the pollutant as hazardous under section 112 and establish emission standards for both new and existing sources for this pollutant. Pollutants regulated under section 112 are those which "may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." The emission standards established by EPA are to protect the public health with an "ample margin of safety."

Section 112 has been considered in the past to be the primary statutory mechanism for controlling toxic air pollutants. However, a number of issues have complicated its implementation. For example, issues not resolved clearly to date include the establishment of toxicity to humans based on animal and/or human data, extrapolation from high-dose animal tests to low-dose human exposure at actual ambient concentrations, and identification of the appropriate level of emission controls for pollutants for which health effects thresholds have not been demonstrated adequately.

not rel

The appropriate use under section 112 of quantitative health risk estimates has been a key issue due to the uncertainty inherent in such estimates. To develop these estimates, it often is necessary to extrapolate toxic effects found at relatively high levels for animals or from exposures to humans in a workplace to very low levels typically found in ambient community exposures. On the exposure side, there is uncertainty about emission rates, dispersion patterns, short-term and long-term movement (migration) of people, and indoor versus outdoor toxic air pollutant concentration patterns. Monitoring data are limited and difficult to obtain for use in exposure assessment. All of these factors make it difficult to know the absolute magnitude of the risk to human health based on the available data.

[illegible] 5/12

Another key issue that has been encountered in implementing section 112 is whether protection should focus on individual or aggregate risk. If we can place any credence on risk estimates, results to date indicate that the total cancer incidence (number of cases per year) associated with exposure to toxic air pollutants, even on a nationwide basis, is small for a given pollutant, although individual risks for a limited number of people living close to emission sources often are estimated to be relatively high.

~~Finally, section 112 lacks an explicit economic test. Thus, a literal interpretation of section 112 would not preclude zero emissions requirements for non-threshold pollutants.~~

Principally because the Clean Air Act does not provide specific guidance on these issues, it has been difficult to establish definitive criteria for the evaluation and control of toxic air pollutants under section 112. As such, EPA has been reluctant to implement actions under this section without a clear indication that the costs of control are not unreasonably disproportionate to the health benefits when compared to other regulated pollutants. Administrative, legal, and legislative requirements, coupled with a lack of acceptable criteria for decision-making, have resulted in an evaluation process that can take from 5 to 7 years before regulations are in place. As a result of this lengthy process, while many potential pollutants are under evaluation, few have reached the final decision stage.

2. Toxic Substances Control Act

The Toxic Substances Control Act authorizes EPA to obtain information from industry on the production, use, health effects, and other matters concerning chemical substances and mixtures. If warranted after considering the costs, risks, and benefits of a substance, EPA may regulate its manufacture, processing, distribution in commerce, use, and disposal. A toxic substance as defined under this Act is any chemical substance or mixture that may present an unreasonable risk of injury to health and the environment. This definition can include pollutants in any environmental medium.

The key sections of the Toxic Substances Control Act are: section 4, testing of chemical substances and mixtures; section 5, manufacturing and processing notices (premanufacturing notification); section 6, regulation of hazardous chemical substances and mixtures; section 7, imminent hazards; and section 8, reporting and retention of information. Section 9 further states that where other Federal laws are applicable they should be used unless the Administrator determines that it is in the public interest to use this Act.

3. Solid Waste Disposal Act

The Solid Waste Disposal Act (also known as the Resource Conservation and Recovery Act) authorizes EPA to identify hazardous wastes, and to develop standards for generators, transporters, treaters, storers and disposers of hazardous waste. A hazardous waste is defined under this Act as any waste or combination of wastes that, because of its quantity, concentration, or physical, chemical, or infectious characteristics, may (a) cause or significantly contribute to an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness, or (b) pose a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported, disposed of, or otherwise managed. Hazardous wastes can result in the release of pollutants to any environmental medium.

The key sections of the Solid Waste Disposal Act are: section 3001, identification and listing of hazardous wastes; section 3002, standards applicable to generators of hazardous waste; section 3003, standards applicable to transporters of hazardous waste; section 3004, standards applicable to owners and operators of hazardous waste treatment storage and disposal facilities; and section 7003, imminent hazard.

4. Comprehensive Environmental Response, Compensation and Liability Act

The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (also known as Superfund) establishes broad Federal authority to deal with releases or threats of releases of hazardous substances from vessels and facilities that may present substantial danger to public health, welfare, and the environment. The Act designates an initial hazardous substances list (section 101(14)) and allows for additions to the list by EPA's Administrator under section 102. Under section 103 of the Act, the National Response Center must be notified when a reportable quantity release occurs. Once notification is received, the government may take remedial or removal action if: (1) there is a release or substantial threat of a release of a hazardous substance or (2) whenever there is a release of other pollutants or contaminants that may present a substantial danger to public health, welfare, and the environment. Response activities are to be taken in accordance with the National Contingency Plan.

The Act provides that owners and operators of vessels or facilities who release a hazardous substance, and certain other persons who contribute to the hazardous substance incident, are liable for response costs incurred and for damages to natural resources. Failure to notify poses criminal liability. There are limitations to liability established in the Act, as well as limited defenses to the liability.

The Act incorporates, by reference, five lists of hazardous substances developed under other environmental statutes into the definition of hazardous substances. These lists are: hazardous air pollutants under section 112 of the Clean Air Act; hazardous substances under section 311 of the Clean Water Act; toxic pollutants under the Clean Water Act section 307(a); hazardous wastes under section 3001 of the Solid Waste Disposal Act (excludes those wastes omitted by statute); and imminently hazardous chemical substances or mixtures for which the EPA Administrator has taken action under section 7 of the Toxic Substances Control Act.

5. Other Statutes

Under some circumstances, toxic air pollutants also may be controlled effectively using the Federal Insecticide, Fungicide and Rodenticide Act, the Clean Water Act, or the Safe Drinking Water Act. The applicability of these statutes depends upon the physical and chemical properties of the pollutant and the medium through which the air pollutant is released to the environment.

III. OVERVIEW

This overview describes the key elements of the proposed evaluation and control process for toxic air pollutants. This process is intended to allow EPA to discharge more effectively its responsibilities on toxic air pollutants. Figure 1 shows the overall process, the flow of information, and the major decision points. The process is summarized below and described in more detail in Sections IV through VII. In order to put the evaluation and control process in perspective, times to accomplish each of the major steps are estimated in Table 1.

The proposed toxic air pollutant evaluation and control process differs principally from the past process in the following ways:

1. While the evaluation and control process continues to rely on section 112 as the primary control alternative, it clarifies the use of other sections of the Clean Air Act and other Federal and non-Federal control alternatives to provide greater flexibility in implementing a control strategy appropriate to the magnitude and type of perceived health risk.

2. The evaluation and control process formally provides, where appropriate, for the use of State and local regulatory authorities, as well as section 111, as control options, and the important role of State and local regulatory agencies in controlling air toxics is more clearly defined.

3. The public and the Science Advisory Board are involved to a greater degree to assure continued awareness of the program and to provide more opportunity for review and comment.

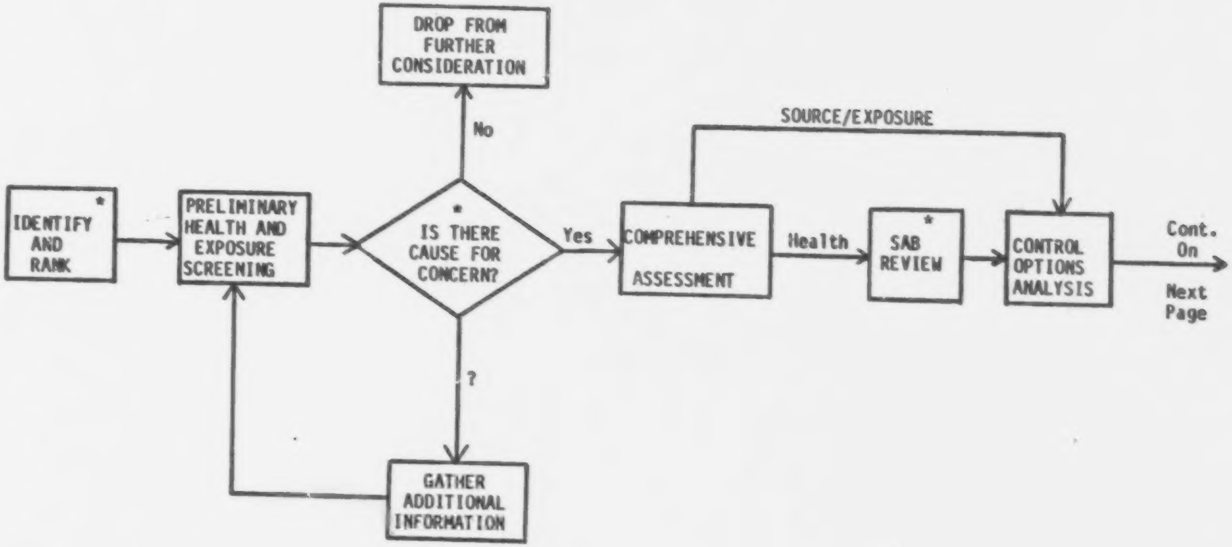
A. Key Elements

The proposed toxic air pollutant evaluation and control process is designed to continue use of section 112 to control air toxics which are clearly "hazardous" in the sense that exposure at ambient levels may

FIGURE 1

TOXIC AIR POLLUTANT EVALUATION AND CONTROL PROCESS

EVALUATION PHASE

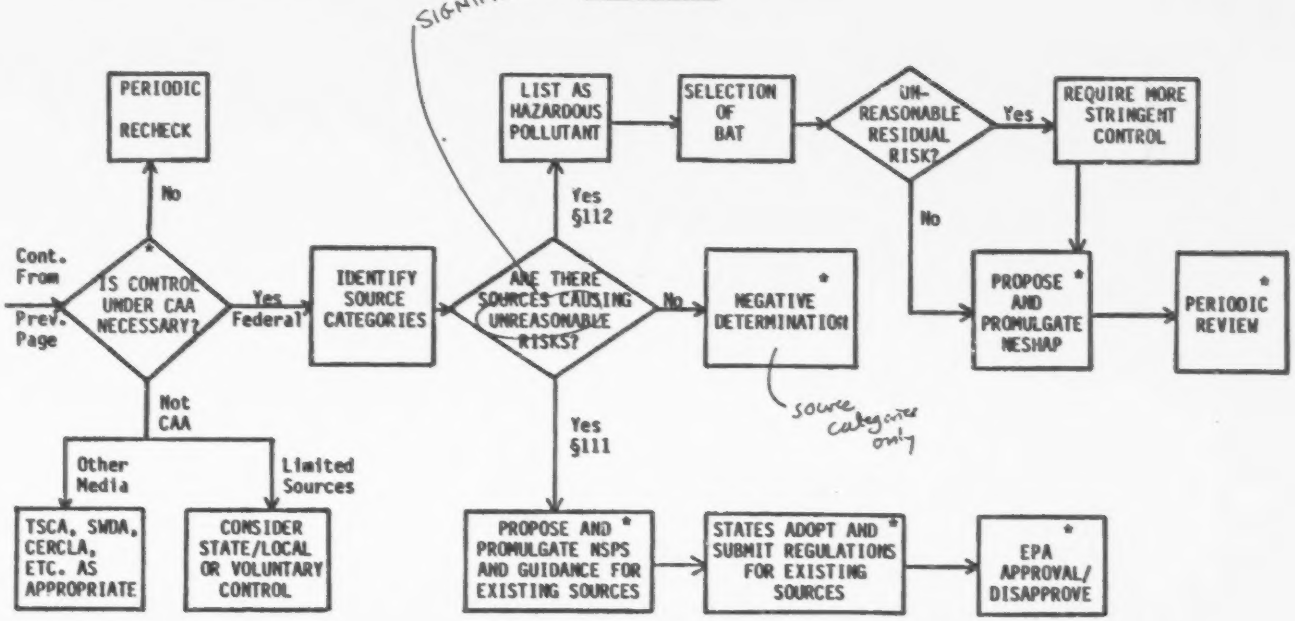

DROP FROM FURTHER CONSIDERATION
No
SOURCE/EXPOSURE
IDENTIFY AND RANK *
PRELIMINARY HEALTH AND EXPOSURE SCREENING
* IS THERE CAUSE FOR CONCERN?
Yes
COMPREHENSIVE ASSESSMENT
Health
* SAB REVIEW
CONTROL OPTIONS ANALYSIS
Cont. On Next Page
?
GATHER ADDITIONAL INFORMATION

*Opportunity for public comment.

FIGURE 1

TOXIC AIR POLLUTANT EVALUATION AND CONTROL PROCESS

CONTROL PHASE


Cont. From Prev. Page
IS CONTROL UNDER CAA NECESSARY? *
No
PERIODIC RECHECK
Yes Federal
IDENTIFY SOURCE CATEGORIES
Not CAA
Other Media
TSCA, SWDA, CERCLA, ETC. AS APPROPRIATE
Limited Sources
CONSIDER STATE/LOCAL OR VOLUNTARY CONTROL
ARE THERE SOURCES CAUSING UNREASONABLE RISKS?
SIGNIFICANT
Yes §112
LIST AS HAZARDOUS POLLUTANT
SELECTION OF BAT
UN-REASONABLE RESIDUAL RISK?
Yes
REQUIRE MORE STRINGENT CONTROL
No
PROPOSE AND PROMULGATE NESHAP *
PERIODIC REVIEW *
No
NEGATIVE DETERMINATION *
source categories only
Yes §111
PROPOSE AND PROMULGATE NSPS AND GUIDANCE FOR EXISTING SOURCES *
STATES ADOPT AND SUBMIT REGULATIONS FOR EXISTING SOURCES *
EPA APPROVAL/ DISAPPROVE *

* Opportunity for public comment.

Table 1

Estimated Times to Complete Process Steps

	Process Step	Estimated Time
1.	Identify Candidates and Rank	Once/year 1-3 mo.
2.	Screen Health and Exposure	Once/year 3-6 mo.
3.	Comprehensive Assessment	1-2 years
4.	SAB Review	3-6 mo.
5.	Control Options	3-6 mo.
6.	Publish Decision	3-6 mo.
7.	Identify Sources and Set Priorities	6-9 mo.
8.	Develop and Promulgate Standards	2-3 yrs.
	Total Time Range	4 yrs.-7 yrs.

reasonably be anticipated to result in an increase in mortality and illness. However, where there is a high degree of uncertainty as to the magnitude of the relative health risks or where the use of other legislative authorities or nonregulatory control options is obvious, the evaluation and control process provides the flexibility to use these alternatives. In order to ensure that public health concerns are dealt with in the most timely and effective manner, and in order to optimize resource use, the process provides for several levels of increasingly detailed analysis. At each level of analysis a decision is made to ensure that the pollutants which receive the most detailed and resource intensive analysis are the most likely candidates for control. The public and the Science Advisory Board will be briefed periodically on the status of these efforts.

B. The Evaluation and Control Process

Over the past several years, EPA has undertaken the systematic identification, ranking, screening and assessment of a number of candidate toxic air pollutants. Additional candidate pollutants will be identified periodically and then ranked using available health and source information. The highest ranking candidates will be screened to assess their potential for relative health risks at ambient exposures. One of the following actions then will be taken: (1) Where information is not adequate to determine the appropriate next step, that information will be gathered. (2) Where public health concerns clearly are not unreasonable, the candidate will be dropped from further active consideration. (3) Where the candidate clearly poses a health concern but can more appropriately be dealt with using a statute other than the Clean Air Act, jurisdiction will be transferred to the appropriate program. (4) Where information indicates a cause for concern, the candidate will undergo comprehensive health and exposure assessment.

This comprehensive assessment is a detailed and resource intensive step that leads to formal Science Advisory Board review in public meetings and closure by the Board when it is satisfied that the document is scientifically sound and adequately represents the latest scientific knowledge. Following closure, all available information along with an analysis of feasible control options is provided to the Administrator.

Several responses are possible at this point in the process: (1) Where public health needs do not indicate that further action is required at this time, control activities will be halted and the candidate placed in a category for periodic reassessment. (2) Where State/local, voluntary or other non-Federal actions are appropriate, EPA will obtain agreements to act from the appropriate party and will provide necessary technical or support information. (3) Where Federal regulatory action is appropriate, the necessary legislative authority will be implemented.

Under the Clean Air Act, the process for developing standards begins with identification and evaluation of the appropriateness of Federal regulation of important source categories. Source categories of minimal concern will be identified as such in proposals not to regulate. Appropriate standards will be developed for source categories of concern. The basis for control will be best available technology (considering cost and other factors), with additional control possible under section 112 if the risk remaining after application of best available technology is determined to be unreasonable.

C. Resolution of Key Issues

The inability to resolve several key issues has contributed to the slow past progress in dealing with toxic air pollutants under the Clean Air

Act. While this process does not provide detailed resolution to these issues, the following summarizes generally how each will be approached:

1. How are potential human carcinogens evaluated? Given that the quality and extent of data vary greatly, EPA will evaluate the nature of the evidence in each case using a "strength or weight of evidence" approach. In the absence of sound scientific evidence to the contrary, prudent public health policy requires that where the strength of evidence is sufficient to find that a pollutant may be a human carcinogen for regulatory purposes, this substance should be assumed to pose some finite level of risk at any ambient exposure.

2. How are relative risk estimates used? To the extent possible, risk estimates (which are derived from quantitative mathematical models) will be used to compare air pollutants, sources, and alternative control options. However, it is recognized that there are uncertainties in the quantification of risks, particularly at low levels of exposure. Hence, the use of quantitative risk information as the prime factor in specific regulatory decision-making necessarily will be limited. The "strength of evidence" consideration always must take priority over the use and application of numerical estimates of risk obtained from various mathematical models.

3. What level of control is appropriate control under section 112? Best available technology (considering costs and other factors) will be required as a minimum. Additional control may be required, after consideration of the health risks, costs, benefits, and other factors, to eliminate unreasonable residual risks.

IV. ASSESSMENT OF POTENTIALLY TOXIC AIR POLLUTANTS

This section details the process by which EPA will assess potentially toxic air pollutants.

A. Identification of Candidates for Evaluation

Since initial identification, ranking and screening efforts on non-criteria air pollutants, EPA has centered its assessment work on a number of candidates. EPA will continue to develop and periodically update its list of potentially toxic air pollutants based on information from sources such as appropriate data bases, the scientific literature, monitoring studies, and biological assays of substances found in the ambient air and source emissions. Available data from other Federal agencies, State or other public testing or regulatory authorities, private research groups, and producing or consuming industries also will be considered. A preliminary ranking of pollutants will be developed based on health effects, production volume, volatility, bioaccumulation potential, and existing standards.

B. Screening

e.g. toluene screened almost solely because of high exposure

Substances of most concern will be selected periodically from the identification process. These will be screened by conducting a preliminary health and/or exposure evaluation. Based on the results, a decision will be made on the preparation of more comprehensive assessments.

The health evaluation screening consists of reviewing published data to make an initial determination of the most important potential health effects associated with the substance. The ambient exposure screening will be based on readily available information, including the number and types of sources emitting the substance in geographical areas where people may be exposed, the estimated quantity of emissions, ambient concentrations which may have been reported, and the number of people living near emitting sources or in the vicinity of ambient measurement sites.

C. Responses to Screening

This section discusses possible responses resulting from the screening process. The public and the Science Advisory Board will be advised periodically of the status of these responses.

1. Comprehensive Assessment

This step is substantially the same as that now practiced and entails expanded evaluation of exposure potential, an expanded assessment of potential sources along with possible impacts of control of these sources, and a comprehensive assessment of all important health effects. The comprehensive health assessment document is submitted to the EPA Science Advisory Board (SAB) for an evaluation as to scientific adequacy, and comments are solicited from the public for review by both EPA and the SAB.

2. Obtain Additional Information

Additional information may be needed before a judgment can be made on the screening results. The process for gathering this information could encompass a variety of approaches. EPA initially will rely on information resulting from hazardous substance data bases (such as that maintained in the Superfund program), technical studies, EPA monitoring and research requests to industrial and other sources, requests to other Federal programs (e.g., the National Toxicology Program), and requests to State and local agencies. If these do not provide the necessary information, provisions of section 114 of the Clean Air Act, applicable sections (4, 8a and 8d) of the Toxic Substances Control Act, and information gathering authorities of other environmental legislation may be used to obtain more data.

3. No Further Action

This response will be taken if available information clearly indicates that potential public health risks from exposure to ambient concentrations are not unreasonable.

V. RESPONSE TO COMPREHENSIVE ASSESSMENT

This section describes the decision-making process that follows assessment. A control options analysis first will be performed and made available to the public for review and comment. Following consideration of comments, EPA will determine the action to be taken and announce its decision in the Federal Register.

In the control options analysis, all relevant data are summarized and feasible control options identified to provide the information necessary to make informed decisions concerning the need for and extent of control. Appropriate information considered at this stage includes applicable legislative authorities, health and environmental impacts, sources of emissions, control technologies and estimated costs to improve those controls, estimated population exposure and other data. Control options for consideration include the following (note that these options are not mutually exclusive):

A. Federal Regulation

Where the potential public health impact is sufficient to warrant a national program, EPA first will consider the use of the Clean Air Act, including sections 108-110, 111 and 112. In some instances the use of another statute will be more appropriate to ensure the most timely and effective reduction in public health risk. In those cases, the control process will be consistent with the mandate of those laws, and may differ from that of the Clean Air Act process described here. Other statutes include the Toxic Substances Control Act, the Solid Waste Disposal Act, and the Comprehensive Environmental Response, Compensation and Liability Act. Criteria for selection of appropriate regulatory responses are discussed in Section VII.

B. State/local Actions

Action on the State or local level may be appropriate where the number of sources is small or geographically localized, where state regulations may be more effective than Federal regulations, and where the resulting controls are verifiable by EPA. Where State/local regulation is appropriate, EPA will obtain agreements to act from the appropriate Agency and then will provide technical and scientific support to the State and local agencies in such cases to ensure that actions are scientifically supportable.

C. Information Dissemination

In some instances, principally where the relative risks to the public health are not unreasonable but where there may be some cause for concern, dissemination of information on the possible health risks and measures for reducing those risks will be indicated. For example, where available information indicates that infrequent emissions or incidents are the dominant cause for concern, EPA will prepare information on the concerns and appropriate control measures and provide it to State and local agencies, industry and the public.

D. Other Non-regulatory Actions

In some instances, it may be much more timely and effective to seek voluntary control. Such control might be appropriate when there are a limited number of sources, all of which agree to reduce emissions to levels sufficient to protect the public health. Voluntary measures will be recommended only where health protective and verifiable by EPA. Enforceable compliance agreements will be strongly encouraged as an integral part of this option.

E. No Further EPA Action

This option is indicated when available information clearly indicates that public health needs will not be served by further action.

VI. ESTABLISHMENT OF FEDERAL REGULATIONS UNDER THE CLEAN AIR ACT

This section describes the process to be used where Federal regulation under the Clean Air Act is warranted. A brief discussion of other environmental legislation is contained in Section VII.

A. Identification of Source Categories

Preliminary to determining the sources for which Federal regulation is appropriate, a screening study is undertaken to identify the various types of emitting sources and to indicate which types may be significant enough to warrant further study toward the possible development of regulations. Among the screening factors considered are the magnitude of emissions, estimates of ambient concentrations based on model plant assumptions, the number and distribution of the sources, and the potential applicability of stationary source regulations.

B. Evaluation of Appropriateness of Federal Regulation

The significant types of emitting sources are studied in substantial detail to determine those for which the proposal of Federal regulations

Per CMA comment

is appropriate and those for which the proposal of Federal regulations is not. For the former, proposed regulations are published in the Federal Register; for the latter, proposals not to regulate are published. Public comments are solicited in both cases, and final Agency determinations (which are judicially reviewable) are published after consideration of the comments. Determinations of whether to propose regulations are based on consideration of a number of factors, including the potential for improving emission control by requiring the use of the best available technology, the practicability of producing effective and enforceable Federal regulations, the multimedia impacts of air pollution control, the number, distribution, and operational life of the sources, rough estimates of the potential for adverse health impacts, and the impact of Federal regulations on the economic condition of the industry.

C. Priorities

Whenever resources do not permit the simultaneous development of regulations for all of the sources for which Federal regulation has been determined to be appropriate, higher priorities will be given to those types of sources for which the adverse health impacts appear to be the more severe and for which the potential for improved emission control is higher.

D. Development of Emission Standards

Standards development will follow those procedures currently utilized by EPA to ensure technical currency and adequacy. These procedures include: (1) encouragement of public input at all stages of the development process, (2) comprehensive technical and economic impact analysis, (3) review by the National Air Pollution Control Techniques Advisory Committee, and (4) proposal and promulgation in accordance with the requirements of the Clean Air and the Administrative Procedures Acts. almost always inapplicable.

E. Periodic Review

EPA will review periodically the information used to support its actions and recommendations to ensure continued protection of public health.

VII. CRITERIA FOR SELECTION OF APPROPRIATE REGULATORY RESPONSE

A. Section 112 (Clean Air Act)

Under section 112 the pollutant is listed as hazardous and emission standards are established for both new and existing sources for that pollutant. Pollutants regulated under this section are those which "may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." The emission standards established by EPA are to protect the public health with an "ample margin of safety." ~~No specific economic tests are provided~~ in ~~section 112.~~

EPA will conduct an evaluation of the health evidence and submit an evaluation report to the Science Advisory Board for review as part of the comprehensive health assessment document. One system for classifying carcinogenicity data for which there is growing scientific support is the International Agency for Research on Cancer criteria for determining whether a compound is likely to be a human or animal carcinogen. Under this system, pollutants which are classed as having "sufficient" evidence of carcinogenicity in humans or animals would be considered as meeting the health test for regulation under section 112. An additional criterion for listing and regulation under section 112 will be evidence of significant human exposure. Other factors to be considered include the technical and administrative feasibility of implementing improved control.

what about significance/ unreasonableness of risk - for pollutant/ for source category ?

For sources that emit toxic air pollutants for which there is no evidence on which to base a threshold, emissions should be limited as a minimum to levels corresponding to the use of best available technology (BAT). This is defined as follows:

1. For new sources, best available technology is that technology which, in the judgment of the Administrator, is the best system of continuous emission reduction which (taking into consideration the cost of achieving such emission reduction, any nonair quality health and environmental impact and energy requirements) the Administrator determines has been adequately demonstrated.

2. For existing sources, best available technology also takes into account the technological and economic impacts associated with retrofits needed to apply control technology options.

Sources in a regulated source category also will be required to limit their emissions by whatever additional amount (additional to BAT) that is necessary, in the Administrator's judgment, to eliminate unreasonable residual risks to public health associated with exposures caused by these emissions.

The principal emphasis in determining any additional level of control that may be required to eliminate unreasonable residual risks remaining after the application of BAT will be on public health concern. Where available, factors considered in the judgment may include:

1. the range of relative health risks to exposed populations caused by exposures that may be encountered after controls are put in place;

2. the economic impacts, technical feasibility, and benefits of control measures beyond BAT;

3. readily identifiable benefits of the substance or the activity producing the risk; and

4. the availability of substitutes and possible health effects resulting from the increased use of substitutes.

For pollutants with serious toxic effects, but where an effects threshold can be reasonably determined, controls should be established at a level that assures that any exposed individuals are protected with an ample margin of safety.

why so protective. probably no legally required

B. Section 111 (Clean Air Act)

Under section 111, new source performance standards (NSPS) are established for specific new or modified sources which "cause or contribute significantly to air pollution which may reasonably be anticipated to endanger public health and welfare." Most, but not all, NSPS are for pollutants which are subject to ambient air standards. In cases where an NSPS affects a pollutant which is not subject to an ambient air standard, States must regulate existing sources (of the type covered by the NSPS), as required by section 111(d).

The decision to use section 111 will depend upon several factors. These include: the magnitude of population exposures; the likelihood that control beyond best available levels might be needed to protect public health; the number of sources and their locations; and, for potential carcinogens, the cancer potency and the weight of the evidence.

The NSPS established by EPA for new and modified sources will be based on criteria exactly as that described for BAT above (i.e., the best technological system of continuous emission reduction that has been adequately demonstrated considering economic, nonair quality health and environmental impact and energy requirements). States must require existing sources of the type covered by the NSPS to meet regulations reflecting BAT for existing sources. EPA will issue guidelines reflecting BAT for such existing sources. For health-based pollutants, regulations adopted by the States ordinarily must be at least as stringent as the guidelines prescribed by EPA unless economic factors, physical limitations or consideration of the remaining useful life of existing sources make application of less stringent regulations significantly more reasonable.

C. Sections 108-110 (Clean Air Act)

In some instances, a substance may be found to cause non-carcinogenic toxic effects that do not meet the "hazardous" criteria and for which emissions to the air result from numerous and diverse sources. If so, establishment of an ambient air quality standard under sections 108-110 will be considered. Promulgation of such a standard would set in motion development of State Implementation Plans for the pollutant.

D. Section 6 (Toxic Substances Control Act)

Section 6 of the Toxic Substances Control Act provides one of the most potentially useful alternatives for control of toxic air pollutants. EPA may use this section where there is reasonable basis to conclude that manufacture, processing, distribution in commerce, use, or disposal of chemical substances or mixtures presents or will represent an unreasonable risk of injury to health or the environment, and the administrative determination is made that regulation under the Clean Air Act is inappropriate or potentially ineffective. Under this Act, EPA balances the risks with the benefits of the activity and determines the most reasonable and prudent means for achieving the necessary control. This can include prohibition or limitation on manufacturing, processing or distribution in commerce, process change, requirement for special warnings or instructions, production and retention of records, prohibition of disposal methods, and requirement for notification of risk of injury. The decision to use section 6 and the method of implementation will be made only after consultation and agreement between the applicable program offices.

E. Other Environmental Legislation

In some instances, when a candidate clearly poses health concerns but the control options analysis shows that the candidate can be dealt with in a more timely and effective manner through other media programs, the appropriate environmental legislation will be utilized. The decision to utilize other legislation will be made after consultation with the program office responsible for implementing the legislative authority deemed most appropriate. If appropriate, jurisdiction will be transferred to the applicable program office.

STAPPA - 4/20

Unhappy w/ BAT - prefer LAER - don't like NSPS's - toxics should be tougher
want us to subcategorize more carefully for toxics
- use more factors - e.g. location, exposed population - than for NSPS

Goodwin: One wants BAT for even threshold pollutants - by legislation, if necessary (N.B. SIP/111(d) available)

Don't tie ourselves to "continuous" systems

Prefer 111/111(d) - because then states can consider retrofit costs case-by-case, instead of allowing these costs to degrade whole std. for existing sources

Rz - also think 111 inappropriate, because if it meets 111 criteria, it meets 112 also

Also EPA has been reluctant to share toxics information, for fear that states would then regulate

Can "bio-accumulation" be addressed in this policy?

Dr. Bernard H. [illegible]

1019 W [illegible]

EPA Planned to Alter Carter Policy on Cancer

Anne Gorsuch Burford departed official Washington months ago, but the memos she wrote as director of the Environmental Protection Agency (EPA) live on in an afterlife of their own. A particularly fascinating document turned up recently on Capitol Hill, where hundreds of EPA papers came to rest during last spring's congressional investigations. This one belongs to a series of papers entitled "EPA Emergent Issues Reports." Dated 29 April 1982, it is addressed to Craig Fuller, secretary to the Cabinet, and signed by Gorsuch.

Like others in the "emergent issues" series, it attempts to alert the White House to political troubles that may arise out of decisions made at EPA. It covers a score of topics. One of the most interesting—at least for science policy—is a short item at the end of a long section on pesticides. The note is important because it indicates unequivocally that the EPA under Gorsuch set out to relax the cancer prevention standards used by the Carter Administration. It shows that the EPA's top leader and her colleagues at the White House knew that cancer policy was being changed and that it was shifting in a direction that some would perceive as exposing the public to a greater risk.

Specifically, the memo discusses an item on page 8 headed "permethrin." This was a pesticide which had been under review for many years. Animal tests suggested that it was a carcinogen for mice but not a strong one. In the previous Administration, the EPA set strict, low-tolerance levels for residues of potential carcinogens in food, even in ambiguous cases like this. In the permethrin decision, however, the new EPA leaders changed course. They decided to allow higher residue levels, arguing that some experts had concluded that, regardless of the mouse data, permethrin did not look like a compound that would cause cancer in humans. Describing this for the White House, the Gorsuch memo says that the EPA "will soon (next month) be issuing tolerances for permethrin—an insecticide. Environmentalists will be critical because this action would not have occurred under Carter Administration Cancer policy—a policy this Administration is changing. Congressional attention likely."

Although brief, the note does indicate that the EPA's leaders went into the cancer controversy with their eyes open. They may not have been quite as unprepared for the ensuing storm as they made out in congressional testimony later on. However, they clearly were unprepared for the intensity of the storm. —Eliot Marshall

California Universities Block Animal Rights Bill

A piece of California state legislation concerning the use of animals in research was scrapped last week, giving Stanford and the University of California a surprise victory over animal rights groups. "We routed them," said JoAnne Glissen, assistant director for legislative affairs at Stanford.

The legislation, introduced by Senate Pro Tem president David Roberti (D-Hollywood) would have banned the use for research of animals that came from pounds in California and other states. Although seven states have laws that ban the use of pound animals, the California proposal, if passed, would have set a precedent in prohibiting the importation of pound animals for experimental use.

The legislation was expected to pass easily through committee and the state assembly. But at the last minute, before the committee was to take up the bill for discussion, Roberti pulled his bill, apparently because he lacked the votes to get it through.

Glissen credits the universities' success to a "new strategy." Researchers had been writing their legislators to protest the bill, but that did not seem to be working very well. As momentum for the bill picked up, the universities teamed up an unlikely pair to lobby for them—the renowned Stanford cardiovascular surgeon Norman Shumway and soap opera superstar Stuart Damon, better known as Dr. Alan Quartermaine of "General Hospital." Damon took a particular interest in biomedical research because he has a child with juvenile diabetes. The universities also recently held a press conference with two heart transplant patients who extolled the benefits of animal research.

By marching out a leading scientist, movie star, and patients, the universities put an old and effective lobbying technique to new use. Special interest groups commonly follow this pattern to push for particular biomedical research legislation. But its use to counter animal welfare legislation is novel. (Glissen knows the lobbying method well because, until recently, she was on the staff of the U.S. Senate Labor and Human Resources Committee.)

Glissen said that in addition to scientists, "we got people without white coats into the process. That made the difference." The schools may have to use the strategy again if Roberti reintroduces his legislation.

—Marjorie Sun

NSF Urged to Support Academic Supercomputing

The National Science Foundation (NSF) has been urged to spend more than $500 million over the next 3 years to improve computing on university campuses. The recommendation was made in a report delivered to the National Science Board on 18 August by a working group consisting of NSF program managers.

The report recommended in particular that ten supercomputer centers be established for use by academic researchers, and that a substantial investment be made in local facilities for individuals or groups of scientists. The current state of computing in the universities is so poor, the working group said, that "even these investments would not provide computing facilities for academic researchers comparable to those at a typical national or industrial research laboratory."

The National Science Board, which had just adopted a statement of its own urging NSF to devote more resources to engineering, including improving computational facilities on campuses, was receptive. But the Office of Management and Budget will be harder to convince that expenditures of that magnitude—more than double currently planned outlays—are needed. —Colin Norman

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FOR IMMEDIATE RELEASE WEDNESDAY, MAY 19, 1976

EPA ADOPTS INTERIM CANCER ASSESSMENT PROCEDURES

Interim cancer assessment procedures have been adopted by the U.S. Environmental Protection Agency for use in making regulatory decisions where cancer risk is a key factor.

The procedures implement a decision of October 10, 1975, that rigorous assessments of health risk and economic impact will be undertaken as part of the regulatory process.

The preamble to the procedures, signed by EPA Administrator Russell E. Train, sets forth a summary statement on EPA's approach to regulatory action for suspect carcinogens. A copy of the procedures and preamble is attached.

In approving these procedures and guidelines, Administrator Train reemphasizes the Agency's commitment to reducing the burden of suffering from cancer caused by factors in the environment and reaffirms the Agency's policy of weighing risks and benefits in its regulatory approach to carcinogens wherever possible under its enabling legislation. The procedures and guidelines will also ensure that the public has free access to the Agency's deliberations at every step of the process.

The interim cancer assessment procedures are being circulated for comment to the other Federal agencies and organizations with responsibilities in this area, including the Departments of Health, Education, and Welfare; Interior; Labor; Commerce; and Agriculture and the Council on Environmental Quality; the National Science Foundation, EPA's Science Advisory Board and the EPA Pesticide Advisory Committee.

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EPA FORM 1510-1 (REV. 6-72)

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R-109

INTERIM PROCEDURES & GUIDELINES FOR HEALTH RISK AND ECONOMIC IMPACT ASSESSMENTS OF SUSPECTED CARCINOGENS

PREAMBLE

In issuing the Interim Procedures and Guidelines for Health Risk and Econonic Impact Assessments of Suspected Carcinogens, I think it appropriate to state once again EPA's approach to regulatory action for suspect carcinogens.

Cancer is the second ranking cause of death in this country; it has a particulary severe impact on the affected individuals and their families in terms of physical and mental suffering and economic costs. There is evidence that a substantial amount of human cancer is caused by chemical and physical agents in the environment. Bioassay programs, currently testing hundreds of substances, are beginning to show that some important industrial and agricultural chemicals are carcinogenic for animals and are, therefore, candidates for regulatory action.

The EPA, by law, has responsibility to regulate many agents which may either cause or promote the development of cancer. At present, EPA is charged with the responsibility to prohibit or restrict the use of carcinogenic pesticides. EPA also has authority to regulate those carcinogens which are emitted directly to the outside air by stationary sources (such as factories) and motor vehicles, or discharged into water from point sources, or found in drinking water. Other agencies such as the Occupational Safety and Health Administration and the Food and Drug Administration also have responsibilities to regulate carcinogens. It is important to emphasize that there are serious regulatory gaps which permit undesirable exposure of the public to carcinogens. I have strongly advocated the passage of a toxic substances bill to help close those gaps.

Regulatory action against chemical carcinogens is relatively new. Until the late 1950's, no agents, either chemical or physical, had been regulated in this country on the basis of their carcinogenic action with the sole exception of ionizing radiation, which had been known to cause cancer since the turn of the century. Standards of permissible exposure to ionizing radiation were set by the arbitrary use of safety factors applied to exposure levels that were known to have produced damaging health effects. It was not assumed that these permissible exposure standards were safe but rather that they represented upper limits of exposure with the understanding that actual exposures were to be kept as low as possible. In the debate over the health effects of radioactive fallout from atomic weapons in the 1950's, the evidence for a no-threshold concept for cancer induction emerged, which supported the idea that there is no such thing as a completely safe dose; in other words any exposure, however small, will confer some risk of cancer on the exposed population.

Evidence has accumulated that indicates that the no-threshold concept can also be applicable to chemical carcinogens. On the basis of this concept, the first significant regulatory legislation relating to chemical carcinogens, the Delaney Clause of the Pure Food and Drug Act, imposed a complete ban on any food additive that showed evidence of tumorigenic activity for humans or animals. This statutory requirement represents the approach of eliminating all risk, However, it has become increasingly clear that in many areas risks cannot be eliminated completely without unacceptable social and economic consequences.

Consonant with this view, the Federal Insecticide, Fungicide, Rodenticide Act (FIFRA), which is the enabling legislation for the control of health hazards for pesticides, requires a balancing of risks and benefits as the basis for final regulatory action. We, thus, have a comparable conceptual basis for the regulation of chemicals as for ionizing radiation where the philosophy has been to eliminate or reduce exposure to the greatest extent possible consistent with the acceptability of the costs involved.

I believe that it is important to emphasize the two-step nature of the decision-making process with regard to the regulation of a potential carcinogen. Although different EPA statutory authorities have different requirements, in general two decisions must be made with regard to each potential carcinogen. The first decision is whether a particular substance constitutes a cancer risk. The second decision is what regulatory action, if any, should be taken to reduce that risk.

With respect to the first decision -- whether a particular substance constitutes a cancer risk -- in very few cases is it possible to "prove" that a substance will cause cancer in man, because in most instances the evidence is limited to animal studies. In this regard, a substance will be considered a presumptive cancer risk when it causes a statistically significant excess incidence of benign or malignant tumors in humans or animals. However, the decision that a cancer risk may exist does not mean that the EPA will automatically take regulatory action. In the case of pesticides, the decision that a presumptive cancer risk exists will trigger the detailed and independent risk and economic assessments that form the basis for the second decision, namely, what, if any, regulatory action to take to eliminate or restrict the use of the pesticide. In other regulatory areas, for example those under the Clean Air Act, the Federal Water Pollution Control Act, or the Safe Drinking Water Act where a large number of suspect carcinogens may exist in the atmosphere or public water supplies, the detailed risk benefit assessment will, because of limited Agency resources, necessarily have to be carried out on a priority basis in terms of which agents appear to be the most important.

Once the detailed risk and benefit analyses are available, I must consider the extent of the risk, the benefits conferred by the substance, the availability of substitutes and the costs of control of the substance. On the basis of careful review, I may determine that the risks are so small or the benefits so great that no action or only limited action is warranted. Conversely, I may decide that the risks of some or all uses exceed the benefits and that stronger action is essential.

In considering the risks, it will be necessary to view the evidence for carcinogenicity in terms of a warning signal, the strength of which is a function of many factors including those relating to the quality and scope of the data, the character of the toxicological response, and the possible impact on public health. It is understood that qualifications relating to the strength of the evidence for carcinogenicity may be relevant to this consideration because of the uncertainties in our knowledge of the qualitative and quantitative similarities of human and animal responses. In all events, it is essential in making decisions about suspect carcinogens that all relevant information be taken into consideration.

In my opinion, the current guidelines represent a significant improvement in the Agency's approach to the processes of decision-making for carcinogens by providing improved procedures for making risk and benefit assessments while providing the maximum opportunity for public review of the Agency's deliberations. However, while these guidelines should improve Agency procedures, I do not view them as representing a change in the Agency's cancer policy. Earlier regulatory decisions involving various pesticides were also based in each case on a comprehensive evaluation of the scientifc evidence and a careful weighing of risks and benefits. These decisions in every instance resulted in selective control measures rather than a complete prohibition of use.

I want to emphasize that I will not permit these new procedural guidelines to unduly delay regulatory decision-making. I will be closely reviewing them to assure that they do not do so. If they do cause undue delay, they will be revised. I would like to point out that these guidelines provide a means of organizing available information rather than requirements for the acquisition of new information.

I believe that the approach presented here is a significant step toward the objective of achieving real benefits in improved public health while avoiding the burden of undesirable regulatory action. I recognize that the aspect of cancer research dealing specifically with the issues involved in decision-making is relatively undeveloped, but hopefully the commitment of this Agency and other Federal agencies to the development of new knowledge in this area will improve the scientific basis for regulatory decisions and that the Interim Procedures and Guidelines will thereby benefit from periodic revision.

I consider it extremely important that the leading government agencies work closely with each other and with experts outside the government in the field of carcinogenicity in the development of government procedures and policies concerning cancer. I am publishing these interim procedures and the guidelines in the Federal Register not only to provide public notice of the approach which EPA will be following in our current activities but also to stimulate commentary from all sources upon that approach. I am also furnishing copies of these Interim Procedures and Guidelines to and requesting the views of the Secretaries of Health, Education, and Welfare, Interior, Labor, Commerce and Agriculture and also the Council on Environmental Quality, the National Academy of Sciences, the National Science Foundation, EPA's Pesticide Policy Advisory Committee and EPA's Science Advisory Board, among others. I also plan to meet personally with leading authorities in this area as part of a continuing process to discuss these cancer policies and exchange information and views.

Administrator

Interim Administrative Procedures for Regulatory Decisions Involving Suspected Carcinogens

Procedures described in this paper provide a more uniform Agency approach to regulatory decisions involving cancer risk. Procedure A applies to pesticide decisions involving the cancellation, suspension and registration of potentially carcinogenic pesticides. Procedure B applies to other selected Agency decisions where the pivotal factor in the decision is cancer risk.

The purpose of these procedures is to assure that appropriate analyses of the risks and benefits of suspected carcinogenic chemicals are performed as part of the regulatory process. Appendices I and II establish guidelines for risk assessment and economic impact analyses. These guidelines are procedural guidelines and are not intended to affect the substantive regulatory standards of any statute. Therefore, the assessment of the risk posed by potentially carcinogenic substances will be made pursuant to the individual standards of the applicable statute and regulations. Furthermore these analyses will be carried out within the constraints of Agency resources and will not delay actions by the Agency to address urgent environmental problems.

The Cancer Assessment Group (CAG) is an advisory body comprised of senior scientists from within the Agency with a liaison member from the Department of Health, Education and Welfare. It will also utilize, as appropriate, expert consultants and advisors from various Federal Agencies and the private sector. The CAG will conduct analyses of data related to risk and make recommendations to the lead program office and the appropriate Working Group concerning the risk associated with each suspect carcinogen. These analyses will be directed towards risk assessment and will be conducted independently of economic impact analyses. The CAG will also review the final risk assessment portion of the regulatory package.

Applicability

For all decisions involving the cancellation, suspension, reregistration and registration of potentially carcinogenic pesticides, Procedure A will be followed inclusive of the preparation of (1) a risk assessment pursuant to the interim guidelines contained in Appendix I and (2) an economic impact analysis pursuant to the interim guideline contained in Appendix II.

For the following rulemaking, where the pivotal factor in the decision is cancer risk, the procedures outlined in EPA Order 1000.6 will be followed, and in addition, a risk assessment pursuant to Appendix I will be prepared and will be reviewed in accordance with Procedure B:

1. Proposed regulations to augment the current list of toxic substances published pursuant to Section 307(a) of the FWPCA and any standard proposed under this augmented list.
2. Primary drinking water regulations or revisions thereof under Section 1412 of SDWA.
3. Additions to or revisions of the water quality criteria (pursuant to Section 304(a) of FWPCA) currently pending publication, except that detailed exposure patterns and estimates of cancer risk need not be prepared.

4. Proposed technology-based regulations or revisions pursuant to Sections 301, 304, 306, 307(b) and 307(c) of the FWPCA (proposed after April 1, 1977), and Section 111 of the CAA, except that detailed exposure patterns and estimates of cancer risk need not be prepared.

For all other rulemaking under existing legislation which involves the regulation of a potential carcinogen(s), and which is not currently under development, the determination of whether and to what extent to use Appendix I and Procedure B will be made at the time the Administrator approves the plan for such rulemaking.

Where the development of a surrogate parameter is being proposed to regulate one or more potential carcinogens and perhaps other pollutants (e.g., a total organic carbon standard for drinking water), the risk assessment, as required above, will address at least one of the potential carcinogens and should address, to the extent feasible, as many of the others as possible.

All risk assessments need only be based on currently available information. These procedures do not require the undertaking of research or monitoring to expand the available data base.

A. Procedure for Pesticide Decisions Involving Potential Carcinogens

This procedure is similar to the current procedure for informal rulemaking set forth by EPA Order 1000.6.

1. Formation of the Working Group

The Deputy Assistant Administrator for Pesticides, in cooperation with the Office of Planning and Management, establishes a working group.

2. OPP/Working Group Responsibility

The Office of Pesticide Programs (OPP), in consultation with the Working Group, is responsible for developing a Data Summary Report, a Position Document (including health risk assessment and the economic impact analysis) and a proposed Federal Register Notice at the appropriate points in the regulatory process. Guidelines for health risk assessment and economic analysis are included as Appendices I and II.

3. Review of a Suspect Chemical Prior to Reregistration or the Issuance of a Rebuttable Presumption Against Registration (RPAR)

a. Data relevant to the carcinogenicity of a pesticide is submitted to the CAG for review and comment. Following review by the CAG, a Data Summary Report is prepared by OPP and the Working Group. This report includes a summary of all available data relevant to carcinogenicity.

b. A draft Position Document including the Data Summary Report, a summary of the issues surrounding potential regulatory actions, and a proposed Federal Register Notice are presented to the Pesticide Chemical Review Committee (PCRC) which includes a representative from the CAG.

c. On the basis of PCRC comments, the OPP and the Working Group revise the draft Position Document and the Federal Register Notice. The PCRC reviews the revised package.

d. The package recommending a reregistration or the issuance of a RPAR goes to the Deputy Assistant Administrator for Pesticide Programs for a final decision.

4. Post-RPAR: Issuance of a Notice of Intent to Cancel, Suspend or Reregister

a. After a RPAR is issued, and rebuttal information if any is submitted, the OPP and the Working Group develop a final Position Document. This document includes a summary of all information available in rebuttal of the RPAR, a recommended finding on whether or not the presumption against registration has been rebutted (including the risk assessment), economic impact analysis as necessary, a summary of the issues surrounding potential regulatory actions, and a draft Federal Register Notice.

b. The final Position Document is reviewed by PCRC and the risk assessment is reviewed by CAG.

c. If the decision is to reregister the product, a notice to this effect is published in the Federal Register.

d. If the decision is to cancel or suspend the product, the proposed notice of intent to cancel or suspend is forwarded to USDA and the Scientific Advisory Panel for comment, pursuant to the 1975 amendments to Section 6(b) of FIFRA. However, if it is determined that suspension of the pesticide is necessary to prevent an imminent hazard to humans, the 1975 amendments provide for waiver of the requirement for consultation with USDA and the Scientific Advisory Panel.

The notice of intent to register, cancel or suspend, including the risk assessment and economic impact analyses, is circulated for General Counsel and Assistant Administrator concurrence and forwarded to the Administrator for a final decision.

B. Other Rulemaking to Regulate Carcinogens

All other Agency decisions involving carcinogenesis as the pivotal factor will follow EPA Order 1000.6 with the following additions:

1. The CAG will review the relevant data during the development of the rulemaking and make recommendations to the lead office and the working group regarding the interpretation of the data and provide other advice, as appropriate, concerning the risk assessment.
2. The CAG will review that portion of the rulemaking package containing the risk assessment. CAG comments will be presented to the Steering Committee.

C. External Scientific Review

In addition to the external reviews required by statute and the 1000.6 process, other external scientific review will be obtained in appropriate cases as determined by the lead program office. This review may take place at any time in the development of the regulatory package.

While risk and economic impact analyses may be reviewed externally, regulatory recommendations will not normally be submitted for external review. Reviewers for risk analyses may be from the Science Advisory Board, National Cancer Institute, or other appropriate Institutions.

APPENDIX I

Interim Guideline
for
Carcinogen Risk Assessment

1.0 Introduction

This preliminary guideline describes the general framework to be followed in developing an analysis of carcinogen risks and some salient principles to be used in evaluating the quality of data and formulating judgements concerning the nature and magnitude of the cancer hazard from suspect carcinogens.

This guideline is to be used within the policy framework already provided by applicable statutes and does not alter such policies. The guideline provides a general format for analyzing and organizing available data. It does not imply that one kind of data or another is prerequisite for regulatory action to control, prohibit, or allow the use of a carcinogen. Also, the guideline does not change any statutory-prescribed standards as to which party has the responsibility of demonstrating the safety, or alternatively the risk, of an agent.

The analysis of health risks will be carried out independently from considerations of the socio-economic consequences of regulatory action.

The risk assessment document will contain or identify by reference the background material essential to substantiate the evaluations contained therein.

2.0 General Principles Concerning the Assessment of Carcinogenesis Data

The central purpose of the health risk assessment 1/ is to provide a judgement concerning the weight of evidence that an agent is a potential human carcinogen and, if so, how great an impact it is likely to have on public health.

Judgements about the weight of evidence involve considerations of the quality and adequacy of the data and the kinds of responses induced by the suspect carcinogen. The best evidence that an agent is a human carcinogen comes from epidemiological studies in conjunction with confirmatory animal tests. Substantial evidence is provided by animal tests that demonstrate the induction of malignant tumors in one or more species including benign tumors that are generally recognized as early stages of malignancies. Suggestive evidence includes the induction of only those nonlife shortening benign tumors which are generally accepted as not progressing to malignancy, and indirect tests of tumorigenic activity, such as mutagenicity, in-vitro cell transformation, and initation-promotion skin tests in mice. Ancillary reasons that bear on judgements about carcinogenic potential, e.g., evidence from systematic studies that relate chemical structure to carcinogenicity should be included in the assessment.

When an agent is judged to be a potential human carcinogen, estimates should be made of its possible impact on public health at current and anticipated levels of exposure. The available techniques

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1/ This health risk assessment is part of the risk-benefit analyses. In actions taken to regulate pesticides, this assessment is made after a determination that a health risk exists.

for assessing the magnitude of cancer risk to human populations on the basis of animal data only are very crude due to uncertainties in the extrapolation of dose-response data to very low dose levels and also because of differences in levels of susceptibility of animals and humans. Hence, the risk estimates should be regarded only as rough indications of effect. Where appropriate, a range of estimates should be given on the basis of several modes of extrapolation.

Expert scientific judgements in the areas of toxicology, pathology, biometry, and epidemiology are required to resolve uncertainties about the quality, adequacy, and interpretation of experimental and epidemiology data to be used for the risk assessment.

3.0 Format of the Risk Analysis

3.1 Exposure Patterns

This section should summarize the known and possible modes of exposure attendant to the various uses of the agent. It should include or identify by reference available data on factors relevant to effective dosage, physical and chemical parameters, e.g., solubility, particle size for aerosols, skin penetration, absorption rates, etc. Interaction of agents which may produce a synergistic or antagonistic effect should also be indicated, if available.

3.2 Metabolic Characteristics

This section should summarize known metabolic characteristics including transport, fate and excretion, and biochemical similarities to other known classes of carcinogens at high and low dose levels and should provide comparisons between relevant species as well as variations in different strains of certain species.

3.3 Experimental Carcinogenesis Studies

Available experimental reports should be summarized. If some experiments are to be rejected for the risk assessment, give reasons for doing so. Reprints of key papers and reports should be included as appendices to the analysis.

Judgements should be provided on the quality of the experimental data and their interpretations for each study on the basis of (a) experimental protocols, (b) survival rates in controls particularly in relation to acceptance of negative results, (c) incidence of spontaneous tumors in the control compared to general laboratory experience for the same species or strain, (d) diagnostic criteria and nomenclature used for tumor characterization (additional evaluation of histological material should be obtained when appropriate), and (e) observed results of positive controls (i.e., a test group given a standardized exposure to a known carcinogen) in light of expected results.

3.4 Epidemiological Studies

Summarize epidemiological studies, together with critiques of the work with respect to its limitations and significance. Summarize other published critiques whether supportive or at variance with the judgement made here.

3.5 Cancer Risk Estimates

3.5.1 Exposure Patterns

Describe likely exposure levels with respect to long-term temporal trends, short-term temporal patterns, and weighted averages for both the total exposed populations and for subgroups whose exposure patterns may be distinctly different from the average. Characterize, to the extent possible, the size of the exposed population for each of the above categories with an indication of whether the exposures are likely to involve children and pregnant women. Discuss the adequacy of the methods used to estimate exposures and indicate the range of uncertainty in the estimates.

3.5.2 Dose-Response Relationships

Both human and animal data should be used as available. Include available human data, even if inadequate for a characterization of the actual magnitude of risk, where such data could be helpful in interpreting animal responses in relation to human sensitivity.

3.5.3 Estimates of Cancer Risk

The procedure will involve a variety of risk extrapolation models, e.g., the linear non-threshold model and the log-probit model. Analyses will be done separately for all suitable experimental data and human epidemiological data. The results should be presented in terms of excess lifetime incidence, or average excess cancer rates; life-shortening estimates should also be made when the data permit. The uncertainty in the data and extrapolation techniques should be clearly indicated. The results predicted for humans should be presented in relationship to the current cancer experience in the assumed target organ(s).

Some judgements should be included regarding the relevance of the mode of exposure used in animal studies to that associated with human exposure.

4.0 Summary

The summary section of the risk assessment should provide a statement which encompasses answers to the following questions: (1) How likely is the agent to be a human carcinogen? (2) If the agent is a human carcinogen, what is the estimated impact on human health?

APPENDIX II

INTERIM GUIDELINE FOR ECONOMIC IMPACT ANALYSIS OF PROPOSED REGULATORY ACTIONS TO CONTROL CARCINOGENIC PESTICIDES

The purpose of this guideline is to define the factors to be considered and the procedures to be utilized in assessing the economic impact resulting from future regulatory actions (as described below) affecting carcinogenic pesticides. Economic impact assessment for other regulatory actions to control environmental carcinogens will follow established agency procedures.

The principal concern in the economic analysis will be the assessment of economic impacts on pesticides users and on the consumers of the products of the users. The impacts on pesticide manufacturers are not germane to this type of regulatory decision, in which the risk of the use of a pesticide is compared to the benefit of those uses.

As used in this guideline the economic impact of the regulation is equated to the anticipated loss in benefit from use of the pesticide. For agricultural pesticides the analysis will focus on the impacts on farmers, farm productivity, and consumer costs associated with farm productivity. Similarly, analyses of other pesticides will focus on the impacts on other user groups and related effects on the economy.

Regulatory Procedures

The purpose of this section of the guidelines is to define how the economic impact analysis fits into the regulatory framework for pesticide-related actions.

If a pesticide meets or exceeds criteria defined in 40 CFR §162.11, a Rebuttable Presumption Against Registration (RPAR) will be issued. The Agency will analyze any rebuttal information that is submitted; it may also take into account other available information to determine whether the RPAR has been rebutted. At the conclusion of this risk assessment, the Administrator will be presented with sufficient evidence to determine if the use of a pesticide poses the risk of a significant adverse effect. If such is the case, then the Administrator must determine what type of regulatory response is warranted.

In making that decision, 40 CFR §162.11 provides that the Administrator will be provided with a preliminary assessment of the benefits of the use of the pesticide. Furthermore, §162.11 essentially provides: (1) that if the risks appear to outweigh the benefits, the Administrator will issue a notice of intent to cancel, which may lead to a full adjudicatory hearing on the question of whether the pesticide causes or will cause unreasonable adverse effects on the environment, or (2) if the benefits appear to outweigh the risks, the Administrator will either issue a notice of intent to hold a hearing (adjudicatory or non-adjudicatory) or a notice of intent to register. Such a notice of intent to register provides an opportunity for a hearing upon request (accompanied by submission of a statement of factual reasons) of an interested party that a hearing is warranted. The decision to cancel reached at this time will not result in the removal of a product from the market if the decision is contested. Instead, any such regulatory action will be preceded by a hearing to weigh fully the risks and benefits of the uses of a product.

The benefit evidence provided to the Administrator at this stage is by definition a preliminary staff analysis. A specific effort will be made by the Agency to contact parties that have an interest in the use of the pesticide and to attempt to solicit their comments on the benefits of the pesticide under review. In particular, EPA intends that the U.S. Department of Agriculture will be heavily relied upon from the earliest stages of review to provide its special expertise and data resources on uses.

Because of the many variables surrounding the multiple uses of different pesticides, the benefit or economic impact analysis must of necessity be done on a case-by-case basis. All relevant economic considerations raised in criticisms of the preliminary benefit analysis will be addressed prior to final action.

Content of the Economic Impact Analyses

Based upon all the available information, a preliminary analysis will be developed. Such analysis will be organized in the following manner:

1. Identification of the major uses of the pesticide, including estimated quantities used by crop or other application.
2. Preliminary identification of the minor uses of the pesticide, including estimated quantities used by category such as lawn and garden uses and household uses.
3. Identification of registered alternative products for the uses set forth in (1) and (2) above, including an estimate of their availability.
4. Determination of the change in costs to the user of providing equivalent pesticide treatment with any available substitute products.
5. Assessment of regulation impact upon user productivity (e.g., yield per acre and/or total output) from using available substitute pesticides or from using no other pesticide.
6. If the impacts upon either user costs or productivity are significant, a qualitative assessment of the regulation's impact on production of major agricultural commodities and retail food prices of such commodities.

Environmental Protection Agency, Rm. E-124, 401 M St., SW., Washington, D.C. 20460, (202-426-223).

SUPPLEMENTARY INFORMATION: Flax does not compete well with weeds; weed infestations result in poor stands of flax by reducing germination and competing with the developing flax plants. Wild oats and foxtails are common weed pests of flax. According to the Applicants, these weeds are present in all areas where flax is produced, and due to heavy snowfall this past winter, heavy weed growth delayed planting and shortened the time for use of preplant herbicides.

Eptam, Avadex, Dalapon, Carbyne, MCP Amine and Bromoxynil are registered for selective weed control in flax. According to the Applicants, the registered alternative pesticides are either not effective or are not acceptable control methods for wild oats, wild buckwheat and foxtails for this season's wet conditions.

Minnesota reports that Kittson, Lake of the Woods, Marshall, Pennington, and Roseau Counties, where flax is a major crop, grow from 75,000 to 95,000 acres of flax annually. Significant losses in agricultural income may be incurred, the Applicants claimed, if the exemptions were not granted.

North Dakota reports that Bottineau, Cavalier, Pierce, Pembina, Rollette, and Towner Counties annually grow about 98,000 acres of flax.

The Applicants proposed to use an asulam formulation, Asulox, EPA Reg. No. 359-682, in a single post-emergence application when wild oats are in the three- to four-leaf stage. Applications will be made by both private and commercial State-certified applicators using both ground and air equipment in Minnesota and ground equipment only in North Dakota in the counties named above.

EPA has determined that the available data are adequate to support the proposed use of Asulox on flax. Residues of the active ingredient (a.i.) asulam are not likely to exceed 2.0 parts per million (ppm) in flax seed and its fractions or 2.5 ppm in flax straw. EPA has deemed these levels to be adequate to protect the public health. Appropriate crop rotation restrictions have been imposed. No unreasonable adverse effect on the environment from this use of asulam is anticipated.

After reviewing the application and other available information, EPA has determined that the criteria for exemptions have been met. Accordingly, the Applicants have been granted specific exemptions to use the pesticide noted above until October 31, 1980, to the extent and in the manner set forth in the applications. The specific exemptions are also subject to the following conditions:

1. A single post-emergence application of Asulox (EPA Reg. No. 359-682) is authorized.
2. Application shall be made by air and/or ground equipment in Minnesota and by ground equipment in North Dakota, at a rate not to exceed 1.25 pounds a.i. per acre.
3. A maximum of 10,000 gallons of formulation may be applied to 32,000 acres of flax in the five Minnesota counties mentioned above; a maximum of 68,000 acres of flax in the six North Dakota counties named above may be treated.
4. Applications will be made when wild oats are in the 3-4 leaf stage.
5. All applications shall be made by State-certified private and commercial applicators.
6. Precautions shall be taken to avoid or minimize spray drift from target area. Application may not be made when weather conditions favor spray drift.
7. Residue levels of asulam are not expected to exceed 2.0 ppm in flaxseed and its fractions (meal, oilseed cake, refined oil, and soapstock) and 2.5 ppm in the flax straw. The Food and Drug Administration, U.S. Department of Health and Human Services, has been advised of these actions. Any resulting residues in meat, milk, poultry, and eggs will be well below detectable levels (0.05 ppm in meat, 0.025 ppm in milk, and 0.1 ppm in poultry tissues and eggs).
8. Crops other than small grains may not be planted in the treated area within 12 months of application. Small grain crops may not be planted within ten months of application. Root crops may not be planted in the treated area within 18 months of application. Fodder from grain crops rotated to treated flax fields may not be grazed or cut for forage.
9. All applicable directions, restrictions, and precautions on the EPA-registered label must be followed.
10. Application of Asulox to flax may result in crop injury and reduction in yield under stress conditions or if the herbicide is applied at stages of growth other than those specified.
11. The EPA shall be immediately informed of any adverse effects resulting from the use of Asulox in connection with these exemptions.
12. Each Applicant is responsible for ensuring that all of the provisions of its specific exemption are met and each must submit a report summarizing the results of this program by January 15, 1981.

(Sec. 18 as amended 92 Stat. 819; (7 U.S.C. 136p))

Dated: November 6, 1980.

James M. Conlon,

Associate Deputy Assistant Administrator for Pesticide Programs.

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[HP FRL 1563-2]

Mutagenicity Risk Assessments; Proposed Guidelines

October 30, 1980.

AGENCY: Environmental Protection Agency.

ACTION: Proposed guidelines for Mutagenicity Risk Assessments.

SUMMARY: The Environmental Protection Agency has developed Guidelines for Mutagenicity Risk Assessments (Guidelines). These Guidelines will be used within the policy and procedural framework provided by the various statutes which EPA administers, to guide Agency analysis of mutagenicity data.

These guidelines were developed by an Agency-wide working group, under the direction of the Reproductive Effects Assessment Group (REAG). REAG is an advisory body located within the Agency's Office of Research and Development, which serves as the senior Agency technical review group for mutagenicity assessment performed by Agency program offices. A preliminary draft of the Guidelines was sent for review to approximately 30 scientists in the field of chemical mutagenesis within government, universities in the United States and abroad, and the private sector. In general, the comments received were favorable, and changes in the draft were made, as appropriate, in response to them. In addition to soliciting public comments on the Guidelines by publication of this **Federal Register** Notice, the Agency is providing them to the Agency's Science Advisory Board for review.

FOR FURTHER INFORMATION CONTACT: Dr. Donna Kuroda, 202/426-2275.

DATES: Comments must be submitted by February 11, 1980.

ADDRESS: Reproductive Effects Assessment Group (RD-689), Office of Health and Environmental Assessment, Environmental Protection Agency, 401 M Street SW., Washington, D.C. 20460.

SUPPLEMENTAL INFORMATION:

I. Introduction

As a result of the progress in the control of infectious diseases, increase in average human lifespans, and better procedures for identifying genetic disorders, a considerable heritable genetic disease burden has been recognized in the human population. It is estimated that at least 10% of all human disease is genetically related, resulting from changes in the composition or arrangement of genes and chromosomes (1, 2). Such genetic diseases can lead to structural or functional health impairments. Moreover, many of these diseases are expressed in infancy and in early childhood and are chronic in nature. As a result, they cause a severe impact upon the affected individuals and their families in terms of physical and mental suffering and economic losses, and upon society in general, which may often become responsible for institutional care. Some examples of genetic diseases and syndromes genetically based include Down's syndrome and Klinefelter's syndrome, cystic fibrosis, hemophilia, sickle cell anemia, and achondroplastic dwarfism. Other commonly recognized diseases which are likely to have a genetic basis include hypercholesterolemia, rheumatic fever, hypertension, pyloric stenosis, glaucoma, allergies, and several types of mental retardation. These diseases are only a few of the thousands which are associated with genetic errors (3).

Mutations are largely recognized as being deleterious to the organisms receiving them. The adverse effects may be manifested at the biochemical, cellular, and physiological levels of organization. Although mutations are the building blocks for further evolutionary change of species, it is believed that increases in the mutation rate above the spontaneous level will lead to an accumulation of deleterious mutations in the human population (4). Since the existing incidence of human genetic disease is already sizable, the Agency wants to control exposures to chemicals which may add to this existing burden.

Because of the technical and industrial expansion during the last two decades, a large number of chemicals have been released into our environment. Some have been shown to have mutagenic activity in mammalian and submammalian test systems, and thus have the potential to increase genetic damage in the human population. Chemicals with mutagenic activity in various test systems have been found widely distributed among drugs, food additives, cosmetics, industrial compounds, pesticides, and consumer products. As our knowledge of genetics and disease etiology increases, it is reasonable to believe that we will become aware of some chemically-induced human genetic effects. The extent to which exposure to these natural and man-made environmental agents may have increased the amount of genetic damage in the present human population and contributed to the mutational "load" that will be transmitted to future generations is unknown at this time. Since the prospect of curing most heritable diseases caused by mutagens in the near future is unlikely, minimizing exposure to mutagens is among the best available means to protect against further deterioration of the human gene pool.

This document describes the procedures that the Environmental Protection Agency (EPA) will follow to evaluate the risk associated with the exposure of humans to chemical mutagens. Previously, the Agency indicated its intent to develop procedures in the "Criteria for Evaluating the mutagenicity of chemicals" which describe the basic concerns and need for such a document (5). These procedures will ensure quality and consistency in the Agency's scientific risk assessments for mutagenic effects. The necessity for a consistent approach to evaluate the mutagenic risk from chemical substances arises from the authority conferred upon the Agency by a number of statutes to regulate potential mutagens. These statutes include the Federal Insecticide, Fungicide, and Rodenticide Act, the Toxic Substances Control Act, the Clean Air Act, the Federal Water Pollution Control Act, the Safe Drinking Water Act, and the Resource Conservation and Recovery Act.

The mutagenicity risk assessments prepared pursuant to these guidelines will be utilized within the requirements and constraints of the applicable statutes to arrive at regulatory decisions concerning mutagenicity. The standards of the applicable statutes and regulations may dictate that additonal considerations, e.g., the economic and social benefits associated with use of the chemical substance, will come into play in reaching appropriate regulatory decisions.

In order to ensure the quality and consistency of the Agency's scientific risk assessments, EPA has created the Reproductive Effects Assessment Group (REAG) in its Office of Research and Development. This group will have the responsibility for carrying out an oversight function for the Agency's risk assessment activities to ensure consistent application of mutagenicity risk assessment procedures, as well as the responsibility to conduct mutagenicity risk assessments for program offices when deemed necessary. The REAG will utilize, as appropriate, expert consultants and advisors from various Federal agencies, academia, and the private sector to assist in reviewing mutagenicity risk assessments performed by the program offices or to assist REAG in performing risk analyses at the request of the program offices.

The assessment of heritable mutagenic risk to humans involves a two-step process. First of all, the mutagenicity of the chemical must be determined, and secondly, evidence must be evaluated to determine if the chemical reaches or affects gonadal organs. Results from such an evaluation can be combined with expected human exposure information, when available, and the impact of the mutagenic risk for human populations can be estimated to the extent possible.

II. Concepts Relating to Heritable Mutagenic Risk

For the purposes of these guidelines, a mutagen is defined as a chemical substance or mixture of substances that can induce alterations in the DNA of either somatic or germinal cells of organisms. The Agency is concerned with the risk associated with both germ cell mutations and somatic mutations. Mutations carried in germ cells can be inherited by future generations and contribute to genetic disease, whereas mutations occurring in somatic cells may be implicated in the etiology of several disease states, including cancer (6,7). These guidelines, however, only concern genetic damage as it relates to germ cell mutations. The position on the use of mutagenicity test results in the assessment of carcinogenic risk has been described in the Agency's Interim Cancer Assessment Procedures (8).

The potential for mutagenic risk to germ cells can be identified on the basis of tests involving either germ or somatic cells. In the case of somatic cell tests, however, the quantitation of mutagenic risk depends on evidence that the chemical or its metabolites reaches or affects a critical target (DNA) in germinal tissue.

There are several mutagenic endpoints of concern to the Agency. These include point mutations (i.e., change in the base sequence of DNA) and structural and numerical chromosome aberrations. Structural aberrations include deficiencies, duplications, inversions, and translocations, whereas numerical aberrations refer to gains or losses of whole chromosomes (aneuploidy) or sets of chromosomes (haploidy, polyploidy). The Agency's Office of Pesticide Programs already has published various testing standards for the detection of mutagenic effects (9).

For some mutagenic mechanisms, it is conceivable that only one or a few molecules of the active compound may cause heritable changes in DNA. Mutagenic effects may also arise by mechanisms not directly related to chemical alterations of DNA, such as interference with DNA synthesis, DNA repair, or nuclear division processes.

III. Rationale for Using Nonhuman Test Systems

The virtual universality of DNA as the genetic material and of the genetic code provides a rationale for using various non-human test systems to predict the intrinsic mutagenicity of test chemicals. Additional support is provided by the observation that chemicals causing genetic effects in one species or test system frequently cause similar effects in other species or systems. Although cells of any species could theoretically be used to detect and predict genetic change in other species, certain test systems offer notable advantages, such as increased sensitivity; ease and speed of conducting the tests; low cost; anatomical, histological, and/or metabolic similarities to humans; suitability for handling large numbers of test organisms; a large data base; and a basis for characterizing genetic events.

Nonhuman test systems must be used because of the lack of suitable epidemiological data on germ cell mutations in humans. Clear epidemiological evidence for an increased mutation rate in humans due to exposure to a chemical substance is unavailable at this time. Presently only a very few of the estimated many thousands of genes in the human genome are useful as markers in determining the point mutation rates within human populations; there is a huge amount of genetic variability in the human population; and humans have a long generation time. Dominant and sex-linked recessive mutations can be detected in the first generation following treatment; however, autosomal recessive mutations and those affecting multifactorial traits largely go unnoticed because they are not recognized in the heterozygous state. Information on chromosomal aberrations induced in humans can be obtained with cytological techniques, but this information is limited. Thus, in most cases, although mutational changes occur in humans, they will be unobserved or unexpressed until future generations.

IV. Risk Assessment

A. Testing Systems

Assessing risks as a result of exposure to chemical mutagens is a complex task because of the diversity in chemical structure of mutagens and the various ways they can interfere with genetic processes. Therefore, in order to adequately assess the mutagenic potential of a chemical, data from several test systems may be needed to evaluate the ability of a chemical to produce chromosomal alterations, point mutations, and primary DNA damage (manifested by DNA repair or recombinational events). There are many testing systems currently available which can contribute information about the mutagenic potential of a test compound.

Test systems for detecting point mutations include those in bacteria, eukaryotic microorganisms, insects, and mammalian somatic cells in culture, as well as tests for detecting germinal mutations in intact mammals (e.g., the mouse specific locus test). Positive results in a mouse specific locus test argue strongly that a chemical is a potential human mutagen because the test demonstrates the mutation occurs in germinal cells and is transmitted to the next generation. However, since the development of mouse specific locus data is limited by the requirements for special strains of mice and a large number of test animals, it is not expected that many chemicals will be tested using this system. To obtain data on a large number of environmental pollutants, it will be necessary to rely on other tests to assess the risk of point mutations. Although prokaryotes, lower eukaryotes, and mammalian cells in culture do not have the ability to carry out all types of metabolic conversions that occur in intact mammals, the addition of a metabolic activation system derived from mammalian tissues has made these systems a valuable tool for detecting mutagens.

For detecting chromosomal aberrations, the following test systems have been employed: *in vitro* and *in vivo* mammalian cytogenetic tests, insect tests for heritable chromosome effects, dominant lethal tests in rodents, the heritable translocation test in rodents, and the mouse sex chromosome loss test. The heritable translocation test in rodents is among the best available tests for detecting heritable chromosomal rearrangements in mammals; the mouse X-chromosome loss test is a value in detecting the unequal distribution of chromosomal material during gametogenesis.

Other tests have been designed to detect nondisjunction (unequal distribution of chromosomes during cell division) in such diverse systems as the mouse, *Drosophila*, fungi, and mammalian cells in culture. The present data base for these systems, however, is limited at this time. Primary DNA damage which provides information bearing on the mutagenicity of a chemical can also be detected by a variety of test systems, such as unscheduled DNA repair synthesis in mammalian cells, mitotic recombination in yeast, and sister-chromatid exchange in mammalian somatic and germ cells.

The test systems mentioned above are not necessarily the only test systems that will provide the best evidence of mutagenicity. These systems are enumerated merely to demonstrate the breadth of the available techiques for assessing mutagenic potential, and to indicate the types of data which the Agency will consider in its evaluation of mutagenic potential. Most systems possess certain limitations which must be taken into account. The selection and performance of appropriate tests for evaluating the risks associated with human exposure to any suspected mutagen will depend on sound scientific judgment and experience, and may necessitate consultation with geneticists familiar with the sensitivity and experimental design of the test system in question. In view of the rapid advances in test methodology, the Agency expects that more relevant tools for predicting human mutagenic potential will become available with time. The Agency will closely monitor developments in mutagenicity evaluation and will refine its risk assessment scheme as better test systems become available.

B. Weight-of-Evidence Approach

The judgment as to whether a chemical substance is likely to be a potential human mutagen will be based on a weight-of-evidence approach that involves and considers the quality and adequacy of all the available data.

A positive response in the mouse specific locus test alone can provide strong evidence to regard a chemical as a potential human mutagen. As an alternative to the mouse specific locus test, positive responses in any two different point mutation test systems plus evidence for the presence of the test substance and/or its metabolites in mammalian gonadal organs also provides strong evidence that a compound is a potential human mutagen. Positive results in at least two systems are desirable because of the possibility of obtaining false positive or species-specific positives.

The heritable translocation test or the X chromosome loss test for chromosomal alterations in mice can provide equally strong evidence that a chemical is a potential human mutagen. Both tests detect events that are induced in treated mammals and transmitted to progeny where they are scored. In the absence of positive findings in these two test systems, positive results in two *in vivo* somatic cell cytogenetic test systems or in an *in vitro* cytogenetic test system and an *in vivo* somatic cell cytogenetic test system, coupled with evidence for the presence of the test substance and/or its metabolites in mammalian gonadal organs, will provide strong evidence that a compound is a potential human mutagen.

The test systems described here may not detect all the genetic events that are of concern to humans. Furthermore, the sensitivity of some of these systems to detect mutations is not known and may be low for certain chemical classes. Nevertheless, positive responses in these tests currently provide the best experimental evidence that a chemical has the potential to produce genetic damage in people.

If data suitable for applying the criteria just described are not available, then the chemical substance under consideration will be assessed by examining all data that give insight to its mutagenic activity. The following factors will be considered:

1. Genetic endpoints (e.g., point mutations, chromosomal mutations) detected by the test systems.
2. Sensitivity and predictive value of the test systems for various classes of chemical compounds.
3. Number of different test systems used for detecting each genetic endpoint.
4. Consistency of the results obtained in different test systems and different species.
5. Whether the tests are conducted in accordance with appropriate test protocols agreed upon by experts in the field.
6. Evidence that a chemical reaches or affects the germinal tissue, as provided by such sources as data demonstrating the alkylation of DNA or other cellular macromolecules, unscheduled DNA synthesis, sister chromatid exchange, or chromosome aberrations, in germinal cells; and non-specific accumulation of radioactive label in the gonads following administration of the labeled chemical. Other relevant evidence includes adverse gonadal effects following acute, subchronic or chronic toxicity testing; and adverse reproductive effects, such as decreased fertilization index, reduced sperm count, or abnormal sperm morphology.

Data on these factors will be used to determine whether there is strong, substantial, or only suggestive evidence that a particular chemical may be potentially mutagenic to humans.

Although definitive proof of non-mutagenicity is not possible, it seems appropriate that a chemical could be classified operationally as a non-mutagen if it gives a negative response in an adequate number of test systems that are together capable of detecting all genetic endpoints of concern, namely, point mutations, chromosomal effects, and primary DNA damage. Furthermore, if a chemical does not reach or affect gonadal organs, it can be considered not to cause heritable mutations. Stringent scientific criteria (e.g., adequate sample size, suitable experimental design, appropriate concentration range, proper controls, and adequate number of tests) should be required for acceptance of negative results to assure that the failure to detect genetic activity or the presence of the chemical in the gonadal tissue is not based on inadequacies of the test procedures.

C. Quantitative Assessment of Results

The degree of risk can be estimated by combining the preceding qualitative evaluation for mutagenicity with the potential for human exposure. When there is a lack of appropriate mutagenicity data to make a quantitative estimate, risk can still be expressed in meaningful descriptive terms. To do this, the strength of evidence that the agent possesses intrinsic mutagenic activity, that it reaches or affects gonadal organs, and the extent and pattern of human exposure to the chemical, must be examined. From these elements a judgment as to the degree of human risk may be derived. When appropriate mutagenicity data are available for making a quantitative estimate of genetic risk to humans, two major approaches are available. One approach uses only experimental data on germinal alterations induced in intact mammals. Test systems providing such data are the heritable translocation, X-chromosome loss, and specific locus tests. These tests are complete in that they directly measure genetic damage in germ cells which is observed in a subsequent generation. In this approach, information on the level of human exposure is used with experimental data on induced mutation frequency, usually obtained at much higher exposure levels. An estimate of human risk is obtained by extra polating the induced mutation frequency downward to the approximate level of anticipated human exposure. With point mutational effects, there is a strong theoretical basis for the use of the linear or no-threshold models for this extrapolation (10, 11, 12). For certain chromosomal alterations , the biological mechanisms are generally thought to fit a multiple-hit model. Other mathematical models for extrapolation should, therefore, be considered when the dose-response data are able to fit an appropriate model. Linear extrapolation of the experimental data on translocations, for example, are likely to overestimate the risks present at lower levels of exposure and a model other than the one-hit model should be considered.

The other experimental approach for assessing genetic risk uses biochemical data from intact mammals and mutagenicity data from validated test systems (13, 14, 15). The intact mammal is used primarily for relating the exposure level for a chemical to germ cell dose. This involves relating the level of mutagen-DNA interactions in germ cells to the specific exposure level by an appropriate route of administration. This information is then used with results obtained from mutagenicity test systems in which the relationship between the induction of mutations and interactions with DNA can be made. Using mutagen-DNA interactions as the common denominator, a relationship can be constructed between exposure and the induced mutation frequency. In some cases, measurements of DNA binding induced by a particular chemical agent can be determined at levels of exposures which may affect human populations, and potential human risk can be determined without the extrapolation of the data to lower levels. Such results would not require a high to low exposure level extrapolation to determine potential human risk. In those instances where the lowest experimental exposure level is greatly above those levels to which humans are exposed, it will be assumed that DNA binding is directly proportional to the exposure level unless there are compelling reasons to believe otherwise.

For some mutagenic events, DNA may not necessarily be the critical target. Interaction of chemicals with other macromolecules, such as tubulin which is involved in the separation of chromosomes during nuclear division, can lead to chromosomal nondisjunction. At present the means are not available to make strictly quantitative assessments for these types of mutagenic mechanisms. Ongoing research should provide the means to make future quantitative risk assessments for mutagens acting through these mechanisms.

In performing mutagenicity risk assessments, it is important to account for the sensitivity of the tests in detecting specific mutagenic endpoints. For example, although chemical substances which interact with DNA are likely to cause both point and chromosomal mutations, it is expected that point mutations may be relatively more frequent than chromosomal mutations at lower exposure levels. The former event can be initiated by a single interaction with DNA, whereas the latter may involve more than one interaction. Therefore, when assessing the mutagenic risk associated with low human exposures to the chemical, the point mutation data would generally be the more sensitive and appropriate information source.

Any risk assessment should clearly delineate the strengths and weakness of assumptions made, the uncertainties in the methodology, and the rationale used in reaching the conclusion, e.g., similar or different routes of exposure, metabolic difference between humans and test animals. The results should be expressed in terms of the excess of genetic disease per year or per lifetime, or the fractional increase in the assumed background spontaneous mutation rate of humans, or the radiation equivalency.

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Dated: November 4, 1980.

Douglas M. Costle,

Administrator.

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[OPP-180523; PH-FRL 1668-2]

New York; Issuance of Specific Exemption for Fenvalerate on Cabbage

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has granted a specific exemption to the New York Department of Environmental Protection (hereinafter referred to as the "Applicant") to use fenvalerate (Pydrin) on 12,000 acres of cabbage in New York to control the cabbage looper. The specific exemption is issued under the Federal Insecticide, Fungicide, and Rodenticide Act.

DATE: The specific exemption expires on November 1, 1980.

FOR FURTHER INFORMATION CONTACT: Libby Welch, Registration Division (TS-767), Office of Pesticide Programs, Environmental Protection Agency, Rm. E-124, 401 M St., SW., Washington, D.C. 20460, (202-426-0223).

SUPPLEMENTARY INFORMATION: The Applicant anticipates that an emergency situation will develop with respect to cabbage loopers on cabbage in upstate counties of New York. The Applicant contends that if the cool weather sets in during the last 35 days of cabbage growth, the cabbage looper will not be controllable. The Applicant claims that use of the pesticides registered for control of the cabbage looper is not practicable because:

1. The pesticide Phosdrin is not very effective against the pest and its residual action is short.

2. The pesticide Diazinon has not been effective in New York for many years.

3. Methomyl and *Bacillus thuringiensis* do not control the cabbage looper when temperatures are low.

4. While Monitor generally controls the pest, it may not be used within 35 days of harvest. This 35-day preharvest interval is a critical period for cabbage due to the unusually high infestation of the cabbage looper.

The Applicant estimates that the cabbage industry in New York could lose up to $12 million without adequate control.

The Applicant proposes to use fenvalerate at a rate of 0.1 to 0.2 pounds active ingredient (a.i.) per acre on up to 12,000 acres of cabbage. More than 50 percent of the fields checked in a county would have to be infested with cabbage loopers in numbers greater than one to two loopers per plant, as determined by research on Cooperative Extension personnel. Application would be made only when anticipated average daily temperature during the spray period is below 8° F during the day and/or 55–60° F at night.

EPA has determined that residues of fenvalerate in or cabbage should not exceed 2 parts per million (ppm) from the proposed use. EPA has judged this level to be adequate to protect the public health. No unreasonable hazard to the environment is anticipated from this program. Since fenvalerate is highly toxic to aquatic vertebrates and invertebrates and to bees, appropriate conditions to protect them having been imposed.

After reviewing the application and other available information, EPA has determined that the criteria for an exemption have been met. Accordingly, the Applicant has been granted a specific exemption to use the pesticide

United States
Environmental Protection
Agency

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EPA

Research and Development

THE CARCINOGEN ASSESSMENT GROUP'S METHOD FOR DETERMINING THE UNIT RISK ESTIMATE FOR AIR POLLUTANTS

Prepared for

Office of Air Quality Planning and Standards
Office of Air, Noise, and Radiation
U.S. Environmental Protection Agency

Prepared by
Carcinogen Assessment Group
Office of Health and
Environmental Assessment
Washington DC 20460

THE CARCINOGEN ASSESSMENT GROUP'S
METHOD FOR DETERMINING THE UNIT RISK ESTIMATE
FOR AIR POLLUTANTS

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August 8, 1980

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NOTICE

The Carcinogen Assessment Group (CAG), located in the Office of Health and Environmental Assessment of EPA's Office of Research and Development, is a small group of scientists who perform an advisory assessment function for EPA's regulatory offices. The CAG analyzes existing scientific data and furnishes the regulatory offices with an evaluation of the carcinogenicity and levels of carcinogenic risk associated with chemicals in various exposure situations, as best can be determined from currently available scientific data.

The CAG reports are prepared for internal Agency use in response to EPA's regulatory office needs. They range from brief chemical profiles to very extensive evaluations, depending upon the nature of a request. The reports are used by the regulatory offices for regulatory decision making as appropriate. The reports are revised and edited based on regulatory office needs and the availability of resources.

This document was prepared at the request of the EPA Office of Air Quality Planning and Standards.

METHOD FOR DETERMINING THE UNIT RISK ESTIMATE FOR AIR POLLUTANTS

INTRODUCTION

The unit risk estimate for an air pollutant is defined as the lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1 ug/m^3 of the agent in the air which they breathe. This calculation is done to estimate in quantitative terms the affectiveness (potency) of the agent as a carcinogen. Unit risk estimates are used for two purposes: 1) to compare the carcinogenic potency of several agents with each other, and 2) to give a crude indication of the population risk which might be associated with air exposure to these agents, if the actual exposures are known. These two uses have different limitations.

In order to use these estimates intelligently, the nature of the source data used and the assumptions necessary to derive the estimates must be clearly understood. This appendix discusses the general approach and the assumptions common to most unit risk estimates. The credence one can ascribe to each risk estimate depends heavily on the quality of the studies on which the estimate is based and the relevance of these studies to the evaluation of air exposure in humans.

PROCEDURES FOR DETERMINATION OF UNIT RISK BASED ON BIOASSAY DATA

The data used for the quantitative estimate is one or both of two types: 1) lifetime animal studies, and 2) human studies where excess cancer risk has been associated with exposure to the agent. In animal studies it is assumed, unless evidence exists to the contrary, that if a carcinogenic response occurs at the dose levels used in the study, then responses will also occur at all lower doses with an incidence determined by the extrapolation model discussed below.

A. Choice of Model

There is no really solid scientific basis for any mathematical extrapolation model which relates carcinogen exposure to cancer risks at the extremely low concentrations that must be dealt with in evaluating environmental hazards. For practical reasons such low levels of risk cannot be measured directly either by animal experiments or by epidemiologic studies. We must, therefore, depend on our current understanding of the mechanisms of carcinogens for guidance as to which risk model to use. At the present time the dominant view of the carcinogenic process involves the concept that most agents which cause cancer also cause irreversible damage to DNA. This position is reflected by the fact that a very large proportion of agents which cause cancer are also mutagenic. There is reason to expect the quantal type of biological response that is characteristic of mutagenesis is associated with a linear non-threshold dose-response relationship. Indeed, there is substantial evidence from mutagenesis studies with both ionizing radiation and a wide variety of chemicals that this type of dose-response model is the appropriate one to use. This is particularly true at the lower end of the dose-response curve; at higher doses, there can be an upward curvature probably reflecting the effects of multistage processes on the mutagenic response. The linear non-threshold dose-response relationship is also consistent with the relatively few epidemiological studies of cancer responses to specific agents that contain enough information to make the evaluation possible (e.g., radiation induced leukemia, breast and thyroid cancer, skin cancer induced by arsenic in drinking water, liver cancer induced by aflatoxin in the diet). There is also some evidence from animal experiments that is consistent with the linear non-threshold model (e.g., liver tumors induced in mice by 2-acetylaminofluorene in the large scale ED_{01} study at the National Center for Toxicological Research and the initiation stage of the two-stage carcinogenesis model in rat liver and mouse skin).

Because it has the best, albeit limited, scientific basis of any of the current mathematical extrapolation models, the linear non-threshold model has been adopted as the primary basis for risk extrapolation to low levels of the dose-response relationship. The risk estimates made with this model should be regarded as conservative, representing the most plausible upper-limit for the risk, i.e., the true risk is not likely to be higher than the estimate, but it could be smaller.

The mathematical formulation chosen to describe the linear, non-threshold dose-response relationship at low doses is the improved multistage model developed by Dr. K. Crump. This model employs enough arbitrary constants to be able to fit almost any monotonically increasing dose-response data and it incorporates a procedure for estimating the largest possible linear slope (in the 95% confidence limit sense) at low extrapolated doses that is consistent with the data at all dose levels of the experiment.

B. Description of the Low Dose Extrapolation Model

Let P(d) represent the lifetime risk (probability) of cancer at dose d. The multistage model has the form

$$P(d) = 1 - \exp\left[-(q_0 + q_1 d + q_2 d^2 + \ldots + q_k d^k)\right]$$

where

$$q_i \geq 0, \; i = 0, 1, 2, \ldots, k$$

$$A(d) = 1 - \exp\left[-(q_1 d + q_2 d^2 + \ldots + q_k d^k)\right]$$

where

$$A(d) = \frac{P(d) - P(o)}{1 - P(o)}$$

is the extra risk over background rate at dose d.

The point estimate of the coefficients q_i, $i = 0, 1, 2, \ldots, k$ and consequently the extra risk function A(d) at any given dose d is calculated by maximizing the likelihood function of the data.

The point estimate and the 95% upper confidence limit of the extra risk A(d) are calculated by using the computer program GLOBAL 79 developed by Crump and Watson (1979). At low doses, upper 95% confidence limits on the extra risk and lower 95% confidence limits on the dose producing a given risk are determined from a 95% upper confidence limit, q_1^*, on a parameter q_1. Whenever $q_1 > 0$, at low doses the extra risk A(d) has approximately the form $A(d) = q_1 \times d$. Therefore, $q_1 \times d$ is a 95% upper confidence limit on the extra risk and R/q_1^* is a 95% lower confidence limit on the dose producing an extra risk of R. Let L_0 be the maximum value of the log-likihood function. The upper limit q_1 is calculated by increasing q_1 to a value q_1^* such that when the log-likihood is remaximized subject to this fixed value q_1^* for the linear coefficient, the resulting maximum value of the log-likihood L_1 satisfies the equation

$$2\,(L_0 - L_1) = 2.70554$$

where 2.70554 is the cumulative 90% point of the chi-square distribution with one degree of freedom, which corresponds to a 95% upper limit (one-sided). This approach of cumputing the upper confidence limit for the extra risk A(d) is an improvement on the Crump et al. (1977) model. The upper confidence limit for the extra risk calculated at low doses is always linear. This is conceptually consistent with the linear non-threshold concept discussed earlier. The slope q_1^* is taken as an upper bound of the potency of the chemical in inducing cancer at low doses.

In fitting the dose-response model, the number of terms in the polynomial is chosen equal to (h-1) where h is the number of dose groups in the experiment including the control group.

Whenever the multistage model does not fit the data sufficiently well, data at the highest dose is deleted and the model is refitted to the rest of the data. This is continued until an acceptable fit to the data is obtained. To determine whether or not a fit is acceptable, the chi-square statistic

$$X^2 = \sum_{i=1}^{h} \frac{(X_i - N_i P_i)^2}{N_i P_i (1 - P_i)}$$

is calculated where N_i is the number of animals in the i^{th} dose group, X_i is the number of animals in the i^{th} dose group with a tumor response, P_i is the probability of a response in the i^{th} dose group estimated by fitting the multistage model to the data, and h is the number of remaining groups. The fit is determined to be unacceptable whenever X^2 is larger than the cumulative 99% point of the chi-square distribution with f degree of freedom, where f equals the number of dose groups minus the number of non-zero multistage coefficients.

SELECTION OF DATA USED TO ESTIMATE PARAMETERS IN THE EXTRAPOLATION MODEL AND HUMAN EQUIVALENCE DOSE CONVERSION

For some chemicals, several studies in different animal species, strains, and sexes each run at several doses and different routes of exposure are available. A choice must be made of which of the data sets from several studies to use in the model. It is also necessary to correct for metabolism differences between species and absorption factors via different routes of administration. The procedures used in evaluating these data are consistent with the approach of making a maximum-likely risk estimate. They are listed below.

1. The tumor incidence data are separated according to organ sites or tumor types. The set of data (i.e., dose and tumor incidence) used in the model is the set where the incidence is statistically significantly higher than the control for at least one test dose level and/or where the tumor incidence rate shows a statistically significant trend with respect to dose level. The data set which gives the highest estimate of the lifetime carcinogenic risk q_1^* is selected in most cases. However, efforts are made to exclude data sets which produce spuriously high risk estimates because of a small number of animals. That is, if two sets of data show a similar dose-response relationship and one has a very small sample size, the set of data which has larger sample size is selected for calculating the carcinogenic potency.

2. If there are two or more data sets of comparable size which are identical with respect to species, strain, sex, and tumor sites, the geometric mean of q_1^*, estimated from each of these data sets, is used for risk assessment. The geometric mean of numbers A_1, A_2, ..., A_m is defined as

$$(A_1 \times A_2 \times \ldots \times A_m)^{1/m}$$

3. If two or more significant tumor sites are observed in the same study and if the data are available, the number of animals with at least one of the specific tumor sites under consideration is used as incidence data in the model.

4. Following the suggestion of Mantel and Schneiderman (1977), we assume that mg/surface area/day is an equivalent dose between species. Since, to a close approximation, the surface area is proportional to the 2/3 rds power of the weight as would be the case for a perfect sphere, the exposure in mg/day per 2/3 rds power of the weight is also considered to be an equivalent exposure. In an animal experiment this equivalent dose is computed in the following manner. Let

L_e = duration of experiment

l_e = duration of exposure

m = average dose per day in mg during administration of the agent (i.e., during l_e)

W = average weight of the experimental animal

Then, the lifetime average exposure is

$$d = \frac{l_e \times m}{L_e \times W^{2/3}}$$

Often exposures are not given in units of mg/day and it becomes necessary to convert the given exposures into mg/day. For example, in most feeding studies exposure is in terms of ppm in the diet. In this case the exposure in mg/day is

$$m = \text{ppm} \times F \times r$$

where ppm is parts per million in the diet of the carcinogenic agent, F is the weight of the food consumed per day in kg, and r is the absorption fraction. In the absence of any data to the contrary, r is assumed to be equal to one. For a uniform diet, the weight of the food consumed is proportional to the calories required which, in turn, is proportional to the surface area or 2/3rds power of the weight, so that

$$m \propto ppm \times W^{2/3} \times r$$

or

$$\frac{m}{rW^{2/3}} \propto ppm$$

As a result, ppm in the diet is often assumed to be an equivalent exposure between species. However, we feel that this is not justified since the calories/kg of food is very different in the diet of man compared to laboratory animals primarily due to moisture content differences. Instead we use an empirically derived food factor $f = F/W$ which is the fraction of a species body weight that is consumed per day as food. We use the rates given below.

Species	W	f
Man	70	0.028
Rat	0.35	0.05
Mice	0.03	0.13

Thus, when the exposure is given as a certain dietary concentration in ppm the exposure in $mg/W^{2/3}$ is

$$\frac{m}{r \times W^{2/3}} = \frac{ppm \times F}{W^{2/3}} = \frac{ppm \times f \times W}{W^{2/3}} = ppm \times f \times W^{1/3}$$

When exposure is given in terms of mg/kg/day = $m/Wr = s$, the conversion is simply

$$\frac{m}{rW^{2/3}} = s \times W^{1/3}$$

When exposure is via inhalation, the calculation of dose can be considered for two cases where 1) the carcinogenic agent is either a completely water soluble gas or an aerosal, and is absorbed proportionally to the amount of air breathed in, and 2) where the carcinogen is a poorly water soluble gas which reaches an equilibrium between the air breathed and the body compartments. After equilibrium is reached, the rate of absorption of these agents is expected to be proportional to the metabolic rate which, in turn, is proportional to the rate of oxygen consumption which, in turn, is a function of surface area.

Case 1

Agents that are in the form of particulate matter or virtually completely absorbed gases such as SO_2 can reasonably be expected to be absorbed proportional to the breathing rate. In this case the exposure in mg/day may be expressed as

$$m = I \times v \times r$$

where I = inhalation rate per day in m^3, v = mg/m^3 of the agent in air, and r = the absorption fraction.

The inhalation rates, I, for various species can be calculated from the observations (FASEB 1974) that 25 g mice breathe 34.5 liters/day and 113 g rats breathe 105 liters/day. For mice and rats of other weights, W (in kilograms), the surface area proportionality can be used to find breathing rates in m^3/day as follows:

$$\text{For mice, } I = 0.0345\ (W/0.025)^{2/3}\ m^3/\text{day}$$

$$\text{For rats, } I = 0.105\ (W/0.113)^{2/3}\ m^3/\text{day}$$

For humans, the values of 20 m^3/day* is adopted as a standard breathing rate (ICRP 1977).

The equivalent exposure in mg/$W^{2/3}$ for these agents can be derived from the air intake data in way analogous to the food intake data. The empirical factors for the air intake per kg per day, $i = I/W$ based upon the previous stated relationships are tabulated below.

Species	W	i = I/W
Man	70	0.29
Rat	0.35	0.64
Mice	0.03	1.3

Therefore, for particulates or completely absorbed gases, the equivalent exposure in mg/$W^{2/3}$ is

$$d = \frac{m}{W^{2/3}} = \frac{Ivr}{W^{2/3}} = \frac{iWvr}{W^{2/3}} = iW^{1/3}\ vr$$

In the absence of experimental information or a sound theoretical argument to the contrary, the fraction absorbed, r, is assumed to be the same for all species.

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*From "Recommendation of the International Commission on Radiological Protection," page 9. The average breathing rate is 10^7 cm^3 per 8-hour workday and 2×10^7 cm^3 in 24 hours.

Case 2

The dose in mg/day of partially soluble vapors is proportional to the O_2 consumption, which, in turn, is proportional to $W^{2/3}$ and is also proportional to the solubility of the gas in body fluids, which can be expressed as an absorption coefficient r for the gas. Therefore, expressing the O_2 consumption as $O_2 = k\ W^{2/3}$ where k is a constant independent of species, it follows that

$$m = k\ W^{2/3} \times v \times r$$

or

$$d = \frac{m}{W^{2/3}} = kvr$$

As with Case 1, in the absense of experimental information or a sound theoretical argument to the contrary, the absorption fraction, r, is assumed to be the same for all species. Therefore, for these substances a certain concentration in ppm or ug/m^3 in experimental animals is equivalent to the same concentration in humans. This is supported by the observation that the minimum alveolar concentration necessary to produce a given "stage" of anesthesia is similar in man and animals (Dripps et al. 1977). When the animals are exposed via the oral route and human exposure is via inhalation or vice-versa, the assumption is made, unless there is pharmacokinetic evidence to the contrary, that absorption is equal by either exposure route.

5. If the duration of experiment (Le) is less than the natural lifespan of the test animal (L), the slope q_1^*, or more generally the exponent g(d), is increased by multiplying a factor $(L/L_e)^3$. We assume that if the average dose, d, is continued, the age-specific rate of cancer will continue to increase as a constant function of the background rate. The age-specific rates for humans increases at least by the 2nd power of the age and often by a considerably higher power as demonstrated by Doll (1971). Thus, we would expect

the cumulative tumor rate to increase by at least the 3rd power of age. Using this fact, we assume that the slope q_1^*, or more generally the exponent g(d), would also increase by at least the 3rd power of age. As a result, if the slope q_1^* [or g(d)] is calculated at age L_e, we would expect that if the experiment had been continued for the full lifespan, L, at the given average exposure, the slope q_1^* [or g(d)] would have been increased by at least $(L/L_e)^3$.

This adjustment is conceptually consistent to the proportional hazard model proposed by Cox (1972) and the time-to-tumor model considered by Crump et al. (1979) where the probability of cancer by age t and at dose d is given by

$$P(d,t) = 1 - \exp[-f(t) \times g(d)]$$

CALCULATION OF THE UNIT RISK

The risk associated with d mg/kg$^{2/3}$/day is obtained from GLOBAL 79 and, for most cases of interest to risk assessment, can be adequately approximated by $P(d) = 1 - \exp(-q_1^*d)$. A "unit risk" in units X is simply the risk corresponding to an exposure of X = 1. To estimate this value we simply find the number of mg/kg$^{2/3}$/day corresponding to one unit of X and substitute this value into the above relationship. Thus, for example, if X is in units of ug/m^3 in the air, we have that for case (1) $d = 0.29 \times 70^{1/3} \times 10^{-3}$ mg/kg$^{2/3}$/day and for case (2) d = 1, when ug/m^3 is the unit used to compute parameters in animal experiment.

If exposures are given in terms of ppm in air we may simply use the fact that

$$1 \text{ ppm} = 1.2 \times \frac{\text{molecular weight (gas)}}{\text{molecular weight (air)}} \text{ mg/m}^3$$

Note, an equivalent method of calculating unit risk would be to use mg/kg for the animal exposures and then increase the jth polynomial coefficient by an amount

$$(W_h/W_a)^{j/3} \qquad j = 1, 2, \ldots, k$$

and use mg/kg equivalents for the unit risk values.

ESTIMATION OF UNIT RISK BASED ON HUMAN DATA

If human epidemiology studies and sufficiently valid exposure information are available for the compound, they are always used in some way. If they show a carcinogenic effect, the data are analyzed to give an estimate of the linear dependence of cancer rates on lifetime average dose, which is equivalent to the factor B_H. If they show no carcinogenic effect when positive animal evidence is available, then it is assumed that a risk does exist but it is smaller than could have been observed in the epidemiology study, and an upper-limit of the cancer incidence is calculated assuming hypothetically that the true incidence is just below the level of detection in the cohort studied, which is determined largely by the cohort size. Whenever possible, human data are used in preference to animal bioassay data.

In human studies, the response is measured in terms of the relative risk of the exposed cohort of individuals compared to the control group. In the analysis of this data it is assumed that the excess risk, or relative risk minus one, $R(X_1) - 1$, is proportional to the lifetime average exposure, X_1, and that it is the same for all ages. It follows that the lifetime risk in the general population exposed to a lifetime average concentration X_2, $P(X_2)$, is equal to $[R(X_1) - 1]X_2/X_1$ multiplied by the lifetime risk at that site in the general population. The unit risk estimate is the value of P when X_2 is 1 ug/m^3. Except for an unusually well-documented human study, the confidence limit for the excess risk P is not calculated, due to the difficulty of accounting for the uncertainty inherent in the data (exposure and cancer response).

INTERPRETATION OF UNIT RISK

The unit risk estimate is a rough indication of the relative potency of a given agent compared with other carcinogens. The comparative potency of different agents is more reliable when the comparison is based on studies in the same test species, strain, and sex and by the same route of exposure, preferably by inhalation.

For several reasons the unit risk estimate is only an approximate indication of the absolute risk in populations exposed to known air concentrations. First, there are important species differences in uptake, metabolism, and organ distribution of carcinogens, as well as species differences in target site susceptibility, immunological responses, hormone function, dietary factors, and disease. Secondly, the concept of equivalent doses for humans compared to animals on a mg/surface area basis is virtually without experimental verification regarding carcinogenic response. Finally, human populations are variable with respect to genetic constitution and diet, living environment, activity patterns, and other cultural factors.

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Part V

Environmental Protection Agency

Water Quality Criteria Documents; Availability

ENVIRONMENTAL PROTECTION AGENCY

[FRL 1623-3]

Water Quality Criteria Documents; Availability

AGENCY: Environmental Protection Agency.

ACTION: Notice of Water Quality Criteria Documents.

SUMMARY: EPA announces the availability and provides summaries of water quality criteria documents for 64 toxic pollutants or pollutant categories. These criteria are published pursuant to section 304(a)(1) of the Clean Water Act.

AVAILABILITY OF DOCUMENTS: Summaries of both aquatic-based and health-based criteria from the documents are published below. Copies of the complete documents for individual pollutants may be obtained from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161, (703-487-4650). A list of the NTIS publication order numbers for all 64 criteria documents is published below. These documents are also available for public inspection and copying during normal business hours at: Public Information Reference Unit, U.S. Environmental Protection Agency, Room 2404 (rear), 401 M St., S.W., Washington, D.C. 20460. As provided in 40 CFR Part 2, a reasonable fee may be charged for copying services. Copies of these documents are also available for review in the EPA Regional Office libraries.

Copies of the documents are not available from the EPA office listed below. Requests sent to that office will be forwarded to NTIS or returned to the sender.

1. Acenaphthene, PB81-117269.
2. Acrolein, PB81-117277.
3. Acrylonitrile, PB81-117285.
4. Aldrin/Dieldrin, PB81-117301.
5. Antimony, PB81-117319.
6. Arsenic, PB81-117327.
7. Asbestos, PB81-117335.
8. Benzene, PB81-117293.
9. Benzidine, PB81-117343.
10. Beryllium, PB81-117350.
11. Cadmium, PB81-117368.
12. Carbon Tetrachloride, PB81-117376.
13. Chlordane, PB81-117384.
14. Chlorinated benzenes, PB81-117392.
15. Chlorinated ethanes, PB81-117400.
16. Chloroalkyl ethers, PB81-117418.
17. Chlorinated naphthalene, PB81-117426.
18. Chlorinated phenols, PB81-117434.
19. Chloroform, PB81-117442.
20. 2-chlorophenol, PB81-117459.
21. Chromium, PB81-117467.
22. Copper, PB81-117475.
23. Cyanides, PB81-117483.
24. DDT, PB81-117491.
25. Dichlorobenzenes, PB81-117509.
26. Dichlorobenzidine, PB81-117517.
27. Dichloroethylenes, PB81-117525.
28. 2,4-dichlorophenol, PB81-117533.
29. Dichloropropanes/propenes, PB81-117541.
30. 2,4-dimethylphenol, PB81-117558.
31. Dinitrotoluene, PB81-117566.
32. Diphenylhydrazine, PB81-117731.
33. Endosulfan, PB81-117574.
34. Endrin, PB81-117582.
35. Ethylbenzene, PB81-117590.
36. Fluoranthene, PB81-117608.
37. Haloethers, PB81-117616.
38. Halomethanes, PB81-117624.
39. Heptachlor, PB81-117632.
40. Hexachlorobutadiene, PB81-117640.
41. Hexachlorocyclohexane, PB81-117657.
42. Hexachlorocyclopentadiene, PB81-117665.
43. Isophorone, PB81-117673.
44. Lead, PB81-117681.
45. Mercury, PB81-117699.
46. Naphthalene, PB81-117707.
47. Nickel, PB81-117715.
48. Nitrobenzene, PB81-117723.
49. Nitrophenols, PB81-117749.
50. Nitrosamines, PB81-117756.
51. Pentachlorophenol, PB81-117764.
52. Phenol, PB81-117772.
53. Phthalate esters, PB81-117780.
54. Polychlorinated biphenyls (PCBs), PB81-117798.
55. Polynuclear aromatic hydrocarbons, PB81-117806.
56. Selenium, PB81-117814.
57. Silver, PB81-117822.
58. Tetrachloroethylene, PB81-117830.
59. Thallium, PB81-117848.
60. Toluene, PB81-117855.
61. Toxaphene, PB81-117863.
62. Trichloroethylene, PB81-117871.
63. Vinyl chloride, PB81-117889.
64. Zinc, PB81-117897.

FOR FURTHER INFORMATION CONTACT: Dr. Frank Gostomski, Criteria and Standards Division (WH-585), United States Environmental Protection Agency, 401 M Street, S.W., Washington, D.C. 20460, (202) 245-3042.

SUPPLEMENTARY INFORMATION:

Background

Pursuant to section 304(a)(1) of the Clean Water Act, 33 U.S.C. 1314(a)(1), EPA is required to periodically review and publish criteria for water quality accurately reflecting the latest scientific knowledge:

> (A) on the kind and extent of all identifiable effects on health and welfare including, but not limited to, plankton, fish, shellfish, wildlife, plant life, shorelines, beaches, esthetics, and recreation which may be expected from the presence of pollutants in any body of water, including groundwater, (B) on the concentration and dispersal of pollutants, or their byproducts, through biological, physical, and chemical processes, and (C) on the effects of pollutants on biological community diversity, productivity, and stability, including information on the factors affecting rates of eutrophication and rates of organic and inorganic sedimentation for varying types of receiving waters.

EPA is today announcing the availability of criteria documents for 64 of the 65 pollutants designated as toxic under section 307(a)(1) of the Act. The document on TCDD (Dioxin) will be published within the next month after review of recent studies. Criteria for the section 307(a)(1) toxic pollutants being published today will replace the criteria for those same pollutants found in the EPA publication, *Quality Criteria for Water,* (the "Red Book.") Criteria for all other pollutants and water constituents found in the "Red Book" remain valid. The criteria published today have been derived using revised methodologies for determining pollutant concentrations that will, when not exceeded, reasonably protect human health and aquatic life. Draft criteria documents were made available for public comment (44 FR 15926, March 15, 1979, 44 FR 43660, July 25, 1979, 44 FR 56628, October 1, 1979). These final criteria have been derived after consideration of all comments received.

These criteria documents are also issued in satisfaction of the Settlement Agreement in *Natural Resources Defense Council, et al.* v. *Train,* 8 E.R.C. 2120 (1976), modified, 12 E.R.C. 1833 (D.D.C. 1979). Pursuant to paragraph 11 of that agreement, EPA is required to publish criteria documents for the 65 pollutants which Congress, in the 1977 amendments to the Act, designated as toxic under section 307(a)(1). These documents contain recommended maximum permissible pollutant concentrations consistent with the protection of aquatic organisms, human health, and some recreational activities. Although paragraph 11 imposes certain obligations on the Agency, it does not create additional authority.

The Development of Water Quality Criteria

Section 304(a)(1) criteria contain two essential types of information: (1) discussions of available scientific data on the effects of pollutants on public health and welfare, aquatic life and recreation, and (2) quantitative concentrations or qualitative assessments of the pollutants in water which will generally ensure water quality adequate to support a specified water use. Under section 304(a)(1), these criteria are based solely on data and scientific judgments on the relationship between pollutant concentrations and environmental and human health effects. Criteria values do not reflect considerations of economic or technological feasibility.

Publication of water quality criteria of this type has been an ongoing process which EPA, and its predecessor Agency, the Federal Water Pollution Control Administration, have been engaged in since 1968. At that time the first Federal compilation of water quality criteria, the so-called "Green Book" *(Water Quality Criteria)*, was published. As now, these criteria contained both narrative discussions of the environmental effects of pollutants on a range of possible uses and concentrations of pollutants necessary to support these uses. Since that time, water quality criteria have been revised and expanded with publication of the "Blue Book" *(Water Quality Criteria 1972)* in 1973 and the "Red Book" *(Quality Criteria for Water)* in 1976.

Since publication of the Red Book there have been substantial changes in EPA's approach to assessing scientific data and deriving section 304(a)(1) criteria. Previous criteria were derived from a limited data base. For many pollutants, an aquatic life criterion was derived by multiplying the lowest concentration known to have acute lethal effect on half of a test group of an aquatic species (the LC50 value) by an application factor in order to protect against chronic effects. If data showed a substance to be bioaccumulative or to have other significant long-term effects, a factor was used to reduce the indicated concentrations to a level presumed to be protective. Criteria for the protection of human health were similarly derived by considering the pollutants' acute, chronic, and bioaccumulative effects on non-human mammals and humans.

Although a continuation of the process of criteria development, the criteria published today were derived using revised methodologies (Guidelines) for calculating the impact of pollutants on human health and aquatic organisms. These Guidelines consist of systematic methods for assessing valid and appropriate data concerning acute and chronic adverse effects of pollutants on aquatic organisms, non-human mammals, and humans. By use of these data in prescribed ways, criteria are formulated to protect aquatic life and human health from exposure to the pollutants. For some pollutants, bioconcentration properties are used to formulate criteria protective of aquatic life uses. For almost all of the pollutants, bioconcentration properties are used to assess the relative extent of human exposure to the pollutant either directly through ingestion of water or indirectly through consumption of aquatic organisms. Human health criteria for carcinogens are presented as incremental risks to man associated with specific concentrations of the pollutant in ambient water. The Guidelines used to derive criteria protective of aquatic life and human health are fully described in appendices B and C, respectively, of this Notice.

The Agency believes that these Guidelines provide criteria which more accurately reflect the effects of these pollutants on human health and on aquatic organisms and their uses. They are based on a more rational and consistent approach for using scientific data. These Guidelines were developed by EPA scientists in consultation with scientists from outside the Agency and they have been subjected to intensive public comment.

Neither the Guidelines nor the criteria are considered inflexible doctrine. Even at this time, EPA is taking action to employ the resources of peer review groups, including the Science Advisory Board, to evaluate recently published data, and EPA is conducting its own evaluation of new data to determine whether revisions to the criteria documents would be warranted.

The criteria published today are based solely on the effect of a single pollutant. However, pollutants in combination may have different effects because of synergistic, additive, or antagonistic properties. It is impossible in these documents to quantify the combined effects of these pollutants, and persons using criteria should be aware that site-specific analysis of actual combinations of pollutants may be necessary to give more precise indications of the actual environmental impacts of a discharge.

Relationship of the Section 304(a)(1) Criteria to Regulatory Programs

Section 304(a)(1) criteria are not rules and they have no regulatory inpact. Rather, these criteria present scientific data and guidance on the environmental effect of pollutants which can be useful to derive regulatory requirements based on considerations of water quality impacts. Under the Clean Water Act, these regulatory requirements may include the promulgation of water quality-based effluent limitations under section 302, water quality standards under section 303, or toxic pollutant effluent standards under section 307. States are encouraged to begin to modify or, if necessary, develop new programs necessary to support the implementation of regulatory controls for toxic pollutants. As appropriate, States may incorporate criteria for toxic pollutants, based on this guidance, into their water quality standards.

Section 304(a)(1) criteria have been most closely associated with the development of State water quality standards, and the "Red Book" values have, in the past, been the basis for EPA's assessments of the adequacy of State requirements. However, EPA is now completing a major review of its water quality standards policies and regulations. After consideration of comments received on an Advance Notice of Proposed Rulemaking (43 FR 29588, July 10, 1978) and the draft criteria documents, the Agency intends to propose, by the end of this year, a revised water quality standards regulation which will clarify the Agency's position on a number of significant standards issues.

With the publication of these criteria, however, it is appropriate to discuss EPA's current thinking on standards issues relating to their use. This discussion does not establish new regulatory requirements and is intended as guidance on the possible uses of these criteria and an indication of future rulemaking the Agency may undertake. No substantive requirements will be established without further opportunity for public comment.

Water Quality Standards

Section 303 of the Clean Water Act provides that water quality standards be developed for all surface waters. A water quality standard consists basically of two parts: (1) A "designated use" for which the water body is to be protected (such as "agricultural," "recreation" or "fish and wildlife"), and (2) "criteria" which are numerical pollutant concentration limits or narrative statements necessary to preserve or achieve the designated use. A water quality standard is developed through State or Federal rulemaking proceedings and must be translated into enforceable effluent limitations in a point source (NPDES) permit or may form the basis of best management practices applicable to nonpoint sources under section 208 of the Act.

Relationship of Section 304(a)(1) Criteria to the Criteria Component of State Water Quality Standards:

In the ANPRM, EPA announced a policy of "presumptive applicability" for section 304(a)(1) criteria codified in the "Red Book." Presumptive applicability meant that a State had to adopt a criterion for a particular water quality parameter at least as stringent as the recommendation in the Red Book unless the State was able to justify a less stringent criterion based on: natural background conditions, more recent scientific evidence, or local, site-specific information. EPA is rescinding the policy of presumptive applicability because it has proven to be too inflexible in actual practice.

Although the section 304(a)(1) criteria represent a reasonable estimate of pollutant concentrations consistent with the maintenance of designated water uses, States may appropriately modify these values to reflect local conditions. In certain circumstances, the criteria may not accurately reflect the toxicity of a pollutant because of the effect of local water quality characteristics or varying sensitivities of local populations. For example, in some cases, ecosystem adaptation may enable a viable, balanced aquatic population to exist in waters with high natural background levels of certain pollutants. Similarly, certain compounds may be more or less toxic in some waters because of differences in alkalinity, temperature, hardness, and other factors.

Methods for adjusting the section 304(a)(1) criteria to reflect these local differences are discussed below.

Relationship of Section 304(a)(1) Criteria to Designated Water Uses:

The criteria published today can be used to support the designated uses which are generally found in State standards. The following section discusses the relationship between the criteria and individual use classifications. Where a water body is designated for more than one use, criteria necessary to protect the most sensitive use should be applied.

1. *Recreation.* Recreational uses of water include such activities as swimming, wading, boating and fishing. Although insufficient data exist on the effects of toxic pollutants resulting from exposure through such primary contact as swimming, section 304(a)(1) criteria based on human health effects may be used to support this designated use where fishing is included in the State definition of "recreation." In this situation only the portion of the criterion based on fish consumption should be used.

2. *Protection and Propagation of Fish and Other Aquatic Life:* The section 304(a)(1) criteria based on toxicity to aquatic life may be used directly to support this designated use.

3. *Agricultural and Industrial Uses:* The section 304(a)(1) criteria were not specifically developed to reflect the impact of pollutants on agricultural and industrial uses. However, the criteria developed for human health and aquatic life are sufficiently stringent to protect these other uses. States may establish criteria specifically designed to protect these uses.

4. *Public Water Supply:* The drinking water exposure component of the human health effects criteria can apply directly to this use classification or may be appropriately modified depending upon whether the specific water supply system falls within the auspices of the Safe Drinking Water Act's (SDWA) regulatory control, and the type and level of treatment imposed upon the supply before delivery to the consumer. The SDWA controls the presence of toxic pollutants in finished ("end-of-tap") drinking water. A brief description of relevant sections of this Act is necessary to explain how the SDWA will work in conjunction with section 304(a)(1) criteria in protecting human health from the effects of toxics due to consumption of water.

Pursuant to section 1412 of the SDWA, EPA has promulgated "National Interim Primary Drinking Water Standards" for certain organic and inorganic substances. These standards establish "maximum contaminant levels" ("MCLs") which specify the maximum permissible level of a contaminant in water which may be delivered to a user of a public water system now defined as serving a minimum of 25 people. MCLs are established based on consideration of a range of factors including not only the health effects of the contaminants but also technological and economic feasibility of the contaminants' removal from the supply. EPA is required to establish revised primary drinking water regulations based on the effects of a contaminant on human health, and include treatment capability, monitoring availability, and costs. Under Section 1401(1)(D)(i) of the SDWA, EPA is also allowed to establish the minimum quality criteria for water which may be taken into a public water supply system.

Section 304(a)(1) criteria provide estimates of pollutant concentrations protective of human health, but do not consider treatment technology, costs and other feasibility factors. The section 304(a)(1) criteria also include fish bioaccumulation and consumption factors in addition to direct human drinking water intake. These numbers were not developed to serve as "end of tap" drinking water standards, and they have no regulatory significance under the SDWA. Drinking water standards are established based on considerations, including technological and economic feasibility, not relevant to section 304(a)(1) criteria. Section 304(a)(1) criteria may be analogous to the recommended maximum contaminant levels (RMCLs) under section 1412(b)(1)(B) of the SDWA in which, based upon a report from the National Academy of Sciences, the Administrator should set target levels for contaminants in drinking water at which "no known or anticipated adverse effects occur and which allows an adequate margin of safety". RMCLs do not take treatment, cost, and other feasibility factors into consideration. Section 304(a)(1) criteria are, in concept, related to the health-based goals specified in the RMCLs. Specific mandates of the SDWA such as the consideration of multi-media exposure, as well as different methods for setting maximum contaminant levels under the two Acts, may result in differences between the two numbers.

MCLs of the SDWA, where they exist, control toxic chemicals in finished drinking water. However, because of variations in treatment and the fact that only a relatively small number of MCLs have been developed, ambient water criteria may be used by the States as a supplement to SDWA regulations. States will have the option of applying MCLs, section 304(a)(1) human health effects criteria, modified section 304(a)(1) criteria or controls more stringent than these three to protect against the effects of toxic pollutants by ingestion from drinking water.

For untreated drinking water supplies, States may control toxics in the ambient water through either use of MCLs (if they exist for the pollutants of concern), section 304(a)(1) human health effects criteria, or a more strigent contaminant level than the former two options.

For treated drinking water supplies serving less than 25 people, States may choose toxics control through application of MCLs (if they exist for the pollutants of concern and are attainable by the type of treatment) in the finished drinking water. States also have the options to control toxics in the ambient water by choosing section 304(a)(1.) criteria, adjusted section 304(a)(1) criteria resulting from the reduction of the direct drinking water exposure component in the criteria calculation to the extent that the treatment procedure reduces the level of pollutants, or a more stringent contaminant level than the former three options.

For treated drinking water supplies serving 25 people or greater, States *must* control toxics down to levels at least as stringent as MCLs (where they exist for the pollutants of concern) in the finished drinking water. However, States also have the options to control toxics in the ambient water by choosing section 304(a)(1) criteria, adjusted section 304(a)(1) criteria resulting from the reduction of the direct drinking water exposure component in the criteria calculation to the extent that the treatment process reduces the level of pollutants, or a more stringent contaminant level than the former three options.

Inclusion of Specific Pollutants in State Standards:

To date, EPA has not required that a State address any specific pollutant in its standards. Although all States have established standards for most conventional pollutants, the treatment of toxic pollutants has been much less extensive. In the ANPRM, EPA suggested a policy under which States would be required to address a set of pollutants and incorporate specific toxic pollutant criteria into water quality standards. If the State failed to incorporate these criteria, EPA would promulgate the standards based upon these criteria pursuant to section 303(c)(4)(B).

In the forthcoming proposed revision to the water quality standard regulations, a significant change in policy will be proposed relating to the incorporation of certain pollutants in State water quality standards. This proposal will differ from the proposal made in the ANPRM. The ANPRM proposed an EPA-published list of pollutants for which States would have had to develop water quality standards. This list might have contained some (or all) of the 65 toxic pollutants. However, the revised water quality standards regulation will propose a process by which EPA will assist States in identifying specific toxic pollutants required for assessment for possible inclusion in State water quality standards. For these pollutants, States will have the option of adopting the published criteria or of adjusting those criteria based on site-specific analysis.

These pollutants would generally represent the greatest threat to sustaining a healthy, balanced ecosystem in water bodies or to human health due to exposure directly or indirectly from water. EPA is currently developing a process to determine which pollutants a State must assess for possible inclusion in its water quality standards. Relevant factors might include the toxicity of the pollutant, the frequency and concentration of its discharge, its geographical distribution, the breadth of data underlying the scientific assessment of its aquatic life and human health effects, and the technological and economic capacity to control the discharge of the pollutant. For some of the pollutants, all States may be required to assess them for possible inclusion in their standards. For others, assessment would be restricted to States or limited to specific water bodies where the pollutants pose a particular site-specific problem.

Criteria Modification Process

Flexibility is available in the application of these and any other valid water quality criteria to regulatory programs. Although in some cases they may be used by the States as developed, the criteria may be modified to refect local environmental conditions and human exposure patterns before incorporation into programs such as water quality standards. If significant impacts of site-specific water quality conditions in the toxicities of pollutants can be demonstrated or significantly different exposure patterns of these pollutants to humans can be shown, section 304(a)(1) criteria may be modified to reflect these local conditions. The term "local" may refer to any appropriate geographic area where common aquatic environmental conditions or exposure patterns exist. Thus, "local" may signify a Statewide, regional, river reach, or entire river basin area. On the other hand, the criteria of some pollutants might be applicable nationwide without the need for adaptation to reflect local conditions. The degree of toxicity toward aquatic organisms and humans characteristic of these pollutants would not change significantly due to local water quality conditions.

EPA is examining a series of environmental factors or water quality parameters which might realistically be expected to affect the laboratory-derived water quality criterion recommendation for a specific pollutant. Factors such as hardness, pH, suspended solids, types of aquatic organisms present, etc. could impact on the chemical's effect in the aquatic environment. Therefore, local information can be assembled and analyzed to adjust the criterion recommendation if necessary.

The Guidelines for deriving criteria for the protection of aquatic life suggest several approaches for modifying the criteria. First, toxicity data, both acute and chronic, for local species could be substituted for some or all of the species used in deriving criteria for the water quality standard. The minimum data requirements should still be fulfilled in calculating a revised criterion. Second, criteria may be specifically tailored to a local water body by use of data from toxicity tests performed with that ambient water. A procedure such as this would account for local environmental conditions in formulating a criterion relevant to the local water body. Third, site-specific water quality characteristics resulting in either enhancement or mitigation of aquatic life toxicity for the pollutant could be factored into final formulation of the criterion. Finally, the criteria may be made more stringent to ensure protection of an individual species not otherwise adequately protected by any of the three modification procedures previously mentioned.

EPA does not intend to have States assess every local stream segment and lake in the country on an individual basis before determining if an adjustment is necessary. Rather, it is envisioned that water bodies having similar hydrological, chemical, physical, and biological properties will be grouped for the purpose of criteria adjustment. The purpose of this effort is to assist States in adapting the section 304(a) criteria to local conditions where needed, thereby precluding the setting of arbitrary and perhaps unnecessarily stringent or underprotective criteria in a water body. In all cases, EPA will still be required, pursuant to section 303(c), to determine whether the State water quality standards are consistent with the goals of the Act, including a determination of whether State-established criteria are adequate to support a designated use.

Criteria for the Protection of Aquatic Life

Interpretation of the Criteria

The aquatic life criteria issued today are summarized in Appendix A of this **Federal Register** notice. Criteria have been formulated by applying a set of Guidelines to a data base for each pollutant. The criteria for the protection of aquatic life specify pollutant concentrations which, if not exceeded, should protect most, but not necessarily all, aquatic life and its uses. The Guidelines specify that criteria should be based on an array of data from organisms, both plant and animal, occupying various trophic levels. Based on these data, criteria can be derived which should be adequate to protect the types of organisms necessary to support an aquatic community.

The Guidelines are not designed to derive criteria which will protect all life stages of all species under all conditions. Generally some life stage of one or more tested species, and probably some untested species, will have sensitivities below the maximum value or the 24-hour average under some conditions and would be adversely affected if the highest allowable pollutant concentrations and the worst conditions existed for a long time. In actual practice, such a situation is not likely to occur and thus the aquatic community as a whole will normally be protected if the criteria are not exceeded. In any aquatic community there is a wide range of individual species sensitivities to the effects of toxic pollutants. A criterion adequate to protect the most susceptible life stage of the most sensitive species would in many cases be more stringent than necessary to protect the overall aquatic community.

The aquatic life criteria specify both maximum and 24-hour average values. The combination of the two values is designed to provide adequate protection of aquatic life and its uses from acute and chronic toxicity and bioconcentration without being as restrictive as a one-number criterion would have to be to provide the same amount of protection. A time period of 24 hours was chosen in order to ensure that concentrations not reach harmful levels for unacceptably long periods. Averaging for longer periods, such as a week or a month for example, could permit high concentrations to persist long enough to produce significant adverse effects. A 24-hour period was chosen instead of a slightly longer or shorter period in recognition of daily fluctuations in waste discharges and of the influence of daily cycles of sunlight and darkness and temperature on both pollutants and aquatic organisms.

The maximum value, which is derived from acute toxicity data, prevents significant risk of adverse impact to organisms exposed to concentrations above the 24-hour average. Merely specifying the average value over a specified time period is insufficient because concentrations of chemicals higher than the average value can kill or cause irreparable damage in short periods. Furthermore, for some chemicals the effect of intermittent high exposures is cumulative. It is therefore necessary to place an upper limit on pollutant concentrations to which aquatic organisms might be exposed. The two-number criterion is intended to describe the highest average ambient water concentration which will produce a water quality generally suited to the maintenance of aquatic life while restricting the extent and duration of the excursions over that average to levels which will not cause harm. The only way to assure the same degree of protection with a one-number criterion would be to use the 24-hour average as a concentration that is not to be exceeded at any time in any place.

Since some substances may be more toxic in freshwater than in saltwater, or vice versa, provision is made for deriving separate water quality criteria for freshwater and for saltwater for each substance. However, for some substances sufficient data may not be available to derive one or both of these criteria using the Guidelines.

Specific aquatic life criteria have not been developed for all of the 65 toxic pollutants. In those cases where there were insufficient data to allow the derivation of a criterion, narrative descriptions of apparent threshold levels for acute and/or chronic effects based on the available data are presented. These descriptions are intended to convey a sense of the degree of toxicity of the pollutant in the absence of a criterion recommendation.

Summary of the Aquatic Life Guidelines

The Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and its Uses were developed to describe an objective, internally consistent, and appropriate way of ensuring that water quality criteria for aquatic life would provide, on the average, a reasonable amount of protection without an unreasonable amount of overprotection or underprotection. The resulting criteria are not intended to provide 100 percent protection of all species and all uses of aquatic life all of the time, but they are intended to protect most species in a balanced, healthy aquatic community. The Guidelines are published as Appendix B of this Notice. Responses to public comments on these Guidelines are attached as Appendix D.

Minimum data requirements are identified in four areas: acute toxicity to animals (eight data points), chronic toxicity to animals (three data points), toxicity to plants, and residues. Guidance is also given for discarding poor quality data.

Data on acute toxicity are needed for a variety of fish and invertebrate species and are used to derive a Final Acute Value. By taking into account the number and relative sensitivities of the tested species, the Final Acute Value is designed to protect most, but not necessarily all, of the tested and untested species.

Data on chronic toxicity to animals can be used to derive a Final Chronic Value by two different means. If chronic values are available for a specified number and array of species, a final chronic value can be calculated directly. If not, an acute-chronic ratio is derived and then used with the Final Acute Value to obtain the Final Chronic Value.

The Final Plant Value is obtained by selecting the lowest plant toxicity value based on measured concentrations.

The Final Residue Value is intended to protect wildlife which consume aquatic organisms and the marketability of aquatic organisms. Protection of the marketability of aquatic organisms is, in actuality, protection of a use of that water body ("commercial fishery"). Two kinds of data are necessary to calculate the Final Residue Value: a bioconcentration factor (BCF) and a maximum permissible tissue concentration, which can be an FDA action level or can be the result of a chronic wildlife feeding study. For lipid soluble pollutants, the BCF is normalized for percent lipids and then the Final Residue Value is calculated by dividing the maximum permissible tissue concentration by the normalized BCF and by an appropriate percent lipid value. BCFs are normalized for percent lipids since the BCF measured for any individual aquatic species is generally proportional to the percent lipids in that species.

If sufficient data are available to demonstrate that one or more of the final values should be related to a water quality characteristic, such as salinity, hardness, or suspended solids, the final value(s) are expressed as a function of that characteristic.

After the four final values (Final Acute Value, Final Chronic Value, Final Plant Value, and Final Residue Value) have been obtained, the criterion is established with the Final Acute Value becoming the maximum value and the lowest of the other three values becoming the 24-hour average value. All of the data used to calculate the four final values and any additional pertinent information are then reviewed to determine if the criterion is reasonable. If sound scientific evidence indicates that the criterion should be raised or lowered, appropriate changes are made as necessary.

The present Guidelines have been revised from the earlier published versions (43 FR 21506, May 18, 1978; 43 FR 29028, July 5, 1978; 44 FR 15926, March 15, 1979). Details have been added in many places and the concept of a minimum data base has been incorporated. In addition, three adjustment factors and the species sensitivity factor have been deleted. These modifications were the result of the Agency's analysis of public comments and comments received from the Science Advisory Board on earlier versions of the Guidelines. These comments and the Resultant modifications are addressed fully in Appendix D to this notice.

Criteria for the Protection of Human Health

Interpretation of the Human Health Criteria

The human health criteria issued today are summarized in Appendix A of this **Federal Register** notice. Criteria for the protection of human health are presented for 62 of the 65 pollutants based on their carcinogenic, toxic, or organoleptic (taste and odor) properties. The meanings and practical uses of the criteria values are distinctly different depending on the properties on which they are based.

The objective of the health assessment portions of the criteria documents is to estimate ambient water concentrations which, in the case of non-carcinogens, prevent adverse health effects in humans, and in the case of suspect or proven carcinogens, represent various levels of incremental cancer risk.

Health assessments typically contain discussions of four elements: Exposure, pharmacokinetics, toxic effects, and criterion formulation.

The exposure section summarizes information on exposure routes: ingestion directly from water, indirectly from consumption of aquatic organisms found in ambient water, other dietary sources, inhalation, and dermal contact. Exposure assumptions are used to derive human health criteria. Most criteria are based solely on exposure from consumption of water containing a specified concentration of a toxic pollutant and through consumption of aquatic organisms which are assumed to have bioconcentrated pollutants from the water in which they live. Other multimedia routes of exposure such as air, non-aquatic diet, or dermal are not factored into the criterion formulation for the vast majority of pollutants due to lack of data. The criteria are calculated using the combined aquatic exposure pathway and also using the aquatic organism ingestion exposure route alone. In criteria reflecting both the water consumption and aquatic organism ingestion routes of exposure, the relative exposure contribution varies with the propensity of a pollutant to bioconcentrate, with the consumption of aquatic organisms becoming more important as the bioconcentration factor (BCF) increases. As additional information on total exposure is assembled for pollutants for which criteria reflect only the two specified aquatic exposure routes, adjustments in water concentration values may be made. The Agency intends to publish guidance which will permit the States to identify significantly different exposure patterns for their populations. If warranted by the demonstration of significantly different exposure patterns, this will become an element of a process to adapt/modify human health-based criteria to local conditions, somewhat analogous to the aquatic life criteria modification process discussed previously. It is anticipated that States at their discretion will be able to set appropriate human health criteria based on this process.

The pharmacokinetics section reviews data on absorption, distribution, metabolism, and excretion to assess the biochemical fate of the compounds in the human and animal system. The toxic effects section reviews data on acute, subacute, and chronic toxicity, synergistic and antagonistic effects, and specific information on mutagenicity, teratogenicity, and carcinogenicity. From this review, the toxic effect to be protected against is identified taking into account the quality, quantity, and weight of evidence characteristic of the data. The criterion formulation section reviews the highlights of the text and specifies a rationale for criterion development and the mathematical derivation of the criterion number.

Within the limitations of time and resources, current published information of significance was incorporated into the human health assessments. Review articles and reports were used for data evaluation and synthesis. Scientific judgment was exercised in reviewing and evaluating the data in each criteria document and in identifying the adverse effects for which protective criteria were published.

Specific health-based criteria are developed only if a weight of evidence supports the occurrence of the toxic effect and if dose/response data exist from which criteria can be estimated.

Criteria for suspect or proven carcinogens are presented as concentrations in water associated with a range of incremental cancer risks to man. Criteria for non-carcinogens represent levels at which exposure to a single chemical is not anticipated to produce adverse effects in man. In a few cases, organoleptic (taste and odor) data form the basis for the criterion. While this type of criterion does not represent a value which directly affects human health, it is presented as an estimate of the level of a pollutant that will not produce unpleasant taste or odor either directly from water consumption or indirectly by consumption of aquatic organisms found in ambient waters. A criterion developed in this manner is judged to be as useful as other types of criteria in protecting designated water uses. In addition, where data are available, toxicity-based criteria are also presented for pollutants with derived organoleptic criteria. The choice of criteria used in water quality standards for these pollutants will depend upon the designated use to be protected. In the case of a multiple use water body, the criterion protecting the most sensitive use will be applied. Finally, for several pollutants no criteria are recommended due to a lack of information sufficient for quantitative criterion formulation.

Risk Extrapolation

Because methods do not now exist to establish the presence of a threshold for carcinogenic effects, EPA's policy is that there is no scientific basis for estimating "safe" levels for carcinogens. The criteria for carcinogens, therefore, state that the recommended concentration for maximum protection of human health is zero. In addition, the Agency has presented a range of concentrations corresponding to incremental cancer risks of 10^{-7} to 10^{-5} (one additional case of cancer in populations ranging from ten million to 100,000, respectively). Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Summary of the Human Health Guidelines

The health assessments and corresponding criteria published today were derived based on *Guidelines and Methodology Used in the Preparation of Health Effect Assessment Chapters of the Consent Decree Water Criteria Documents* (the Guidelines) developed by EPA's Office of Reserch and Development. The estimation of health risks associated with human exposure to environmental pollutants requires predicting the effect of low doses for up to a lifetime in duration. A combination of epidemiological and animal dose/response data is considered the preferred basis for quantitative criterion derivation. The complete Guidelines are presented as Appendix C. Major issues associated with these Guidelines and responses to public comments are presented as Appendix E.

No-effect (non-carcinogen) or specified risk (carcinogen) concentrations were estimated by extrapolation from animal toxicity or human epidemiology studies using the following basic exposure assumptions: a 70-kilogram male person (*Report of the Task Group on Reference Man*, International Commission for Radiation Protection, November 23, 1957) as the exposed individual; the average daily consumption of freshwater and estuarine fish and shellfish products equal to 6.5 grams/day; and the average ingestion of two liters/day of water (*Drinking Water and Health*, National Academy of Sciences, National Research Council, 1977). Criteria based on these assumptions are estimated to be protective of an adult male who experiences average exposure conditions.

Two basic methods were used to formulate health criteria, depending on whether the prominent adverse effect was cancer or other toxic manifestations. The following sections detail these methods.

Carcinogens

Extrapolation of cancer responses from high to low doses and subsequent risk estimation from animal data is performed using a linearized multi-stage model. This procedure is flexible enough to fit all monotonically-increasing dose response data, since it incorporates several adjustable parameters. The multi-stage model is a linear non-threshold model as was the "one-hit" model originally used in the proposed criteria documents. The linearized multi-stage model and its characteristics are described fully in Appendix C. The linear non-threshold concept has been endorsed by the four agencies in the Interagency Regulatory Liaison Group and is less likely to underestimate risk at the low doses typical of environmental exposure than other models that could be used. Because of the uncertainties associated with dose response, animal-to-human extrapolation and other unknown factors, because of the use of average exposure assumptions, and because of the serious public health consequences that could result if risk were underestimated, EPA believes that it is prudent to use conservative methods to estimate risk in the water quality criteria program. The linearized multistage model is more systematic and invokes fewer arbitrary assumptions than the "one-hit" procedure previously used.

It should be noted that extrapolation models provide estimates of risk since a varitey of assumptions are built into any model. Models using widely different assumptions may produce estimates ranging over several orders of magnitude. Since there is at present no way to demonstrate the scientific validity of any model, the use of risk extrapolation models is a subject of debate in the scientific community. However, risk extrapolation is generally recognized as the only tool available at this time for estimating the magnitude of health hazards associated with non-threshold toxicants and has been endorsed by numerous Federal agencies and scientific organizations, including EPA's Carcinogen Assessment Group, the National Academy of Sciences, and the Interagency Regulatory Liaison Group as a useful means of assessing the risks of exposure to various carcinogenic pollutants.

Non-Carcinogens

Health criteria based on toxic effects of pollutants other than carcinogenicity are estimates of concentrations which are not expected to produce adverse effects in humans. They are based upon Acceptable Daily Intake (ADI) levels and are generally derived using no-observed-adverse-effect-level (NOAEL) data from animal studies although human data are used wherever available. The ADI is calculated using safety factors to account for uncertainties inherent in extrapolation from animal to man. In accordance with the National Research Council recommendations *(Drinking Water and Health*, National Academy of Sciences, National Research Council, 1977), safety factors of 10, 100, or 1,000 are used depending on the quality and quantity of data. In some instances extrapolations are made from inhalation studies or limits to approximate a human response from ingestion using the Stokinger-Woodward model (Journal of American Water Works Association, 1958). Calculations of criteria from ADIs are made using the standard exposure assumptions (2 liters of water, 6.5 grams of edible aquatic products, and an average body weight of 70 kg).

Dated: October 24, 1980.

Douglas M. Costle,
Administrator.

Appendix A—Summary of Water Quality Criteria

Acenaphthene

Freshwater Aquatic Life

The available data for acenaphthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,700 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of acenaphthene to sensitive freshwater aquatic animals but toxicity to freshwater algae occur at concentrations as low as 520 µg/l.

Saltwater Aquatic Life

The available data for acenaphthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 970 and 710 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae occurs at concentrations as low as 500 µg/l.

Human Health

Sufficient data is not available for acenaphthene to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 20 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Acrolein

Freshwater Aquatic Life

The available data for acrolein indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 68 and 21 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for acrolein indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 55 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of acrolein to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of acrolein ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 320 µg/l.

For the protection of human health from the toxic properties of acrolein ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 780 µg/l.

Acrylonitrile

Freshwater Aquatic Life

The available data for acrylonitrile indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 7,550 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of acrylonitrile to sensitive freshwater aquatic life but mortality occurs at concentrations as low as 2,600 µg/l with a fish species exposed for 30 days.

Saltwater Aquatic Life

Only one saltwater species has been tested with acrylonitrile and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of acrylonitrile through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .58 µg/l, .058 µg/l and .006 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 6.5 µg/l, .65 µg/l, and .065 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Aldrin-Dieldrin

Dieldrin

Freshwater Aquatic Life

For dieldrin the criterion to protect fresh water aquatic life as derived using the Guidelines is 0.0019 µg/l as a 24-hour average and the concentration should not exceed 2.5 µg/l at any time.

Saltwater Aquatic Life

For dieldrin the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0019 µg/l as a 24-hour average and the concentration should not exceed 0.71 µg/l at any time.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of dieldrin through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .71 ng/l, .071 ng/l, and .0071 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are .76 ng/l, .076 ng/l, and .0076 ng/l respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Aldrin

Freshwater Aquatic Life

For freshwater aquatic life the concentration of aldrin should not exceed 3.0 µg/l at any time. No data are available concerning the chronic toxicity of aldrin to sensitive freshwater aquatic life.

Saltwater Aquatic Life

For saltwater aquatic life the concentration of aldrin should not exceed 1.3 µg/l at any time. No data are available concerning the chronic toxicity of aldrin to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of aldrin through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .74 ng/1, .074 ng/1, and .0074 ng/1, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are .79 ng/1, .079 ng/1, and .0079 ng/1, respectively. Other concentrations respresenting different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Antimony

Freshwater Aquatic Life

The available data for antimony indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 9,000 and 1,600 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae occurs at concentrations as low as 610 µg/l.

Saltwater Aquatic Life

No saltwater organisms have been adequately tested with antimony, and no statement can be made concerning acute or chronic toxicity.

Human Health

For the protection of human health from the toxic properties of antimony ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 146 µg/l.

For the protection of human health from the toxic properties of antimony ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 45,000 µg/l.

Arsenic

Freshwater Aquatic Life

For freshwater aquatic life the concentration of total recoverable trivalent inorganic arsenic should not exceed 440 µg/l at any time. Short-term effects on embryos and larvae of aquatic vertebrate species have been shown to occur at concentrations as low as 40 µg/l.

Saltwater Aquatic Life

The available data for total recoverable trivalent inorganic arsenic indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 508 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trivalent inorganic arsenic to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of arsenic through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 22 ng/l, 2.2 ng/l, and .22 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 175 ng/l, 17.5 ng/l, and 1.75 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Asbestos

Freshwater Aquatic Life

No freshwater organisms have been tested with any asbestiform mineral and no statement can be made concerning acute or chronic toxicity.

Saltwater Aquatic Life

No saltwater organisms have been tested with any asbestiform mineral and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of asbestos through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 300,000 fibers/1,30,000 fibers/1, and 3,000 fibers/1, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Benzene

Freshwater Aquatic Life

The available data for benzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 5,300 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of benzene to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for benzene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 5,100 µg/l and would occur at lower concentrations among species that are more sensitive then those tested. No definitive data are available concerning the chronic toxicity of benzene to sensitive saltwater aquatic life, but adverse effects occur at concentrations as low as 700 µg/l with a fish species exposed for 168 days.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of benzene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 6.6 µg/l, .66 µg/l, and .066 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 400 µg/l, 40.0 µg/l, and 4.0 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Benzidine

Freshwater Aquatic Life

The available data for benzidine indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 2,500 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of benzidine to sensitive freshwater aquatic life.

Saltwater Aquatic Life

No saltwater organisms have been tested with benzidine and no statement can be made concerning acute and chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of benzidine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 1.2 ng/1, .12 ng/1, and .01 ng/1, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5.3 ng/1, .53 ng/1, and .05 ng/1, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Beryllium

Freshwater Aquatic Life

The available data for beryllium indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 130 and 5.3 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Hardness has a substantial effect on acute toxicity.

Salt water Aquatic Life

The limited saltwater data base available for beryllium does not permit any statement concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of beryllium through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 37 ng/l, 3.7 ng/l, and .37 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 641 ng/l, 64.1 ng/l, and 6.41 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Cadmium

Freshwater Aquatic Life

For total recoverable cadmium the criterion (in µg/l) to protect freshwater aquatic life as derived using the Guidelines is the numerical value given by $e^{(1.05[\ln(\text{hardness})]-8.53)}$ as a 24-hour average and the concentration (in μg/l) should not exceed the numerical value given by $e^{(1.05[\ln(\text{hardness})]-3.73)}$ at any time. For example, a hardnesses of 50, 100, and 200 mg/l as $CaCO_3$ the criteria are 0.012, 0.025, and 0.051 μg/l, respectively, and the concentration of total recoverable cadmium should not exceed 1.5, 3.0 and 6.3 μg/l, respectively, at any time.

Saltwater Aquatic Life

For total recoverable cadmium the criterion to protect saltwater aquatic life as derived using the Guidelines is 4.5 μg/l as a 24-hour average and the concentration should not exceed 59 μg/l at any time.

Human Health

The ambient water quality criterion for cadmium is recommended to be identical to the existing drinking water standard which is 10 μg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

Carbon Tetrachloride

Freshwater Aquatic Life

The available data for carbon tetrachloride indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 35,200 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of carbon tetrachloride to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for carbon tetrachloride indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 50,000 μg/l and would occur at lower concentrations among species that are more sensitive that those tested. No data are available concerning the chronic toxicity of carbon tetrachloride to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of carbon tetrachloride through ingestion of contaminated water and contaminated aquatic organisms the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 4.0μg/l, .40 μg/l, and .04 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 69.4 μg/l, 6.94 μg/l, and .69 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Chlordane

Freshwater Aquatic Life

For chlordane the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0043 μg/l as a 24-hour average and the concentration should not exceed 2.4 μg/l at any time.

Saltwater Aquatic Life

For chlordane the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0040 μg/l as a 24-hour average and the concentration should not exceed 0.09 μg/l at any time.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of chlordane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 4.6 ng/l, .46 ng/l, and .046 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 4.8 ng/l, .48 ng/l, and .048 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Chlorinated Benzenes

Freshwater Aquatic Life

The available data for chlorinated benzenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 250 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of the more toxic of the chlorinated benzenes to sensitive freshwater aquatic life but toxicity occurs at concentrations as low as 50 μg/l for a fish species exposed for 7.5 days.

Saltwater Aquatic Life

The available data for chlorinated benzenes indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 160 and 129 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to expcsure of hexachlorobenzene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding recommended criteria are 7.2 ng/l, .72 ng/l, and .072 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 7.4 ng/l, .74 ng/l, and .074 ng/l, respectively.

For the protection of human health from the toxic properties of 1,2,4,5-tetrachlorobenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 38 μg/l.

For the protection of human health from the toxic properties of 1,2,4,5-tetrachlorobenzene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 48 μg/l.

For the protection of human health from the toxic properties of pentachlorobenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 74 μg/l.

For the protection of human health from the toxic properties of pentachlorobenzene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 85 μg/l.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for trichlorobenzene.

For comparison purposes, two approaches were used to derive criterion levels for monochlorobenzene. Based on available toxicity data, for the protection of public health, the derived level is 488 µg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 20 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Chlorinated Ethanes

Freshwater Aquatic Life

The available freshwater data for chlorinated ethanes indicate that toxicity increases greatly with increasing chlorination, and that acute toxicity occurs at concentrations as low as 118,000 µg/l for 1,2-dichloroethane, 18,000 µg/l for two trichloroethanes, 9,320 µg/l for two tetrachloroethanes, 7,240 µg/l for pentachloroethane, and 980 µg/l for hexachloroethane. Chronic toxicity occurs at concentrations as low as 20,000 µg/l for 1,2-dichloroethane, 9,400 µg/l for 1,1,2-trichloroethane, 2,400 µg/l for 1,1,2,2,-tetrachloroethane, 1,100 µg/l for pentachloroethane, and 540 µg/l for hexachloroethane. Acute and chronic toxicity would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available saltwater data for chlorinated ethanes indicate that toxicity increases greatly with increasing chlorination and that acute toxicity to fish and invertebrate species occurs at concentrations as low as 113,000 µg/l for 1,2-dichloroethane, 31,200 µg/l for 1,1,1-trichloroethane, 9,020 µg/l for 1,1,2,2-tetrachloroethane, 390 µg/l for pentachloroethane, and 940 µg/l for hexachloroethane. Chronic toxicity occurs at concentrations as low as 281 µg/l for pentachloroethane. Acute and chronic toxicity would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 1,2-dichloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 9.4 µg/l, .94 µg/l, and .094 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 2,430 µg/l, 243 µg/l, and 24.3 µg/l respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the protection of human health from the toxic properties of 1,1,1-trichloroethane ingested through water and contaminated aquatic organism, the ambient water criterion is determined to be 18.4 mg/l.

For the protection of human health from the toxic properties of 1,1,1,-trichloroethane ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 1.03 g/l.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 1,1,2-trichloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 6.0 µg/l, .6 µg/l, and .06 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 418 µg/l, 41.8 µg/l, and 4.18 µg/l respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 1,1,2,2-tetrachloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 1.7 µg/l, .17 µg/l, and .017 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 107 µg/l, 10.7 µg/l, and 1.07 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of hexachloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 19 µg/l, 1.9 µg/l, and .19 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 87.4 µg/l, 8.74 µg/l, and .87 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for monochloroethane.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for 1,1,-dichloroethane.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for 1,1,1,2,-tetrachloroethane.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for pentachloroethane.

Chlorinated Naphthalenes

Freshwater Aquatic Life

The available data for chlorinated naphthalenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,600 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of chlorinated naphthalenes to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for chlorinated napthalenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 7.5 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of chlorinated naphthalenes to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for chlorinated napthalenes.

Chlorinated Phenols

Freshwater Aquatic Life

The available freshwater data for chlorinated phenols indicate that toxicity generally increases with increasing chlorination, and that acute toxicity occurs at concentrations as low as 30 µg/l for 4-chloro-3-methylphenol to greater than 500,000 µg/l for other compounds. Chronic toxicity occurs at concentrations as low as 970 µg/l for 2,4,6-trichlorophenol. Acute and chronic toxicity would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available saltwater data for chlorinated phenols indicate that toxicity generally increases with increasing chlorination and that acute toxicity occurs at concentrations as low as 440 µg/l for 2,3,5,6-tetrachlorophenol and 29,700 µg/l for 4-chlorophenol. Acute toxicity would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of chlorinated phenols to sensitive saltwater aquatic life.

Human Health

Sufficient data is not available for 3-monochlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 0.1 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 4-monochlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 0.1 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 2,3-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is .04 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 2,5-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is .5 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 2,6-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is .2 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 3,4-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is .3 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 2,3,4,6-tetrachlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 1 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

For comparison purposes, two approaches were used to derive criterion levels for 2,4,5-trichlorophenol. Based on available toxicity data, for the protection of public health, the derived level is 2.6 mg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 1.0 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 2,4,6-trichlorophenol through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 12 µg/l, 1.2 µg/l, and .12 µg/l respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 36 µg/l, 3.6 µg/l, and .36 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 2 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 2-methyl-4-chlorophenol to derive a level which would protect against any potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 1800 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 3-methyl-4-chlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 3000 μg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Sufficient data is not available for 3-methyl-6-chlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 20 μg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Chloroalkyl Ethers

Freshwater Aquatic Life

The available data for chloroalkyl ethers indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 238,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of chloroalkyl ethers to sensitive freshwater aquatic life.

Saltwater Aquatic Life

No saltwater organisms have been tested with any chloroalkyl ether and no statement can be made concerning acute and chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of bis-(chloromethyl)-ether through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .038 ng/l, .0038 ng/l, and .00038 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 18.4 ng/l, 1.84 ng/l, and .184 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of bis (2-chloroethyl) ether through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .3 μg/l, .03 μg/l, and .003 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 13.6 μg/l, 1.36 μg/l, and .136 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the protection of human health from the toxic properties of bis (2-chloroisopropyl) ether ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 34.7 μg/l.

For the protection of human health from the toxic properties of bis (2-chloroisopropyl) ether ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 4.36 mg/l.

Chloroform

Freshwater Aquatic Life

The available data for choloroform indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 28,900 μg/l, and would occur at lower concentrations among species that are more sensitive than the three tested species. Twenty-seven-day LC50 values indicate that chronic toxicity occurs at concentrations as low as 1,240 μg/l, and could occur at lower concentrations among species or other life stages that are more sensitive than the earliest life cycle stage of the rainbow trout.

Saltwater Aquatic Life

The data base for saltwater species is limited to one test and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of chloroform through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 1.90 μg/l, .19 μg/l, and .019 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 157 μg/l, 15.7 μg/l, and 1.57 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

2-Chlorophenol

Freshwater Aquatic Life

The availabe data for 2-chlorophenol indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 4,380 μg/l and would occur at lower concentrations among species that are more sensitive that those tested. No definitive data are available concerning the chronic toxicity of 2-chlorophenol to sensitive freshwater aquatic life but flavor impairment occurs in one species of fish at concentrations as low as 2,000 μg/l.

Saltwater Aquatic Life

No saltwater organisms have been tested with 2-chlorophenol and no statement can be made concerning acute and chronic toxicity.

Human Health

Sufficient data is not available for 2-chlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 0.1 μg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Chromium

Freshwater Aquatic Life

For total recoverable hexavalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.29 μg/l as a 24-hour average and the concentration should not exceed 21 μg/l at any time.

For freshwater aquatic life the concentration (in μg/l) of total recoverable trivalent chromium should not exceed the numerical value given by "$e^{(1.08[\ln(\text{hardness})]+3.48)}$" at any time. For example, at hardnesses of 50, 100 and 200 mg/l as $CaCO_3$ the concentration of total recoverable trivalent chromium should not exceed 2,200, 4,700, and 9,900 μg/l, respectively, at any time. The available data indicate that chronic toxicity to freshwater aquatic life occurs at concentrations as low a 44 μg/l and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

For total recoverable hexavalent chromium the criterion to protect saltwater aquatic life as derived using the Guidelines is 18 μg/l as a 24-hour average and the concentration should not exceed 1,260 μg/l at any time.

For total recoverable trivalent chromium, the availabe data indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 10,300 μg/l, and would occur at lower concentrations amoung species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trivalent chromium to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of Chromium III ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 170 mg/l.

For the protection of human health from the toxic properties of Chromium III ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 3433 mg/l.

The ambient water quality criterion for total Chromium VI is recommended to be identical to the existing drinking water standard which is 50 μg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

Copper

Freshwater Aquatic Life

For total recoverable copper the criterion to protect freshwater aquatic life as derived using the Guidelines is 5.6 μg/l as a 24-hour average and the concentration (in μg/l) should not exceed the numerical value given by $e^{(0.94[\ln(\text{hardness})]-1.23)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l $CaCO_3$ the concentration of total recoverable copper should not exceed 12, 22, and 43 μg/l at any time.

Saltwater Aquatic Life

For total recoverable copper the criterion to protect saltwater aquatic life as derived using the Guidelines is 4.0 μg/l as a 24-hour average and the concentration should not exceed 23 μg/l at any time.

Human Health

Sufficient data is not available for copper to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 1 mg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Cyanide

Freshwater Aquatic Life

For free cyanide (sum of cyanide present as HCN and CN^-, expressed as CN) the criterion to protect freshwater aquatic life as derived using the Guidelines is 3.5 μg/l as a 24-hour average and the concentration should not exceed 52 μg/l at any time.

Saltwater Aquatic Life

The available data for free cyanide (sum of cyanide present as HCN and CN^-, expressed as CN) indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 30 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. If the acute-chronic ratio for saltwater organisms is similar to that for freshwater organisms, chronic toxicity would occur at concentrations as low as 2.0 μg/l for the tested species and at lower concentrations among species that are more sensitive than those tested.

Human Health

The ambient water quality criterion for cyanide is recommended to be identical to the existing drinking water standard which is 200 μg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

DDT and Metabolites

Freshwater Aquatic Life

DDT

For DDT and its metabolites the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0010 μg/l as a 24-hour average and the concentration should not exceed 1.1 μg/l at any time.

TDE

The available data for TDE indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 0.6 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of TDE to sensitive freshwater aquatic life.

DDE

The available data for DDE indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,050 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of DDE to sensitive freshwater aquatic life.

Saltwater Aquatic Life

DDT

For DDT and its metabolites the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0010 μg/l as a 24-hour average and the concentration should not exceed 0.13 μg/l at any time.

TDE

The available data for TDE indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 3.6 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of TDE to sensitive saltwater aquatic life.

DDE

The available data for DDE indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 14 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of DDE to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of DDT through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .24 ng/l, .024 ng/l, and .0024 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are .24 ng/l, .024 ng/l, and .0024 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment of an "acceptable" risk level.

Dichlorobenzenes

Freshwater Aquatic Life

The available data for dichlorobenzenes indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 1,120 and 763 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for dichlorobenzenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 1,970 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dichlorobenzenes to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of dichlorobenzenes (all isomers) ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 400 µg/l.

For the protection of human health from the toxic properties of dichlorobenzenes (all isomers) ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 2.6 mg/l.

Dichlorobenzidines

Freshwater Aquatic Life

The data base available for dichlorobenzidines and freshwater organisms is limited to one test on bioconcentration of 3,3'-dichlorobenzidine and no statement can be made concerning acute or chronic toxicity.

Saltwater Aquatic Life

No saltwater organisms have been tested with any dichlorobenzidine and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of dichlorobenzidine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero base on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .103 µg/l, .0103 µg/l, and .00103 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are .204 µg/l, .0204 µg/l, and .00204 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Dichloroethylenes

Freshwater Aquatic Life

The available data for dichloroethylenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,600 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of dichlorethylenes to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for dichlorethylenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 224,000 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity dichloroethylenes to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 1,1-dichloroethylene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .33 µg/l, .033 µg/l, and .0033 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 18.5 µg/l, 1.85 µg/l, and .185 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficency in the available data for 1,2-dichloroethylene.

2,4-Dichlorophenol

Freshwater Aquatic Life

The available data for 2,4-dichlorophenol indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 2,020 and 365 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive that those tested. Mortality to early life stages of one species of fish occurs at concentrations as low as 70 µg/l.

Saltwater Aquatic Life

Only one test has been conducted with saltwater organisms on 2,4-dichlorophenol and no statement can be made concerning acute or chronic toxicity.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for 2,4-dichlorophenol.

Based on available toxicity data, for the protection of public health, the derived level is 3.09 mg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 0.3 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Dichloropropanes/Dichloropropenes

Freshwater Aquatic Life

The available data for dichloropropanes indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 23,000 and 5,700 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropenes indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 6,060 and 244 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for dichloropropanes indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as 10,300 and 3,040 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low a as 790 µg/l, and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dichloropropenes to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for dichloropropanes.

For the protection of human health from the toxic properties of dichloropropenes ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 87 µg/l.

For the protection of human health from the toxic properties of dichloropropenes ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 14.1 mg/l.

2,4-Dimethylphenol

Freshwater Aquatic Life

The available data for 2,4-dimethylphenol indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 2,120 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dimethylphenol to sensitive freshwater aquatic life.

Saltwater Aquatic Life

No saltwater organisms have been tested with 2,4-dimethylphenol and no statement can be made concerning acute and chronic toxicity.

Human Health

Sufficient data are not available for 2,4-dimethylphenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undersirable taste and odor quality of ambient water, the estimated level is 400 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

2,4-Dinitrotoluene

Freshwater Aquatic Life

The available data for 2,4-dinitrotoluene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 330 and 230 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for 2,4-dinitrotoluenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 590 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of 2,4-dinitrotoluenes to sensitive saltwater aquatic life but a decrease in algal cell numbers occurs at concentrations as low as 370 µg/l.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 2,4-dinitrotoluene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 1.1 µg/l, 0.11 µg/l, and 0.011 µg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 91 µg/l, 9.1 µg/l, and 0.91 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

1,2-Diphenylhydrazine

Freshwater Aquatic Life

The available data for 1,2-diphenylhydrazine indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 270 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of 1,2-diphenylhydrazine to sensitive freshwater aquatic life.

Saltwater Aquatic Life

No saltwater organisms have been tested with 1,2-diphenylhydrazine and no statement can be made concerning acute and chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of 1,2-diphenylhydrazine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 422 ng/l, 42 ng/l, and 4 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5.6 µg/l, 0.56 µg/l, and 0.056 µg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Endosulfan

Freshwater Aquatic Life

For endosulfan the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.056 μg/l as a 24-hour average and the concentration should not exceed 0.22 μg/l at any time.

Saltwater Aquatic Life

For endosulfan the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0087 μg/l as a 24-hour average and the concentration should not exceed 0.034 μg/l at any time.

Human Health

For the protection of human health from the toxic properties of endosulfan ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 74 μg/l.

For the protection of human health from the toxic properties of endosulfan ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 159 μg/l.

Endrin

Freshwater Aquatic Life

For endrin the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0023 μg/l as a 24-hour average and the concentration should not exceed 0.18 μg/l at any time.

Saltwater Aquatic Life

For endrin the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0023 μg/l as a 24-hour average and the concentration should not exceed 0.037 μg/l at any time.

Human Health

The ambient water quality criterion for endrin is recommended to be identical to the existing drinking water standard which is 1 μg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

Ethylbenzene

Freshwater Aquatic Life

The available data for ethylbenzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 32,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of ethylbenzene to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for ethylbenzene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 430 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of ethylbenzene to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of ethylbenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 1.4 mg/l.

For the protection of human health from the toxic properties of ethylbenzene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 3.28 mg/l.

Fluoranthene

Freshwater Aquatic Life

The available data for fluoranthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 3980 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of fluoranthene to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for fluoranthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 40 and 16 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the protection of human health from the toxic properties of fluoranthene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 42 μg/l.

For the protection of human health from the toxic properties of fluoranthene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 54 μg/l.

Haloethers

Freshwater Aquatic Life

The available data for haloethers indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 360 and 122 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

No saltwater organisms have been tested with any haloether and no statement can be made concerning acute or chronic toxicity.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for haloethers.

Halomethanes

Freshwater Aquatic Life

The available data for halomethanes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of halomethanes to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for halomethanes indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 12,000 and 6,400 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. A decrease in algal cell numbers occurs at concentrations as low as 11,500 μg/l.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of chloromethane, bromomethane, dichloromethane, bromodichloromethane, tribromomethane, dichlorodifluoromethane, trichlorofluoromethane, or combinations of these chemicals through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are

1.9 μg/l, 0.19 μg/l, and 0.019 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 157 μg/l, 15.7 μg/l, and 1.57 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Heptachlor

Freshwater Aquatic Life

For heptachlor the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0038 μg/l as a 24-hour average and the concentration should not exceed 0.52 μg/l at any time.

Saltwater Aquatic Life

For heptachlor the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0036 μg/l as a 24-hour average and the concentration should not exceed 0.053 μg/l at any time.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of heptachlor through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 2.78 ng/l, .28 ng/l, and .028 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 2.85 ng/l, .29 ng/l, and .029 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Hexachlorobutadiene

Freshwater Aquatic Life

The available data for hexachlorobutadiene indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 90 and 9.3 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for hexachlorobutadiene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 32 μg/l and would occur at lower concentrations among species that are more sensitive that those tested. No data are available concerning the chronic toxicity of hexachlorobutadiene to sensitive saltwater aquatic life

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of hexachlorobutadiene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 4.47 μg/l, 0.45 μg/l, and 0.045 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 500 μg/l, 50 μg/l, and 5 μg/l respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Hexachlorocyclohexane

Lindane

Freshwater Aquatic Life

For Lindane the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.080 μg/l as a 24-hour average and the concentration should not exceed 2.0 μg/l at any time.

Saltwater Aquatic Life

For saltwater aquatic life the concentration of lindane should not exceed 0.16 μg/l at any time. No data are available concerning the chronic toxicity of lindane to sensitive saltwater aquatic life.

BHC

Freshwater Aquatic Life

The available date for a mixture of isomers of BHC indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 100 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of a mixture of isomers of BHC to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available date for a mixture of isomers of BHC indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 0.34 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of a mixture of isomers of BHC to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of alpha-HCH through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 92 ng/l, 9.2 ng/l, and .92 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 310 ng/l, 31.0 ng/l, and 3.1 ng/l respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of beta-HCH through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 163 ng/l, 16.3 ng/l, and 1.63 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 547 ng/l, 54.7 ng/l, and 5.47 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of tech-HCH through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 123 ng/l, 12.3 ng/l, and 1.23 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 414 ng/l, 41.4 ng/l, and 4.14 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of gamma-HCH through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 186 ng/l, 18.6 ng/l, and 1.86 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 625 ng/l, 62.5 ng/l, 6.25 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for delta-HCH.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for epsilon-HCH.

Hexachlorocyclopentadiene

Freshwater Aquatic Life

The available data for hexachlorocyclopentadiene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 7.0 and 5.2 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data to hexachlorocyclopentadiene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 7.0 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of hexachlorocyclopentadiene to sensitive saltwater aquatic life.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for hexachlorocyclopentadiene. Based on available toxicity data, for the protection of public health, the derived level is 206 µg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 1.0 µg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Isophorone

Freshwater Aquatic Life

The available data for isophorone indicate that acute toxicity to freshwater aquatic life ocurs at concentrations as low as 117,000 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of isophorone to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for isophorone indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 12,900 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of isophorone to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of isophorone ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 5.2 mg/l.

For the protection of human health from the toxic properties of isophorone ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 520 mg/l.

Lead

Freshwater Aquatic Life

For total recoverable lead the criterion (in µg/l) to protect freshwater aquatic life as derived using the Guidelines is the numerical value given by $e^{(2.35[\ln(\text{hardness})]-9.48)}$ as a 24-hour average and the concentration (in µg/l) should not exceed the numerical value given by $e^{(1.22[\ln(\text{hardness})]-0.47)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as $CaCO_3$, the criteria are 0.75, 3.8, and 20 µg/l, respectively, as 24-hour averages, and the concentrations should not exceed 74, 170, and 400 µg/l, respectively, at any time.

Saltwater Aquatic Life

The available data for total recoverable lead indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 668 and 25 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

The ambient water quality criterion for lead is recommended to be identical to the existing drinking water standard which is 50 µg/l. Analysis of the toxic effects data resulted in a calculated level which is protective to human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

Mercury

Freshwater Aquatic Life

For total recoverable mercury the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.00057 µg/l as a 24-hour average and the concentration should not exceed 0.0017 µg/l at any time.

Saltwater Aquatic Life

For total recoverable mercury the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.025 µg/l as a 24-hour average and the concentration should not exceed 3.7 µg/l at any time.

Human Health

For the protection of human health from the toxic properties of mercury ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 144 ng/l.

For the protection of human health from the toxic properties of mercury ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 146 ng/l.

Note.—These values include the consumption of freshwater, estuarine, and marine species.

Naphthalene

Freshwater Aquatic Life

The available data to naphthalene indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 2,300 and 620 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for naphthalene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,350 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of naphthalene to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for naphthalene.

Nickel

Freshwater Aquatic Life

For total recoverable nickel the criterion (in μg/l) to protect freshwater aquatic life as derived using the Guidelines is the numerical value given by $e^{(0.76 [\ln (\text{hardness})] + 1.06)}$ as a 24-hour average and the concentration (in μg/l) should not exceed the numerical value given by $e^{(0.76[\ln (\text{hardness})] + 4.02)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as $CaCO_3$ the criteria are 56, 96, and 160 μg/l, respectively, as 24-hour averages, and the concentrations should not exceed 1,100, 1,800, and 3,100 μg/l, respectively, at any time.

Saltwater Aquatic Life

For total recoverable nickel the criterion to protect saltwater aquatic life as derived using the Guidelines is 7.1 μg/l as a 24-hour average and the concentration should not exceed 140 μg/l at any time.

Human Health

For the protection of human health from the toxic properties of nickel ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 13.4 μg/l.

For the protection of human health from the toxic properties of nickel ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 100 μg/l.

Nitrobenzene

Freshwater Aquatic Life

The available data for nitrobenzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 27,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of nitrobenzene to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for nitrobenzene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 6,680 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrobenzene to sensitive saltwater aquatic life.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for nitrobenzene. Based on available toxicity data, for the protection of public health, the derived level is 19.8 mg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 30 μg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Nitrophenols

Freshwater Aquatic Life

The available data for nitrophenols indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 230 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrophenols to sensitive freshwater aquatic life but toxicity to one species of algae occurs at concentrations as low as 150 μg/l.

Saltwater Aquatic Life

The available data for nitrophenols indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 4,850 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrophenols to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of 2,4-dinitro-o-cresol ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 13.4 μg/l.

For the protection of human health from the toxic properties of 2,4-dinitro-o-cresol ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 765 μg/l.

For the protection of human health from the toxic properties of dinitrophenol ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 70 μg/l.

For the protection of human health from the toxic properties of dinitrophenol ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 14.3 mg/l.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for mononitrophenol.

Using the present guidelines, a satisfactory criterion cannot be derived at this time due to the insufficiency in the available data for tri-nitrophenol.

Nitrosamines

Freshwater Aquatic Life

The available data for nitrosamines indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 5,850 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrosamines to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for nitrosamines indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 3,300,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrosamines to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of n-nitrosodimethylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 14 ng/l, 1.4 ng/l, and .14 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 160,000 ng/l, 16,000 ng/l, and 1,600 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of n-nitrosodiethylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 8 ng/l, 0.8 ng/l, and 0.08 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 12,400 ng/l, 1,240 ng/l, and 124 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure in n-nitrosodi-n-butylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 64 ng/l 6.4 ng/l and .064 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5,868 ng/l, 587 ng/l, and 58.7 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure in n-nitrosodiphenylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 49,000 ng/l 4,900 ng/l and 490 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 161,000 ng/l, 16,100 ng/l, and 1,610 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

For the maximum protection of human health from the potential carcinogenic effects due to exposure in n-nitrosopyrrolidine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk, over the lifetimes are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 160 ng/l 16.0 ng/l and 1.60 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 919,000 ng/l, 91,900 ng/l, and 9,190 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Pentachlorophenol

Freshwater Aquatic Life

The available data for pentachlorophenol indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 55 and 3.2 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for pentachlorophenol indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 53 and 34 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for pentachlorophenol. Based on available toxicity data, for the protection of public health, the derived level is 1.01 mg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 30 μg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Phenol

Freshwater Aquatic Life

The available data for phenol indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 10,200 and 2,560 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for phenol indicate that atute toxicity to saltwater aquatic life occurs at concentrations as low as 5,800 ug/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of phenol to sensitive saltwater aquatic life.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for phenol. Based on available toxicity data, for the protection of public health, the derived level is 3.5 mg/l. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 0.3 mg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criterion have limitations and have no demonstrated relationship to potential adverse human health effects.

Phthalate Esters

Freshwater Aquatic Life

The available data for phthalate esters indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 940 and 3 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for phthalate esters indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 2944 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of phthalate esters to sensitive saltwater aquatic life but toxicity to one species of algae occurs at concentrations as low as 3.4 µg/l.

Human Health

For the protection of human health from the toxic properties of dimethyl-phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 313 mg/l.

For the protection of human health from the toxic properties of dimethyl-phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 2.9 g/l.

For the protection of human health from the toxic properties of diethyl-phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 350 mg/l.

For the protection of human health from the toxic properties of diethyl-phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 1.8 g/l.

For the protection of human health from the toxic properties of dibutyl-phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 34 mg/l.

For the protection of human health from the toxic properties of dibutyl-phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 154 mg/l.

For the protection of human health from the toxic properties of di-2-ethylhexyl-phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 15 mg/l.

For the protection of human health from the toxic properties of di-2-ethylhexyl-phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 50 mg/l.

Polychlorinated Biphenyls

Freshwater Aquatic Life

For polychlorinated biphenyls the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.014 µg/l as a 24-hour average. The available data indicate that acute toxicity to freshwater aquatic life probably will only occur at concentrations above 2.0 µg/l and that the 24-hour average should provide adequate protection against acute toxicity.

Saltwater Aquatic Live

For polychlorinated biphenyls the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.030 µg/l as a 24-hour average. The available data indicate that acute toxicity to saltwater aquatic life probably will only occur at concentrations above 10 µg/l and that the 24-hour average should provide adequate protection against acute toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of PCBs through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are .79 ng/l, 0.79 ng/l, and .0079 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are .79 ng/l, .079 ng/l, and .0079 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Polynuclear Aromatic Hydrocarbons (PAHs)

Freshwater Aquatic Life

The limited freshwater data base available for polynuclear aromatic hydrocarbons, mostly from short-term bioconcentration studies with two compounds, does not permit a statement concerning acute or chronic toxicity.

Saltwater Aquatic Life

The available data for polynuclear aromatic hydrocarbons indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 300 ug/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of polynuclear aromatic hydrocarbons to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of PAHs through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 28 ng/l, 2.8 ng/l, and .28 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 311 ng/l, 31.1 ng/l, and 3.11 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Selenium

Freshwater Aquatic Life

For total recoverable inorganic selenite the criterion to protect freshwater aquatic life as derived using the Guidelines is 35 µg/l as a 24-hour average and the concentration should not exceed 260 µg/l at any time.

The available data for inorganic selenate indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 760 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of inorganic selenate to sensitive freshwater aquatic life.

Saltwater Aquatic Life

For total recoverable inorganic selenite the criterion to protect saltwater aquatic life as derived using the Guidelines is 54 μg/l as a 24-hour average and the concentration should not exceed 410 μg/l at any time.

No data are available concerning the toxicity of inorganic selenate to saltwater aquatic life.

Human Health

The ambient water quality criterion for selenium is recommended to be identical to the existing drinking water standard which is 10 μg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

Silver

Freshwater Aquatic Life

For freshwater aquatic life the concentration (in μg/l) of total recoverable silver should not exceed the numerical value given by "e[1.72(ln (hardness)-6.52)]" at any time. For example, at hardnesses of 50, 100, 200 mg/l as $CaCO_3$ the concentration of total recoverable silver should not exceed 1.2, 4.1, and 13 μg/l, respectively, at any time. The available data indicate that chronic toxicity to freshwater aquatic life may occur at concentrations as low as 0.12 μg/l.

Saltwater Aquatic Life

For saltwater aquatic life the concentration of total recoverable silver should not exceed 2.3 μg/l at any time. No data are available concerning the chronic toxicity of silver to sensitive saltwater aquatic life.

Human Health

The ambient water quality criterion for silver is recommended to be identical to the existing drinking water standard which is 50 μg/l. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

Tetrachloroethylene

Freshwater Aquatic Life

The available data for tetrachloroethylene indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 5,280 and 840 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Saltwater Aquatic Life

The available data for tetrachloroethylene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations low as 10,200 and 450 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of tetrachloroethylene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 8 μg/l, .8 μg/l, and .08 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 88.5 μg/l, 8.85 μg/l, and .88 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Thallium

Freshwater Aquatic Life

The available data for thallium indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 1,400 and 40 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to one species of fish occurs at concentrations as low as 20 μg/l after 2,600 hours of exposure.

Saltwater Aquatic Life

The available data for thallium indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,130 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of thallium to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of thallium ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 13 μg/l.

For the protection of human health from the toxic properties of thallium ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 48 μg/l.

Toluene

Freshwater Aquatic Life

The available data for toluene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 17,500 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of toluene to sensitive freshwater aquatic life.

Saltwater Aquatic Life

The available data for toluene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 6,300 and 5,000 μg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the protection of human health from the toxic properties of toluene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 14.3 mg/l.

For the protection of human health from the toxic properties of toluene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 424 mg/l.

Toxaphene

Freshwater Aquatic Life

For toxaphene the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.013 μg/l as a 24-hour average and the concentration should not exceed 1.6 μg/l at any time.

Saltwater Aquatic Life

For saltwater aquatic life the concentration of toxaphene should not exceed 0.070 μg/l at any time. No data are available concerning the chronic toxicity of toxaphene to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of toxaphene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 7.1 ng/l, .71 ng/l, and .07 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 7.3 ng/l, .73 ng/l, and .07 ng/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Trichloroethylene

Freshwater Aquatic Life

The available data for trichloroethylene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 45,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trichloroethylene to sensitive freshwater aquatic life but adverse behavioral effects occurs to one species at concentrations as low as 21,900 μg/l.

Saltwater Aquatic Life

The available data for trichloroethylene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,000 μg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trichloroethylene to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of trichloroethylene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 27 μg/l, 2.7 μg/l, and .27 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 807 μg/l, 80.7 μg/l, and 8.07 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Vinyl Chloride

Freshwater Aquatic Life

No freshwater organisms have been tested with vinyl chloride and no statement can be made concerning acute or chronic toxicity.

Saltwater Aquatic Life

No saltwater organisms have been tested with vinyl chloride and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects due to exposure of vinyl chloride through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5}, 10^{-6}, and 10^{-7}. The corresponding criteria are 20 μg/l, 2.0 μg/l, and .2 μg/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5,240 μg/l, 525 μg/l, and 52.5 μg/l, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

Zinc

Freshwater Aquatic Life

For total recoverable zinc the criterion to protect freshwater aquatic life as derived using the Guidelines is 47 μg/l as a 24-hour average and the concentration (in μg/l) should not exceed the numerical value given by $e^{(0.83 [\ln(\text{hardness})] + 1.95)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/l as $CaCO_3$ the concentration of total recoverable zinc should not exceed 180, 320, and 570 μg/l at any time.

Saltwater Aquatic Life

For total recoverable zinc the criterion to protect saltwater aquatic life as derived using the Guidelines is 58 μg/l as a 24-hour average and the concentration should not exceed 170 μg/l at any time.

Human Health

Sufficient data is not available for zinc to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 5 mg/l. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have not demonstrated relationship to potentia adverse human health effects.

Appendix B—Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses

Introduction

This version of the Guidelines provides clarifications, additional details, and technical and editorial changes in the last version published in the **Federal Register** [44 FR 15970 (March 15, 1979)]. This version incorporates changes resulting from comments on previous versions and from experience gained during U.S. EPA's use of the previous versions. Future versions of the Guidelines will incorporate new ideas and data as their usefulness is demonstrated.

Criteria may be expressed in several forms. The numerical form is commonly used, but descriptive and procedural forms can be used if numerical criteria are not possible or desirable. The purpose of these Guidelines is to describe an objective, internally consistent and appropriate way of deriving numerical water quality criteria for the protection of the uses of, as well as the presence of, aquatic organisms.

A numerical criterion might be thought of as an estimate of the highest concentration of a substance in water which does not present a significant risk to the aquatic organisms in the water and their uses. Thus the Guidelines are intended to derive criteria which will protect aquatic communities by protecting most of the species and their uses most of the time, but not necessarily all of the species all of the time. Aquatic communities can tolerate some stress and occasional adverse effects on a few species, and so total protection of all of the species all of the time is not necessary. Rather, the Guidelines attempt to provide a reasonable and adequate amount of protection with only a small possibility of considerable overprotection or underprotection. Within these constraints, it seems appropriate to err on the side of overprotection.

The numerical aquatic life criteria derived using the Guidelines are expressed as two numbers, rather than the traditional one number, so that the criteria can more accurately reflect toxicological and practical realities. The combination of both a maximum value and a 24-hour average value is designed to provide adequate protection of aquatic life and its uses from acute and chronic toxicity to animals, toxicity to plants and bioconcentration by aquatic organisms without being as restrictive as a one-number criterion would have to be to provide the same amout of protection. The only way to assure the same degree of protection with a one-number criterion would be to use the 24-hour average as a concentration that is not to be exceeded at any time in any place.

The two-number criterion is intended to identify an average pollutant concentration which will produce a water qualtiy generally suited to the maintenance of aquatic life and its uses while restricting the extent and duration of excursions over the average so that the total exposure will not cause unacceptable adverse effects. Merely specifying an average value over a time period is insufficient, unless the period of time is rather short, because of concentration higher than the average value can kill or cause substantial damage in short periods. Furthermore, for some substances the effect of intermittent high exposures is cumulative. It is therefore necessary to place an upper limit on pollutant concentrations to which aquatic organisms might be exposed, especially when the maximum value is not much higher than the average value. For some substances the maximum may be so much higher than the 24-hour average that in any real-world situation the maximum will never be reached if the 24-hour average is achieved. In such cases the 24-hour average will be limiting and the maximum will have no practical significance, except to indicate that elevated concentrations are acceptable as long as the 24-hour average is achieved.

These Guidelines have been developed on the assumption that the results of laboratory tests are generally useful for predicting what will happen in field situations. The resulting criteria are meant to apply to most bodies of water in the United States, except for the Great Salt Lake. All aquatic organisms and their common uses are meant to be considered, but not necessarily protected, if relevant data are available, with at least one specific exception. This exception is the accumulation of residues of organic compounds in the siscowet subspecies of lake trout which occurs in Lake Superior and contains up to 67% fat in the fillets (Thurston, C.E., 1962, Physical Characteristics and Chemical Composition of Two Subspecies of Lake Trout, J. Fish. Res. Bd. Canada 19:39–44). Neither siscowet nor organisms in the Great Salt Lake are intentionally protected by these Guidelines because both may be too atypical.

With appropriate modifications these Guidelines can be used to derive criteria for any specified geographical area, body of water (such as the Great Salt Lake), or group of similar bodies of water. Thus with appropriate modifications the Guidelines can be used to derive national, state, or local criteria if adequate information is available concerning the effects of the substance of concern on appropriate species and their uses. However, the basic concepts described in the Guidelines should be modified only when sound scientific evidence indicates that a criterion produced using the Guidelines would probably significantly overprotect or underprotect the presence or uses of aquatic life.

Criteria produced by these Guidelines are not enforceable numbers. They may be used in developing enforceable numbers, such as water quality standards and effluent standards. However, the development of standards may take into account additional factors such as social, legal, economic, and hydrological considerations, the environmental and analytical chemistry of the substance, the extrapolation from laboratory data to field situations, and the relationship between the species for which data are available and the species which are to be protected.

Because fresh water and salt water (including both estuarine and marine waters) have basically different chemical compositions and because freshwater and saltwater species rarely inhabit the same water simultaneously, separate criteria should be derived for these two kinds of waters. However, for some substances sufficient data may not be available to allow derivation of one or both of these criteria using the Guidelines.

These Guidelines are meant to be used after a decision is made that a criterion is needed for a substance. The Guidelines do not address the rationale for making that decision. If the potential for adverse effects on aquatic life and its uses are part of the basis for deciding whether or not a criterion is needed for a substance, these Guidelines may be helpful in the collection and interpretation of relevant data.

I. Define the Substance for Which the Criterion Is To Be Derived

A. Each separate chemical which would not ionize significantly in most natural bodies of water should usually be considered a separate substance, except possibly for structurally similar organic compounds that only differ in the number and location of atoms of a specific halogen, and only exist in large quantities as commercial mixtures of the various compounds, and apparently have similar chemical, biological, and toxicological properties.

B. For chemicals, which would ionize significantly in most natural bodies of water, such as inorganic salts, organic acids and phenols, all forms that would be in chemical equilibrium should usually be considered one substance. For metals, each different valence and each different covalently bonded organometallic compound should usually be considered a separate substance.

C. The definition of the substance may also need to take into account the analytical chemistry and fate of the substance.

II. Collect and Review Available Data

A. Collect all available data on the substance concerning (1) toxicity to, and bioaccumulation by, aquatic animals and plants, (2) FDA action levels, and (3) chronic feeding studies with wildlife.

B. Discard all data that are not available in hard copy (publication, manuscript, letter, memorandum, etc.) with enough supporting information to indicate that acceptable test procedures were used and that the results are reliable. Do not assume that all published data are acceptable.

C. Discard questionable data. For example, discard data from tests for which no control treatment existed, in which too many organisms in the control treatment died or showed signs of stress or disease, or in which distilled or deionized water was used as the dilution water for aquatic organisms. Discard data on formulated mixtures and emulsifiable concentrates of the substance of concern, but not necessarily data on technical grade material.

D. Do not use data obtained using:

1. Brine shrimp, because they usually only occur naturally in water with salinity greater than 35 g/kg.

2. Species that do not have reproducing wild populations resident in—but not necessarily native to—North America. Resident North American species of fishes are defined as those listed in "A List of Common and Scientific Names of Fishes from the United States and Canada", 3rd ed., Special Publication No. 6, American Fisheries Society, Washington, D.C., 1970. Data obtained with non-resident species can be used to indicate relationships and possible problem areas, but cannot be used in the derivation of criteria.

3. Organisms that were previously exposed to significant concentrations of the test material or other pollutants.

III. Minimum Data Base

A. A minimum amount of data should be available to help ensure that each of the four major kinds of possible adverse effects receives some consideration. Results of acute and chronic toxicity tests with a reasonable number and variety of aquatic animals are necessary so that data available for tested species can be considered a useful indication of the sensitivities of the numerous untested species. The requiurements concerning toxicity to aquatic plants are less stringent because procedures for conducting tests with plants are not as well developed and the interpretation of the results is more questionable. Data concerning bioconcentration by aquatic organisms can only be used if other relevant data are available.

B. To derive a criterion for freshwater aquatic life, the following should be available:

1. Acute tests (see Section IV) with freshwater animals in at least eight different families provided that of the eight species:

—at least one is a salmonid fish
—at least one is a non-salmonid fish
—at least one is a planktonic crustacean
—at least one is a benthic crustacean
—at least one is a benthic insect
—at least one of the benthic species is a detritivore

2. Acute-chronic ratios (see Section VI) for at least three species of aquatic animals provided that of the three species:

—at least one is a fish
—at least one is an invertebrate
—at least one is a freshwater species (the other two may be saltwater species)

3. At least one test with a freshwater alga or a chronic test with a freshwater vascular plant (see Section VIII). If plants are among the aquatic organisms that are most sensitive to the substance, tests with more than one species should be available.

4. At least one acceptable bioconcentration factor determined with an aquatic animal species, if a maximum permissible tissue concentration is available (see Section IX).

C. To derive a criterion for saltwater aquatic life, the following should be available:

1. Acute tests (see Section IV) with saltwater animals in at least eight different families provided that of the eight species:

—at least two different fish families are included
—at least five different invertebrate families are included
—either the Mysidae or Penaeidae family or both are included
—at least one of the invertebrate families is in a phylum other than Arthropoda

2. Acute-chronic ratios (see Section VI) for at least three species of aquatic animals provided that of the three species:

—at least one is a fish
—at least one is an invertebrate
—at least one is a saltwater species (the other two may be freshwater species)

3. At least one test with a saltwater alga or a chronic test with a saltwater vascular plant (see Section VIII). If plants are among the aquatic organisms most sensitive to the substance, tests with more than one species should be available.

4. At least one acceptable bioconcentration factor determined with an aquatic animal species, if a maximum permissible tissue concentration is available (see Section IX).

D. If all the requirements of the minimum data base are met, a criterion can usually be derived, except in special cases. For example, a criterion might not be possible if the acute-chronic ratios vary greatly with no apparent pattern. Also, if a criterion is to be related to a water quality characteristic, (see Sections V and VII), more data will be necessary.

Similarly, if the minimum data requirements are not satisfied, generally a criterion should not be derived, except in special cases. One such special case would be when less than the minimum amount of acute and chronic data are available, but the available data clearly indicate that the Final Residue Value would be substantially lower then either the Final Chronic Value or the Final Plant Value.

IV. Final Acute Value

A. Appropriate measures of the acute (short-term) toxicity of the substance to various species of aquatic animals are used to calculate the Final Acute Value. If acute values are available for fewer than twenty species, the Final Acute Value probably should be lower than the lowest value. On the other hand, if acute values are available for more than twenty species, the Final Acute Value probably should be higher than the lowest value, unless the most sensitive species is an important one. Although the procedure used to calculate the Final Acute Value has some limitations, it apparently is the best of the procedures currently available.

B. Acute toxicity tests should be conducted using procedures such as those described in:

ASTM Standard E 729–80, Practice for Conducting Acute Toxicity Tests with Fishes, Macroinvertebrates, and Amphibians. American Society for Testing and Materials, 1916 Race Street, Philadelphia, PA 19103.

ASTM Standard E 724–80, Practice for Conducting Static Acute Toxicity Tests with Larvae of Four Species of Bivalve Molluscs. American Society for Testing and Materials, 1916 Race Street, Philadelphia, PA 19103.

C. Results of acute tests in which food was added to the test solutions should not be used, because this may unnecessarily affect the results of the test.

D. Results of acute tests conducted with embryos should not be used (but see Section IV.E.2), because this is often an insensitive life stage.

E. Acute values should be based on endpoints and lengths of exposure appropriate to the life stage of the species tested. Therefore, only the following kinds of data on acute toxicity to aquatic animals should be used:

1. 48-hr EC50 values based on immobilization and 48-hr LC50 values for first-instar (less than 24 hours old) daphnids and other cladocerans, and second- or third-instar midge larvae.

2. 48- to 96-hr EC50 values based on incomplete shell development and 48- to 96-hr LC50 values for embryos and larvae of barnacles, bivalve molluscs (clams, mussels, oysters, and scallops), sea urchins, lobsters, crabs, shrimps, and abalones.

3. 96-hr EC50 values based on decreased shell deposition for oysters.

4. 96-hr EC50 values on immobilization or loss of equilibrium or both and 96-hr LC50 values for aquatic animals, except for cladocerans, midges, and animals whose behavior or physiology allows them to avoid

exposure to toxicant or for whom the acute adverse effect of the exposure cannot be adequately measured. Such freshwater and saltwater animals include air-breathing mollusca, unionid clams, operculate snails, and bivalve molluscs, except for some species that cannot "close up" and thus prevent exposure to toxicant, such as the bay scallop (*Argopecten irradians*).

F. For the use of LC50 or EC50 values for durations shorter and longer than those listed above, see Section X.

G. If the acute toxicity of the substance to aquatic animals has been shown to be related to a water quality characteristic such as hardness for freshwater organisms or salinity for saltwater organisms, a Final Acute Equation should be derived based on that water quality characteristic. Go to Section V.

H. If the acute toxicity of the substance has not been adequately shown to be related to a water quality characteristic, for each species for which at least one acute value is available, calculate the geometric mean of the results of all flow-through tests in which the toxicant concentrations were measured. For a species for which no such result is available, calculate the geometric mean of all available acute values, i.e., results of flow-through tests in which the toxicant concentrations were not measured and results of static and renewal tests based on initial total toxicant concentrations.

Note.—The geometric mean of N numbers is obtained by taking the N^{th} root of the product of N numbers. Alternatively, the geometric mean can be calculated by adding the logarithms of the N numbers, dividing the sum by N, and taking the antilog of the quotient. The geometric mean of two numbers can also be calculated as the square foot of the product of the two numbers. The geometric mean of one number is that number. Either natural (base e) or common (base 10) logarithms can be used to calculate geometric means as long as they are used consistently within each set of data, i.e., the antilog used must match the logarithm used.

I. Count the number = N of species for which a species mean acute value is available.

J. Order the species mean acute values from low to high. Take the common logarithms of the N values (log mean values).

K. The intervals (cell widths) for the lower cumulative proportion calculations are 0.11 common log units apart, starting from the lowest log value. The value of 0.11 is an estimate of average precision and was calculated from replicate species acute values.

L. Starting with the lowest log mean value, separate the N values into intervals (or cells) calculated in Step IV. K.

M. Calculate cumulative proportions for each non-empty interval by summing the number of values in the present and all lower intervals and dividing by N. These calculations only need to be done for the first three non-empty intervals (or cells).

N. Calculate the arithmetic mean of the log mean values for each of the three intervals.

O. Using the two interval mean acute values and cumulative proportions closest to 0.05, linearly extrapolate or interpolate to the 0.05 log concentration. The Final Acute Value is the antilog of the 0.05 concentration.

In other words, where

Prop(1) and conc(1) are the cumulative proportion and mean log value for the lowest non-empty interval.

Prop(2) and conc(2) are the cumulative proportion and mean log value for the second lowest non-empty interval.

A = Slope of the cumulative proportions

B = The 0.05 log value

Then:

A = [0.05 − Prop(1)]/[Prop(2) − Prop(1)]

B = conc(1) + A [conc(2) − conc(1)]

Final Acute Value = 10^{B}

P. If for an important species, such as a recreationally or commercially important species, the geometric mean of the acute values from flow-through tests in which the toxicant concentrations were measured is lower than the Final Acute Value, then that geometric mean should be used as the Final Acute Value.

Q. Go to Section VI.

V. Final Acute Equation

A. When enough data are available to show that acute toxicity to two or more species is similarly affected by a water quality characteristic, this effect can be taken into account as described below. Pooled regression analysis should produce similar results, although data available for individual species would be weighted differently.

B. For each species for which comparable acute toxicity values are available at two or more different values of a water quality characteristic which apparently affects toxicity, perform a least squares regression of the natural logarithms of the acute toxicity values on the natural logarithms of the values of the water quality characteristic. (Natural logarithms [logarithms to the base e, denoted as ln] are used herein merely because they are easier to use on some hand calculators and computers than common logarithms [logarithms to the base 10]. Consistent use of either will produce the same result.) No transformation or a different transformation may be used if it fits the data better, but appropriate changes will be necessary throughout this section.

C. Determine whether or not each acute slope is meaningful, taking into account the range and number of values of the water quality characteristic tested. For example, a slope based on four data points may be of limited value if it is based only on data for a narrow range of values of the water quality characteristic. On the other hand, a slope based on only two data points may be meaningful if it is consistent with other information and if the two points cover a broad enough range of the water quality characteristic. If meaningful slopes are not available for at least two species or if the available slopes are not similar, return to Section IV. H., using the results of tests conducted under conditions and in water similar to those commonly used for toxicity tests with the species.

D. Calculate the mean acute slope (V) as the arithmetic average of all the meaningful acute slopes for individual species.

E. For each species calculate the geometric mean (W) of the acute toxicity values and the geometric mean (X) of the related values of the water quality characteristic.

F. For each species calculate the logarithmic intercept (Y) using the equation: Y = ln W − V(ln X).

G. For each species calculate the species mean acute intercept as the antilog of Y.

H. Obtain the Final Acute Intercept by using the procedure described in Section IV. I–O, except insert "Intercept" for "Value".

I. If for an important species, such as a recreationally or commercially important species, the intercept calculated only from results of flow-through tests in which the toxicant concentrations were measured is lower than the Final Acute Intercept, then that intercept should be used as the Final Acute Intercept.

J. The Final Acute Equation is written as $e^{(V[\ln(\text{water quality characteristic})]+\ln Z)}$, where V = mean acute slope and Z = Final Acute Intercept.

VI. Final Chronic Value

A. The Final Chronic Value can be calculated in the same manner as the Final Acute Value or by dividing the Final Acute Value by the Final Acute-Chronic Ratio, depending on the data available. In some cases it will not be possible to calculate a Final Chronic Value.

B. Use only the results of flow-through (except renewal is acceptable for daphnids) chronic tests in which the concentrations of toxicant in the test solutions were measured.

C. Do not use the results of any chronic test in which survival, growth, or reproduction among the controls was unacceptably low.

D. Chronic values should be based on endpoints and lengths of exposure appropriate to the species. Therefore, only the results of the following kinds of chronic toxicity tests should be used:

1. Life-cycle toxicity tests consisting of exposures of each of several groups of individuals of a species to a different concentration of the toxicant throughout a life cycle. To ensure that all life stages and life processes are exposed, the test should begin with embryos or newly hatched young less than 48 hours old (less than 24 hours old for daphnids), continue through maturation and reproduction, and with fish should end not less than 24 days (90 days for salmonids) after the hatching of the next generation. For fish, data should be obtained and analyzed on survival and growth of adults and young, maturation of males and females, embryos spawned per female, embryo viability (salmonids only) and hatchability. For daphnids, data should be obtained and analyzed on survival and young per female.

2. Partial life-cycle toxicity tests consisting of exposures of each of several groups of individuals of a species of fish to a different concentration of the toxicant through most portions of a life cycle. Partial life-cycle tests are conducted with fish species that require more than a year to reach sexual maturity, so that the test can be completed in less than 15 months, but still expose all major life stages to the toxicant. Exposure to the toxicant begins with immature juveniles at least 2 months prior to active gonad development, continues through maturation and reproduction, and ends not less than 24 days (90 days for salmonids) after the hatching of the next generation. Data should be obtained and analyzed on survival and growth of adults and young, maturation of males and females, embryos spawned per female, embryo viability (salmonids only) and hatchability.

3. Early-life-stage toxicity tests consisting of 28- to 32-days (60 days post-hatch for salmonids) exposures of the early life stages of a species of fish from shortly after fertilization through embryonic, larval, and early juvenile development. Data should be obtained and analyzed on survival and growth.

E. Do not use the results of an early-life-stage test if results of a life-cycle or partial life-cycle test with the same species are available.

F. A chronic value is obtained by calculating the geometric mean of the lower and upper chronic limits from a chronic test. A lower chronic limit is the highest tested concentration (1) in an acceptable chronic test, (2) which did not cause the occurrence (which was statistically significantly different from the control at $p = 0.05$) of a specified adverse effect, and (3) below which no tested concentration caused such an occurrence. An upper chronic limit is the lowest tested concentration (1) in an acceptable chronic test, (2) which did cause the occurrence (which was statistically significantly different from the control at $p = 0.05$) of a specified adverse effect and (3) above which all tested concentrations caused such an occurrence.

Note.—Various authors have used a variety of terms and definitions to interpret the results of chronic tests, so reported results should be reviewed carefully.

G. If the chronic toxicity of the substance to aquatic animals has been adequately shown to be related to a water quality characteristic such as hardness for freshwater organisms or salinity for saltwater organisms, a Final Chronic Equation should be derived based on that water quality characteristic. Go to Section VII.

H. If chronic values are available for eight species as described in Section III. B.1 or III. C.1, a species mean chronic value should be calculated for each species for which at least one chronic value is available by calculating the geometric mean of all the chronic values for the species. The Final Chronic Value should then be obtained using the procedures described in Section IV. I–O. Then go to Section VI. M.

I. For each chronic value for which at least one appropriate acute value is available, calculate an acute-chronic ratio, using for the numerator the arithmetic average of the results of all standard flow-through acute tests in which the concentrations were measured and which are from the same study as the chronic test. If such an acute test is not available, use for the numerator the results of a standard acute test performed at the same laboratory with the same species, toxicant and dilution water. If no such acute test is available, use the species mean acute value for the numerator.

Note.—If the acute toxicity or chronic toxicity or both of the substance have been adequately shown to be related to a water quality characteristic, the numerator and the denominator must be based on tests performed in the same water.

J. For each species, calcuate the species mean acute-chronic ratio as the geometric mean of all the acute-chronic ratios available for that species.

K. For some substances the species mean acute-chronic ratio seems to be the same for all species, but for other substances the ratio seems to increase as the species mean acute value increases. Thus the Final Acute-Chronic Ratio can be obtained in two ways, depending on the data available.

1. If no major trend is apparent and the acute-chronic ratios for a number of species are within a factor of ten, the final Acute-Chronic Ratio should be calculated as the geometric mean of all the species mean acute-chronic ratios available for both freshwater and saltwater species.

2. If the species mean acute-chronic ratio seems to increase as the species mean acute value increases, the value of the acute-chronic ratio for species whose acute values are close to the Final Acute Value should be chosen as the Final Acute-Chronic Ratio.

L. Calculate the Final Chronic Value by dividing the Final Acute Value by the Final Acute-Chronic Ratio.

M. If the species mean chronic value of an important species, such as a commercially or recreationally important species, is lower than the Final Chronic Value, then that species mean chronic value should be used as the Final Chronic Value.

N. Go to Section VIII.

VII. Final Chronic Equation

A. For each species for which comparable chronic toxicity values are available at two or more different values of a water quality characteristic which apparently affects chronic toxicity, perform a least squares regression of the natural logarithms of the chronic toxicity values on the natural logarithms of the water quality characteristic values. No transformation or a different transformation may be used if it fits the data better, but appropriate changes will be necessary throughout this section. It is probably preferable, but not necessary, to use the same transformation that was used with the acute values in Section V.

B. Determine whether or not each chronic slope is meaningful, taking into account the range and number of values of the water quality characteristic tested. For example, a slope based on four data points may be of limited value if it is based only on data for a narrow range of values of the water quality characteristic. On the other hand, a slope based on only two data points may be meaningful if it is consistent with other information and if the two points cover a broad enough range of the water quality characteristic. If a meaningful chronic slope is not available for at least one species, return to Section VI. H.

C. Calculate the mean chronic slope (L) as the arithmetic average of all the meaningful chronic slopes for individual species.

D. For each species calculate the geometric mean (M) of the toxicity values and the geometric mean (P) of the related values of the water quality characteristic.

E. For each species calculate the logarithmic intercept (Q) using the equation: $Q = \ln M - L(\ln P)$.

F. For each species calculate a species mean chronic intercept as the antilog of Q.

G. Obtain the Final Chronic Intercept by using the procedure described in Section IV. I–O, except insert "Intercept" for "Value".

H. If the species mean chronic intercept of an important species, such as a commercially or recreationally important species, is lower than the Final Chronic Intercept, then that species mean chronic intercept should be used as the Final Chronic Intercept.

I. The Final Chronic Equation is written as $e^{(L \ln(\text{Water quality characteristic}) + \ln R)}$, where L = mean chronic slope and R = Final Chronic Intercept.

VIII. Final Plant Value

A. Appropriate measures of the toxicity of the substance to aquatic plants are used to compare the relative sensitivities of aquatic plants and animals.

B. A value is a concentration which decreased growth (as measured by dry weight, chlorophyll, etc.) in a 96-hr or longer test with an alga or in a chronic test with an aquatic vascular plant.

C. Obtain the Final Plant Value by selecting the lowest plant value from a test in which the toxicant concentrations were measured.

IX. Final Residue Value

A. The Final Residue Value is derived in order to (1) prevent commercially or recreationally important aquatic organisms from exceeding relevant FDA action levels and (2) protect wildlife, including fishes and birds, that eat aquatic organisms from demonstrated adverse effects. A residue value is calculated by dividing a maximum permissible tissue concentration by an appropriate bioconcentration factor (BCF), where the BCF is the quotient of the concentration of a substance in all or part of an aquatic organism divided by the concentration in water to which the organism has been exposed. A maximum permissible tissue concentration is either (1) an action level from the FDA Administrative Guidelines Manual for fish oil or for the edible portion of fish or shellfish, or (2) a maximum acceptable dietary intake based on observations on survival, growth or reproduction in a chronic wildlife feeding study. If no maximum permissible tissue concentration is available, go to Section X because no Final Residue Value can be derived.

B. 1. A BCF determined in a laboratory test should be used only if it was calculated based on measured concentrations of the substance in the test solution and was based on an exposure that continued until either steady-state or 28-days was reached. Steady-state is reached when the BCF does not change significantly over a period of time, such as two days or 16 percent of the length of the exposure, whichever is longer. If a steady-state BCF is not available for a species, the available BCF for the longest exposure over 28 days should be used for that species.

2. A BCF from a field exposure should be used only when it is known that the concentration of the substance was reasonably constant for a long enough period of time over the range of territory inhabited by the organisms.

3. If BCF values from field exposures are consistently lower or higher than those from laboratory exposures, then only those values from field exposures should be used if possible.

4. A BCF should be calculated based on the concentration of the substance and its metabolites, which are structurally similar and are not much more soluble in water than the parent compound, in appropriate tissue and should be corrected for the concentration in the organisms at the beginning of the test.

5. A BCF value obtained from a laboratory or field exposure that caused an observable adverse effect on the test organism may be used only if it is similar to that obtained with unaffected organisms at lower concentrations in the same test.

6. Whenever a BCF is determined for a lipid-soluble substance, the percent lipids should also be determined in the tissue for which the BCF was calculated.

C. A BCF calculated using dry tissue weights must be converted to a wet tissue weight basis by multiplying the dry weight BCF value by 0.1 for plankton and by 0.2 for individual species of fishes and invertebrates.

Note.—The values of 0.2 and 0.1 were derived from data published in:

McDiffett, W. F., 1970. Ecology 51:975–988.

Brocksen, R. W., et al. 1968. J. Wildlife Management 32:53–75.

Cummins, K. W., et al. 1973. Ecology 54: 336–345.

Pesticide Analytical Manual, Volume I, Food and Drug Administration, 1969.

Love, R. M., 1957. In The Physiology of Fishes, Vol. I, M. E. Brown, ed. Academic Press, New York. p. 411.

Ruttner, F., 1963. Fundamentals of Limnology. 3rd ed. Trans. by D. G. Frey and F. E. J. Fry. Univ. of Toronto Press, Toronto.

Some additional values can be found in:

Sculthorpe, C. D., 1967. The Biology of Aquatic Vascular Plants. Arnold Publishing Ltd., London.

D. If enough pertinent data exist, several residue values can be calculated by dividing maximum permissible tissue concentrations by appropriate BCF values.

1. For each available maximum acceptable dietary intake derived from a chronic feeding study with wildlife, including birds and aquatic organisms, the appropriate BCF is based on the whole body of aquatic species which constitute or represent a major portion of the diet of the tested wildlife species.

2. For an FDA action level, the appropriate BCF is the highest geometric mean species BCF for the edible portion (muscle for decapods, muscle with or without skin for fishes, adductor muscle for scallops and total living tissue for other bivalve molluscs) of a consumed species. The highest species BCF is used because FDA action levels are applied on a species-by-species basis.

E. For lipid-soluble substances, it may be possible to calculate additional residue values. Because steady-state BCF values for a lipid-soluble chemical seem to be proportional to percent lipids from one tissue to another and from one species to another, extrapolations can be made from tested tissues or species to untested tissues or species on the basis of percent lipids.

1. For each BCF for which the percent lipids is known for the same tissue for which the BCF was measured, the BCF should be normalized to a one percent lipid basis by dividing the BCF by the percent lipids. This adjustment to a one percent lipid basis makes all the measured BCF values comparable regardless of the species or tissue for which the BCF was measured.

2. Calculate the geometric mean normalized BCF. Data for both saltwater and freshwater species can be used to determine the mean normalized BCF, because the normalized BCF seems to be about the same for both kinds of organisms.

3. Residue values can then be calculated by dividing the maximum permissible tissue concentrations by the mean normalized BCF and by a percent lipids value appropriate to the maximum permissible tissue concentration, *i.e.*,

$$\text{Residue Value} = \frac{\text{(maximum permissible tissue concentration)}}{\text{(mean normalized BCF)(appropriate percent lipids)}}$$

a. For an FDA action level for fish oil, the appropriate percent lipids value is 100.

b. For an FDA action level for fish, the appropriate percent lipids value is 15 for freshwater criteria and 16 for saltwater criteria because FDA action levels are applied on a species-by-species basis to commonly consumed species. The edible portion of the freshwater lake trout averages about 15 percent lipids, and the edible portion of the saltwater Atlantic herring averages about 16 percent lipids (Sidwell, V. D., et al. 1974 Composition of the Edible Portion of Raw (Fresh or Frozen) Crustaceans, Finfish, and Mollusks. I. Protein, Fat, Moisture, Ash, Carbohydrate, Energy Value, and Cholesterol. Marine Fisheries Review 36:21–35).

c. For a maximum acceptable dietary intake derived from a chronic feeding study with wildlife, the appropriate percent lipids is the percent lipids of an aquatic species or group of aquatic species which constitute a major portion of the diet of the wildlife species.

F. The Final Residue Value is obtained by selecting the lowest of the available residue values. It should be noted that in many cases the Final Residue Value will not be low enough. For example, a residue value calculated from an FDA action level would result in an average concentration in the edible portion of a fatty species that is at the action level. On the average half of the individuals of the species would have concentrations above the FDA action level. Also, the results of many chronic feeding studies are concentrations that cause adverse effects.

X. Other Data

Pertinent information that could not be used in earlier sections may be available concerning adverse effects on aquatic organisms and their uses. The most important of these are data on flavor impairment, reduction in survival, growth, or reproduction, or any other adverse effect that has been shown to be biologically significant. Especially important are data for species for which no other data are available. Data from behavioral, micorcosm, field, and physiological studies may also be available.

XI. Criterion

A. The criterion consists of two concentrations, one that should not be exceeded on the average in a 24-hour period and one that should not be exceeded at any time during the 24-hour period. This two-number criterion is intended to identify water quality conditions that should protect aquatic life and its uses from acute and chronic adverse effects of both cumulative and noncumulative substances without being as restrictive as a one-number criterion would have to be to provide the same degree of protection.

B. The maximum concentration is the Final Acute Value or is obtained from the Final Acute Equation.

C. The 24-hour average concentration is obtained from the Final Chronic Value, the Final Plant Value, and the Final Residue Value by selecting the lowest available value, unless other data (see Section X) from tests in which the toxicant concentrations were measured show that a lower value should be used. If toxicity is related to a water quality characteristic, the 24-hour average concentration is obtained from the Final Chronic Equation, the Final Plant Value, and the Final Residue Value by selecting the one that results in the lowest concentrations in the normal range of the water quality characteristic, unless other data (see Section X) from tests in which the toxicant concentrations were measured show that a lower value should be used.

D. The criterion is (the 24-hour average concentration) as a 24-hour average and the concentration should not exceed (the maximum concentration) at any time.

XII. Review

A. On the basis of all available pertinent laboratory and field information, determine if the criterion is consistent with sound scientific evidence. If it is not, another criterion, either higher or lower, should be derived using appropriate modifications of the Guidelines.

These Guidelines were written by Charles E. Stephan, Donald I. Mount, David J. Hansen, John H. Gentile, Gary A. Chapman and William A. Brungs of the U.S.E.P.A. Environmental Research Laboratories in Corvallis, Oregon, Duluth, Minnesota, Gulf Breeze, Florida, and Narragansett, Rhode Island. Numerous other people, many of whom do not work for U.S.E.P.A., provided assistance and suggestions.

Appendix C—Guidelines and Methodology Used in the Preparation of Health Effect Assessment Chapters of the Consent Decree Water Criteria Documents

I. Objective

The objective of the health effect assessment chapters of the ambient water criteria documents is to estimate ambient water concentrations which do not represent a significant risk to the public. These assessments should constitute a review of all relevant information on individual chemicals or chemical classes in order to derive criteria that represent, in the case of suspect or proven carcinogens, various levels of incremental cancer risk, or, in the case of other pollutants, estimates of no-effect levels.

Ideally, ambient water quality criteria should represent levels for compounds in ambient water that do not pose a hazard to the human population. However, in any realistic assessment of human health hazard, a fundamental distinction must be made between absolute safety and the recognition of some risk. Criteria for absolute safety would have to be based on detailed knowledge of dose-response relationships in humans, including all sources of chemical exposure, the types of toxic effects elicited, the existence of thresholds for the toxic effects, the significance of toxicant interactions, and the variances of sensitivities and exposure levels within the human population. In practice, such absolute criteria cannot be established because of deficiencies in both the available data and the means of interpreting this information. Consequently, the individual human health effects chapters propose criteria which minimize or specify the potential risk of adverse human effects due to substances in ambient water. Potential social or economic costs and benefits are not considered in the formulation of the criteria.

II. Types of Criteria

Ambient water quality criteria are based on three types of biological endpoints: carcinogenicity, toxicity (i.e., all adverse effects other than cancer), and organoleptic effects.

For the purpose of deriving ambient water quality criteria, carcinogenicity is regarded as a non-threshold phenomenon. Using this assumption, "safe" or "no effect" levels for carcinogens cannot be established because even extremely small doses must be assumed to elicit a finite increase in the incidence of the response. Consequently, water quality criteria for carcinogens are presented as a range of pollutant concentrations associated with corresponding incremental risks.

For compounds which do not manifest any apparent carcinogenic effect, the threshold assumption is used in deriving a criterion. This assumption is based on the premise that a physiological reserve capacity exists within the organism which is thought to be depleted before clinical disease ensues. Alternatively, it may be assumed that the rate of damage will be insignificant over the life span of the organism. Thus, ambient water quality criteria are derived for non-carcinogenic chemicals, and presumably result in no observable-adverse-effect levels (NOAELs) in the exposed human population.

In some instances, criteria are based on organoleptic characteristics, i.e., thresholds for taste or odor. Such criteria are established when insufficient information is available on toxicologic effects or when the estimate of the level of the pollutant in ambient water based on organoleptic effects is lower than the level calculated from toxicologic data. It should be recognized that criteria based solely on organoleptic effects do not necessarily represent approximations of acceptable risk levels for human health.

Several ambient water quality criteria documents deal with classes of compounds which include chemicals exhibiting varying degrees of structural similarity. Because prediction of biological effects based solely on structural parameters is difficult, the derivation of compound-specific criteria is preferable to a class criterion. A compound-specific criterion is defined as a level derived from data on each individual subject compound that does not represent a significant risk to the public. For some chemical classes, however, a compound-specific criterion cannot be derived for each member of a class. In such instances, it is sometimes justifiable to derive a class criterion in which available data on one member of a class may be used to estimate criteria for other chemicals of the class because a sufficient data base is not available for those compounds.

For some chemicals and chemical classes, the data base was judged to be insufficient for the derivation of a criterion. In those cases, deficiencies in the available information are detailed.

III. Approach

The human health effects chapters attempt to summarize all information on the individual chemicals or classes of chemicals which might be useful in the risk assessment process to develop water quality criteria. Although primary emphasis is placed on identifying epidemiologic and toxicologic data, these assessments typically contain discussions on four topics: existing levels of human exposure, pharmacokinetics, toxic effects, and criterion formulation.

For all documents, an attempt is made to include the known relevant information. Review articles and reports are often used in the process of data evaluation and synthesis. Scientific judgment is exercised in the review and evaluation of the data in each document and in the identification of the adverse effects against which protective criteria are sought. In addition, each of these documents is reviewed by a peer committee of scientists familiar with the specific compound(s). These work groups evaluate the quality of the available data, the completeness of the data summary, and the validity of the derived criterion.

In the analysis and organization of the data, an attempt is made to be consistent with respect to the format and the application of acceptable scientific principles. Evaluation procedures used in the hazard assessment process follow the principles outlined by the National Academy of Sciences in *Drinking Water and Health* (1977) and the guidelines of the Carcinogen Assessment Group of the U.S. EPA.

A. Exposure

The exposure section of the health effects chapters reviews known information on current levels of human exposure to the individual pollutant from all sources. Much of the data was obtained from monitoring studies of air, water, food, soil, and human or animal tissue residues. The major purpose of this section is to provide background information on the contribution of water exposure relative to all other sources. Consequently, the exposure section includes subsections reviewing different routes of exposure including water and food ingestion, inhalation, and dermal contact.

Information on exposure can be valuable in developing and assessing a water quality criterion. In these documents exposure from consumption of contaminated water and contaminated fish and shellfish products is used in criterion formulation. Data for all modes of exposure are useful in relating total intake to the expected contribution from contaminated water, fish, and shellfish. In addition, information for all routes of exposure, not limited to drinking water and fish and shellfish ingestion, can be used to justify or assess the feasibility of the formulation of criteria for ambient water.

The use of fish consumption as an exposure factor requires the quantitation of pollutant residues in the edible portions of the ingested species. Accordingly, bioconcentration factors (BCFs) are used to relate pollutant residues in aquatic organisms to the pollutant concentration in the ambient waters in which they reside.

To estimate the average per capita intake of a pollutant due to consumption of contaminated fish and shellfish the results of a diet survey were analyzed to calculate the average consumption of freshwater and estuarine fish and shellfish (U.S. EPA, 1980). A species is considered to be a consumed freshwater or estuarine fish and shellfish species if at some stage in its life cycle, it is harvested from fresh or estuarine water for human consumption in significant quantities (Stephan, 1980).

Three different procedures are used to estimate the weighted average BCF depending upon the lipid solubility of the chemical and the availability of bioconcentration data.

For lipid-soluble compounds, the average BCF is calculated from the weighted average percent lipids in the edible portions of consumed freshwater and estuarine fish and shellfish which was calculated from data on consumption of each species and its corresponding percent lipids to be 3.0 percent (Stephan, 1980). Because the steady-state BCFs for lipid-soluble compounds are proportional to percent lipids, bioconcentration factors for fish and shellfish can be adjusted to the average percent lipids for aquatic organisms consumed by Americans. For many lipid-soluble pollutants, there exists at least one BCF for which the percent lipid value was measured for the tissues for which the BCF is determined.

With 3.0 percent as the weighted average percent lipids for freshwater and estuarine fish and shellfish in the average diet, a BCF, and a corresponding percent lipid value, the weighted average bioconcentration factor can be calculated.

Example:

Weighted average percent lipids for average diet = 3.0 percent

Measured BCF of 17 for trichloroethylene with bluegills at 4.8 percent lipids

Weighted average BCF for average diet equals

$$17 \times \frac{3.0\%}{4.8\%} = 10.6$$

As an estimate, 10.6 is used for the BCF.

In those cases where an appropriate bioconcentration factor is not available, the equation "$\text{Log BCF} = (0.85 \text{ Log P}) - 0.70$" can be used (Veith, et al. 1979) to estimate the BCF for aquatic organisms containing about 7.6 percent lipids (Veith, 1980) from the octanol/water partition coefficient P. An adjustment for percent lipids in the average diet versus 7.6 percent is made in order to derive the weighted average bioconcentration factor.

For non-lipid-soluble compounds, the available BCFs for the edible portion of consumed freshwater and estuarine fish and shellfish are weighted according to consumption factors to determine a weighted BCF representative of the average diet.

B. Pharmacokinetics

This section summarizes the available information on the absorption, distribution, metabolism, and elimination of the compound(s) in humans and experimental mammals. Conceptually, such information is useful in validation of inter- and intraspecies extrapolations, and in characterizing the modes of toxic action. Sufficient information on absorption and excretion in animals, together with a knowledge of ambient concentrations in water, food, and air, could be useful in estimating body burdens of chemicals in the human population. Distribution data which suggest target organs or tissues are desirable for interspecies comparison techniques. In terms of the derivation of criteria, pharmacokinetic data are essential to estimate equivalent oral doses based on data from inhalation or other routes of exposure.

C. Effects

This section summarizes information on biological effects in both humans and experimental mammals resulting in: acute, subacute, and chronic toxicity, synergism and/or antagonism, teratogenicity, mutagenicity, or carcinogenicity.

The major goal of this section is to survey the suitability of the data for use in assessment of hazard and to determine which biological end-point, i.e., non-threshold, threshold, or organoleptic, should be selected for use in criterion formulation.

Because this section attempts to assess potential human health effects, data on documented human effects are thoroughly evaluated. However, several factors inherent in human epidemiological studies usually preclude the use of such data in generating water quality criteria. These problems, as summarized by the National Academy of Sciences (NAS, 1977) are as follows:

1. Epidemiology cannot tell what effects a material will have until after humans have been exposed. One must not conduct what might be hazardous experiments on man.
2. If exposure has been ubiquitous, it may be impossible to assess the effects of a material, because there is no unexposed control group. Statistics of morbidity obtained before use of a new material can sometimes be useful, but when latent periods are variable and times of introduction and removal of materials overlap, historical data on chronic effects are usually unsatisfactory.
3. It is usually difficult to determine doses in human exposures.
4. Usually, it is hard to identify small changes in common effects, which may nonetheless be important if the population is large.
5. Interactions in a "nature-designed" experiment usually cannot be controlled.

Although these problems often prevent the use of epidemiological data in quantitative risk assessments, qualitative similarities or differences between documented effects in humans and observed effects in experimental mammals are extremely useful in testing the validity of animal-to-man extrapolations. Consequently, in each case, an attempt is made to identify and utilize both epidemiologic and animal dose-response data. Criteria derived from such a confirmed data base are considered to be reliable.

The decision to establish a criterion based on a non-threshold model is made after evaluating all available information on carcinogenicity and supportive information on mutagenicity. The approach and conditions for the qualitative decision of carcinogenicity are outlined in the U.S. EPA Interim Cancer Guidelines (41 FR 21402), in a report by Albert, et al. (1977), and in the Interagency Regulatory Liaison Group (IRLG) guidelines on carcinogenic risks (IRLG, 1979). It is assumed that a substance which induces a statistically significant carcinogenic response in animals has the capacity to cause cancer in humans. A chemical which has not induced a significant cancer response in humans or experimental animals is not identified as a carcinogen, even though its metabolites or close structural analogues might induce a carcinogenic response or it was shown to be mutagenic in an *in vitro* system.

It is recognized that some potential human carcinogens may not be identified by the guidelines given above. For example, compounds for which there is plausible but weak qualitative evidence of carcinogenicity in experimental animal systems (such as data from mouse skin painting or strain A mouse pulmonary adenoma) would be included in this category. The derivation of a criterion for human consumption from these studies in not valid, regardless of the qualitative outcome. In addition, there are certain compounds (e.g., nickel and beryllium) which were shown to be carcinogenic in humans after inhalation exposure by chemical form, but have induced thus far no response in animals or humans via ingesting their soluble salts. Nevertheless, a non-threshold criterion is developed for beryllium because tumors have been produced in animals at a site removed from the site of administration; in contrast, a threshold criterion is recommended for nickel because there is no evidence of tumors at sites distant resulting from administration of nickel solutions by either ingestion or injection.

For those compounds which were not reported to induce carcinogenic effects or for those compounds for which carcinogenic data are lacking or insufficient, an attempt is made to estimate a no-effect level. In many respects, the hazard evaluation from these studies is similar to that of bioassays for carcinogenicity. In order to more closely approximate conditions of human exposure, preference is given to chronic studies involving oral exposures in water or diet over a significant portion of the animal life span. Greatest confidence is placed in those studies which demonstrate dose-related adverse effects as well as no-effect levels.

There is considerable variability in the biological endpoints used to define a no-effect level. They may range from gross effects, such as mortality, to more subtle biochemical, physiological, or pathological changes. Teratogenicity, reproductive impairment, and behavioral effects are significant toxic consequences of environmental contamination. In instances where carcinogenic or other chronic effects occur at exposure levels below those causing teratogenicity, reproductive impairment, or behavioral effects, the former are used in deriving the criterion. For most of the compounds evaluated thus far, teratogenicity and reproductive impairment occur at doses near maximum tolerated levels with dose administration schedules well above estimated environmental exposure levels. Moreover, information on behavioral effects, which could be of significance, is not available for most of the compounds under study. Consequently, most NOAELs derived from chronic studies are based either on gross toxic effects or on effects directly related to functional impairment or defined pathological lesions.

For compounds on which adequate chronic toxicity studies are not available, studies on acute and subacute toxicity assume greater significance. Acute toxicity studies usually involve single exposures at lethal or near lethal doses. Subacute studies often involve exposures exceeding 10 percent of the life span of the test organism, e.g., 90 days for the rat with an average life span of 30 months. Such studies are useful in establishing the nature of the compound's toxic effects and other parameters of compound toxicity, such as target organ effects, metabolic behavior, physiological/biochemical effects, and patterns of retention and tissue distribution. The utility of acute and subacute studies in deriving environmentally meaningful NOELs is uncertain, although McNamara (1976) has developed application factors for such derivations.

In some cases where adequate data are not available from studies utilizing oral routes of administration, no-effect levels for oral exposures may be estimated from dermal or inhalation studies. Such estimates involve approximations of the total dose administered based on assumptions about breathing rates and/or magnitude of absorption.

D. Criterion Rationale

This section reviews existing standards for the chemical(s), summarizes data on current levels of human exposure, attempts to identify special groups at risk, and defines the basis for the recommended criterion.

Information on existing standards is included primarily for comparison with the proposed water quality criteria. Some of the present standards, such as those recommended by the Occupational Safety and Health Administration (OSHA) or the American Conference of Governmental Industrial Hygienists (ACGIH), are based on toxicologic data but are intended as acceptable levels for occupational rather than environmental exposure. Other levels, such as those recommended by the National Academy of Sciences in *Drinking Water and Health* (1977) or in the U.S. EPA Interim Primary Drinking Water Standards, are more closely related to proposed water quality criteria. Emphasis is placed on detailing the basis for the existing standards wherever possible.

Summaries of current levels of human exposure, presented in this section, specifically address the suitability of the data to derive water quality criteria. The identification of special groups at risk, either because of geographical or occupational differences in exposure or biological differences in susceptibility to the compound(s), focuses on the impact that these groups should have on the development of water quality criteria.

The basis for the recommended criteria section summarizes and qualifies all of the data used in developing the criteria.

IV. Guidelines for Criteria Derivation

The derivation of water quality criteria from laboratory animal toxicity data is essentially a two-step procedure. First, a total daily intake for humans must be estimated which establishes either a defined level of risk for non-threshold effects or a no-effect level for threshold effects. Secondly, assumptions must be made about the contribution of contaminated water and the consumption of fish/shellfish to the total daily intake of the chemical. These estimates are then used to establish the tolerable daily intake and consequently the water quality criterion.

A. Non-Threshold Effects

After the decision has been made that a compound has the potential for causing cancers in humans and that data exist which permit the derivation of a criterion, the water concentration which is estimated to cause a lifetime carcinogenic risk of 10^{-5} is determined. The lifetime carcinogenicity risk is the probability that a person would get cancer sometime in his or her life assuming continuous exposure to the compound. The water concentration is calculated by using the low-dose extrapolation procedure proposed by Crump (1980). This procedure is an improvement on the multistage low dose extrapolation procedure by Crump, et al. (1977).

The data used for quantitative estimates are of two types: (1) lifetime animal studies, and (2) human studies where excess cancer risk has been associated with exposure to the agent. In animal studies it is assumed, unless evidence exists to the contrary, that if a carcinogenic response occurs at the dose levels used in the study, then proportionately lower responses will also occur at all lower doses, with an incidence determined by the extrapolation model discussed below.

1. Choice of Model

There is no really solid scientific basis for any mathematical extrapolation model which relates carcinogen exposure to cancer risks at the extremely low levels of concentration that must be dealt with in evaluating the environmental hazards. For practical reasons, such low levels of risk cannot be measured directly either using animal experiments or epidemiologic studies. We must, therefore, depend on our current understanding of the mechanisms of carcinogenesis for guidance as to which risk model to use. At the present time, the dominant view of the carcinogenic process involves the concept that most agents which cause cancer also cause irreversible damage to DNA. This position is reflected by the fact that a very large proportion of agents which cause cancer are also mutagenic. There is reason to expect that the quantal type of biological response that is characteristic of mutagenesis is associated with a linear non-threshold dose-response relationship. Indeed, there is substantial evidence from mutagenesis studies with both ionizing radiation and with a wide variety of chemicals that this type of dose-response model is the appropriate one to use. This is particularly true at the lower end of the dose-response curve; at higher doses, there can be an upward curvature, probably reflecting the effects of multistage processes on the mutagenic response. The linear non-threshold dose-response relationship is also consistent with the relatively few epidemiological studies of cancer responses to specific agents that contain enough information to make the evaluation possible (e.g., radiation-induced leukemia, breast and thyroid cancer, skin cancer induced by arsenic in drinking water, and liver cancer induced by aflatoxin in the diet). There is also some evidence from animal experiments that is consistent with the linear non-threshold hypothesis (e.g., liver tumors induced in mice by 2-acetylaminofluorene in the large scale ED_{01} study at the National Center of Toxicological Research, and the initiation stage of the two-stage carcinogenesis model in the rat liver and the mouse skin).

Because it has the best, albeit limited, scientific basis of any of the current mathematical extrapolation models, the linear non-threshold model has been adopted as the primary basis for risk extrapolation to low levels of the dose-response relationship. The risk assessments made with this model should be regarded as conservative, representing the most plausible upper limit for the risk; i.e., the true risk is not likely to be higher than the estimate, but it could be smaller.

The mathematical formulation chosen to describe the linear, non-threshold dose-response relationship at low doses is the improved multistage model developed by Crump (1980). This model employs enough arbitrary constants to be able to fit almost any monotonically increasing dose-response data and it incorporates a procedure for estimating the largest possible linear slope (in the 95 percent confidence limit sense) at low extrapolated doses that is consistent with the data at all dose levels of the experiment. For this reason, it may be called a "linearized" multistage model.

2. Procedure of Low-Dose Extrapolation Based on Animal Carcinogenicity Data.

A. Description of the Extrapolation Model

Let P(d) represent the lifetime risk (probability) of cancer at dose d. The multistage model has the form

$$P(d) = 1 - \exp[-(q_0 + q_1d + q_2d^2 + \ldots + q_kd^k)]$$

where:

$q_i \geq 0$, and $i = 0, 1, 2, \ldots, k$

Equivalently,

$$A(d) = 1 - \exp[-(q_1d + q_2d^2 + \ldots + q_kd^k)]$$

where:

$$A(d) = \frac{P(d) - P(o)}{1 - P(o)},$$

is the extra risk over background rate at dose d.

The point estimate of the coefficients q_i, $i = 0, 1, 2, \ldots, k$, and consequently the extra risk function A(d) at any given dose d, is calculated by maximizing the likelihood function of the data.

The point estimate and the 95 percent upper confidence limit of the extra risk A(d) are calculated by using the computer program GLOBAL 79 developed by Crump and Watson (1979). Upper 95 percent confidence limits on the extra risk and lower 95 percent confidence limits on the dose producing a given risk are determined from a 95 percent upper confidence limit, q_1^*, on parameter q_1. Whenever $q_1 \neq 0$, at low doses extra risk A(d) has approximately the form $A(d) = q_1 \times d$. Therefore, $q_1 \times d$ is a 95 percent upper confidence limit on the extra risk and R/q_1^* is a 95 percent lower confidence limit on the dose producing an extra risk of R. Let L_0 be the maximum value of the log-likelihood function. The upper limit q_1^* is calculated buy increasing q_1 to a value q_1^* such that when the log-likelihood is again maximized subject to this fixed value q_1^* for the linear coefficient, the resulting maximum value of the log-likelihood L_1 satisfies the equation $2(L_0 - L_1) = 2.70654$ where 2.70554 is the cumulative 90 percent point of the chi-square distribution with one degree of freedom, which corresponds to a 95 percent upper limit (one-sided). This approach of computing the upper confidence limit for the extra risk A(d) is an improvement on the Crump, et al. (1977) model. The upper confidence limit for the extra risk calculated at low doses is always linear. This is conceptually consistent with the linear nonthreshold concept discussed earlier. The slope q_1^* is taken as an upper bound of the potency of the chemical in inducing cancer at low doses.

In fitting the dose-response model, the number of terms in the polynomial g is chosen equal to (h−1), where h is the number of dose groups in the experiment, including the control group.

Whenever the multistage model does not fit the data sufficiently, data at the highest dose is deleted and the model is refitted to the rest of the data. This is continued until an acceptable fit to the data is obtained. To determine whether or not a fit is acceptable, the chi-square statistic:

$$\chi^2 = \sum_{i=1}^{h} \frac{(X_i - N_iP_i)^2}{N_iP_i(1 - P_i)}$$

is calculated, where N_i is the number of animals in the i^{th} dose group, X_i is the number of animals in the i^{th} dose group with a tumor response, P_i is the probability of a response in the i^{th} dose group estimated by fitting the multistage model to the data, and h is the number of remaining groups.

The fit is determined to be unacceptable whenever chi-square (X^2) is larger than the cumulative 99 percent point of the chi-square distribution with f degrees of freedom, where f equals the number of dose groups minus the number of non-zero multistage coefficients.

3. Selection and Form of Data used to Estimate Parameters in the Extrapolation Model.

For some chemicals, several studies in different animal species, strains, and sexes each conducted at several doses and different routes of exposure are available. A choice must be made as to which of the data sets from several studies are to be used in the model. It is also necessary to correct for metabolism differences between species and for differences in absorption via different routes of administration. The procedures, listed below, used in evaluating these data are consistent with the estimate of a maximum-likely-risk.

a. The tumor incidence data are separated according to organ sites or tumor types. The set data (i.e., dose and tumor incidence) used in the model is set where the incidence is statistically significantly higher than the control for at least one test dose level and/or where the tumor incidence rate shows a statistically significant trend with respect to dose level. The data set which gives the highest estimate of lifetime carcinogenic risk q_1^* is selected in most cases. However, efforts are made to exclude data sets which produce spuriously high risk estimates because of a small number of animals. That is, if two sets of data show a similar dose-response relationship and one has a very small sample size, the set of data which has the larger sample size is selected for calculating the carcinogenic potency.

b. If there are two or more data sets of comparable size which are identical with respect to species, strain, sex, and tumor sites, the geometric mean of q_1^*, estimated from each of these data sets is used for risk assessment. The geometric mean of numbers $A_1, A_2, \ldots, A_m$ is defined as $(A_1 \times A_2 \times \ldots \times A_m)^{1/m}$

c. If sufficient data exist for two or more significant tumor sites in the same study, the number of animals with at least one of the specific tumor sites under consideration is used as incidence data in the model.

d. Following the suggestion of Mantel and Schneiderman (1975), we assume that mg/surface area/day is an equivalent dose between species. Since to a close approximation the surface area is proportional to the $2/3$rds power of the weight as would be the case for a perfect sphere, the exposure in mg/$2/3$rds power of the body weight/day is similarly considered to be an equivalent exposure. In an animal experiment, this equivalent dose is computed in the following manner:

Let:

L_e = duration of experiment
l_e = duration of exposure
m = average dose per day in mg during administration of the agent (i.e., during l_e)
W = average weight of the experimental animal.

Then, the lifetime average exposure is

$$d = \frac{l_e \times m}{L_e \times W^{2/3}}$$

Often exposures are not given in units of mg/day, and it becomes necessary to convert the given exposures into mg/day. For example, in most feeding studies, exposure is expressed as ppm in the diet. In this case the exposure (mg/day) is derived by: m = ppm × F × r where ppm is parts per million of the carcinogenic agent in the diet, F is the weight of the food consumed per day in kgms, and r is the absorption fraction.

In the absence of any data to the contrary, r is assumed to be one. For a uniform diet the weight of the food consumed is proportional to the calories required, which, in turn, is proportional to the surface area or the ⅔rds power of the weight, so that: $m \propto ppm \times W^{2/3} \times r$ or

$$\frac{m}{rW^{2/3}} \propto ppm$$

As a result, ppm in the diet is often assumed to be an equivalent exposure between species. However, we feel that this is not justified since the calories/kg of food is significantly different in the diet of man vs. laboratory animals, primarily due to moisture content differences. Instead, we use an empirically derived food factor, $f = F/W$, which is the fraction of a species body weight that is consumed per day as food. We use the rates given below.

Species	W	f
Man	70	0.028
Rat	0.35	0.05
Mice	0.03	0.13

Thus, when the exposure is given as a certain dietary concentration in ppm, the exposure in mg/$W^{2/3}$ is

$$\frac{m}{r \times W^{2/3}} = \frac{ppm \times F}{W^{2/3}} =$$

$$\frac{ppm \times f \times W}{W^{2/3}} = ppm \times f \times W^{1/3}$$

When exposure is given in terms of $mg/kg/day = m/Wr = s$ the conversion is simply:

$$\frac{m}{rW^{2/3}} = s \times W^{1/3}$$

When exposure is via inhalation, the calculation of dose can be considered for two cases where (1) the carcinogenic agent is either a completely water-soluble gas or an aerosol and is absorbed proportionally to the amount of air breathed in, and (2) where the carcinogen is a poorly water-soluble gas which reaches an equilibrium between the air breathed and the body compartments. After equilibrium is reached, the rate of absorption of these agents is expected to be proportional to metabolic rate, which in turn is proportional to the rate of oxygen consumption, which in turn is a function of surface area.

Case 1

Agents that are in the form of particulate matter or virtually completely absorbed gases such as SO_2 can reasonably be expected to be absorbed proportional to the breathing rate. In this case the exposure in mg/day may be expressed as: $m = I \times v \times r$ where I is inhalation rate per day in m^3, v is mg/m^3 of the agent in air, and r is the absorption fraction.

The inhalation rates, I, for various species can be calculated from the observation (FASEB, 1974) that 25 gm mice breathe 34.5 liters/day and 113 gm rats breathe 105 liters/day. For mice and rats of other weights, W, (expressed in kg), the surface area proportionality can be used to determine breathing rates (in m^3/day) as follows:

For mice, $I = 0.0345 (W/0.025)^{2/3} m^3/day$

For rats, $I = 0.105 (W/0.113)^{2/3} m^3/day$

For humans, the values of 20 m^3/day[*] is adopted as a standard breathing rate (ICRP, 1977).

The equivalent exposure in mg/$W^{2/3}$ for these agents can be derived from the air intake data in a way analogous to the food intake data. The empirical factors for the air intake per kg per day, $i = I/W$ based upon the previously stated relationships, are as tabulated below:

Species	W	i=I/W
Man	70	0.29
Rat	0.35	0.64
Mice	0.03	1.3

Therefore, for particulates or completely absorbed gases, the equivalent exposure in mg/$W^{2/3}$ is:

$$\frac{m}{W^{2/3}} = \frac{Ivr}{W^{2/3}} = \frac{iWvr}{W^{2/3}} = iW^{1/3}\,vr$$

In the absence of empirical data or a sound theoretical argument to the contrary, the fraction absorbed, r, is assumed to be the same for all species.

Case 2

The dose in mg/day of partially soluble vapors is proportional to the O_2 consumption which in turn is proportional to $W^{2/3}$ and to the solubility of gas in body fluids, which can be expressed as an absorption coefficient r for the gas. Therefore, when expressing the O_2 consumption as $O_2 = k W^{2/3}$, where k is a constant independent of species, it follows that $m = k W^{2/3} \times v \times r$ or

$$d = \frac{m}{W^{2/3}} = kvr$$

As with Case 1, in the absence of experimental information or a sound theoretical argument to the contrary, the absorption fraction, r, is assumed to be the same for all species. Therefore, for these substances a certain concentration in ppm or μ/m^3 in experimental animals is equivalent to the same concentration in humans. This is supported by the observation that the minimum alveolar concentration, necessary to produce a given "stage" of anesthesia, is similar in man and animals (Dripps, et al. 1977). When the animals were exposed via the oral route and human exposure is via inhalation or vice-versa, the assumption is made, unless there is pharmacokinetic evidence to the contrary, that absorption is equal by either exposure route.

e. If the duration of experiment (L_e) is less than the natural life span of the test animal (L), the slope q_1^*, or more generally the exponent g(d), is increased by multiplying a factor $(L/L_e)^3$. We assume that if the average dose, d, is continued, the age specific rate of cancer will continue to increase as a constant function of the background rate. The age specific rates for humans increase at least by the 2nd power of the age and often by a considerably higher power, as demonstrated by Doll (1971). Thus, we would expect the cumulative tumor rate to increase by at least the 3rd power of age. Using this fact, we assume that the slope q_1^*, or more generally, the exponent g(d), would also increase by at least the 3rd power of age. As a result, if the slope q_1^* [or g(d)] is calculated at age L_e, we would expect that if the experiment had been continued for the full life span, L, at the given average exposure, the slope q_1^* [or g(d)] would have been increased by at least $(L/L_e)^3$.

This adjustment is conceptually consistent to the proportional hazard model proposed by Cox (1972) and the time-to-tumor model considered by Crump, et al. (1977) where the probability of cancer at age t and dose d is given by $P(d,t) = 1 - \exp[-f(t) \times g(d)]$

4. Calculation of Carcinogenic Potency Based on Human Data. If human epidemiology studies and sufficiently valid exposure information are available for the compound, they are always used in some way. If they show a carcinogenic effect, the data are analyzed to give an estimate of the linear dependence of cancer rates on lifetime average dose, which is equivalent to the factor q_1^*. If they show

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[*] From "Recommendation of the International Commission on Radiological Protection," page 9, the average breathing rate is 10^7 cm^3 per 8-hour work day and 2×10^7 cm^3 in 24 hours.

no carcinogenic effect when positive animal evidence is available, then it is assumed that a risk does exist but it is smaller than could have been observed in the epidemiologic study, and an upper limit of the cancer incidence is calculated assuming hypothetically that the true incidence is just below the level of detection in the cohort studied, which is determined largely by the cohort size. Whenever possible, human data are used in perference to animal bioassay data.

In human studies, the response is measured in terms of the relative risk of the exposed cohort of individuals compared to the control group. In the analysis of this data, it is assumed that the excess risk, or relative risk minus one, $R(X)-1$, is proportional to the lifetime average exposure, X, and that it is the same for all ages. It follows that the carcinogenic potency is equal to $[R(X)-1]/X$ multiplied by the lifetime risk at that site in the general population. Except for an unusually well-documented human study, the confidence limit for the excess risk is not calculated, due to the difficulty in accounting for the uncertainty inherent in the data (exposure and cancer response).

5. Calculation of Water Quality Criteria. After the value of q_1^* in $(mg/kg/day)^{-1}$ has been determined, the lifetime risk, P, from an average daily exposure of x mg/kg/day is found from the equation $P = q_1^*x$. Therefore, if the lifetime risk is set at $P = 10^{-5}$ for calculation purposes, the intake, I, in mg/day for a 70 kg person can be found by the equation: $I = 70 \times 10^{-5}/q_1^*$
The intake of the agent from ambient water is assumed to come from two sources: (1) drinking an average of 2 liters of water per day, and (2) ingesting an average of 6.5 grams of fish per day. Because of accumulation of residues in fish, the amount of the pollutant in fish (mg/kg of edible fish) is equal to a factor R times the water concentration (mg/kg of water). Therefore, the total intake I can be written as sum of two terms: $I(mg/day) = C(mg/l) \times R(l/kg\ fish) \times 0.0065\ kg\ fish/day + C(mg/l \times 2l/day = C(2 + 0.0065R)$ where C is the water concentration in mg/l. Therefore, the water concentration in mg/l corresponding to a lifetime risk of 10^{-5} for a 70 kg person is calculated by the formula:

$$C = \frac{70 \times 10^{-5}}{q_1^*(2 + 0.0065\ R)}$$

B. Threshold Effects

1. Use of Animal Toxicity Data (Oral). In developing guidelines for deriving criteria based on noncarcinogenic responses, five types of response levels are considered:

NOEL—No-Observed-Effect-Level
NOAEL—No-Observed-Adverse-Effect-Level
LOEL—Lowest-Observed-Effect-Level
LOAEL—Lowest-Observed-Adverse-Effect-Level
FEL—Frank-Effect-Level

Adverse effects are defined as any effects which result in functional impairment and/or pathological lesions which may affect the performance of the whole organism, or which reduce an organism's ability to respond to an additional challenge.

One of the major problems encountered in consideration of these concepts regards the reporting of "observed effect levels" as contrasted to "observed adverse effect levels". The terms "adverse" vs. "not adverse" are at times satisfactorily defined, but due to increasingly sophisticated testing protocols, more subtle responses are being identified, resulting in a need for judgment regarding the exact definition of adversity.

The concepts listed above (NOEL, NOAEL, LOEL, LOAEL) have received much attention because they represent landmarks which help to define the threshold region in specific experiments. Thus, if a single experiment yields a NOEL, a NOAEL, a LOAEL, and a clearly defined FEL in relatively closely spaced doses, the threshold region has been relatively well defined; such data are very useful for the purpose of deriving a criterion. On the other hand, a clearly defined FEL has little utility in establishing criteria when it stands alone, because such a level gives no indication how far removed the data point is from the threshold region. Similarly, a free-standing NOEL has little utility, because there is no indication of its proximity to the LOEL, since a free-standing NOEL may be many orders of magnitude below the threshold region.

Based on the above dose-response classification system, the following guidelines for deriving criteria have been adopted:

a. A free-standing FEL is unsuitable for the derivation of criteria.

b. A free-standing NOEL is unsuitable for the derivation of criteria. If multiple NOELs are available without additional data on LOELs, NOAELs, or LOAELs, the highest NOEL should be used to derive a criterion.

c. A NOAEL, LOEL, or LOAEL can be suitable for criteria derivation. A well-defined NOAEL from a chronic (at least 90-day) study may be used directly, applying the appropriate uncertainty factor. For a LOEL, a judgment needs to be made whether it actually corresponds to a NOAEL or a LOAEL. In the case of a LOAEL, an additional uncertainty factor is applied; the magnitude of the additional uncertainty factor is judgmental and should lie in the range of 1 to 10. Caution must be exercised not to substitute "Frank-Effect-Levels" for "Lowest-Observable-Adverse-Effect-Levels".

d. If for reasonably closely spaced doses only a NOEL and a LOAEL of equal quality are available, then the appropriate uncertainty factor is applied to the NOEL.

In using this approach, the selection and justification of uncertainty factors are critical. The basic definition and guidelines for using uncertainty factors has been given by the National Academy of Sciences (1977). "Safety Factor" or "Uncertainty Factor" is defined as a number that reflects the degree or amount of uncertainty that must be considered when experimental data in animals are extrapolated to man. When the quality and quantity of experimental data are satisfactory, a low uncertainty factor is used; when data is judged to be inadequate or equivocal, a larger uncertainty factor is used. The following general guidelines have been adopted in establishing the uncertainty factors:

a. Valid experimental results from studies on prolonged ingestion by man, with no indication of carcinogenicity. Uncertainty Factor = 10

b. Experimental results of studies of human ingestion not available or scanty (e.g., acute exposure only) with valid results of long-term feeding studies on experimental animals, or in the absence of human studies, valid animal studies on one or more species. No indication of carcinogenicity. Uncertainty Factor = 100

c. No long-term or acute human data. Scanty results on experimental animals with no indication of carcinogenicity. Uncertainty Factor = 1,000

Considerable judgment must be used in selecting the appropriate safety factors for deriving a criterion. In those cases where the data do not completely fulfill the conditions for one category and appear to be intermediate between two categories an intermediate uncertainty factor is used. Such an intermediate uncertainty factor may be developed based on a logarithmic scale (e.g., 33, being halfway between 10 and 100 on a logarithmic scale).

In determining the appropriate use of the uncertainty factors, the phrase "no indication of carcinogenicity" is interpreted as the absence of carcinogenicity data from animal experimental studies or human epidemiology. Available short-term carcinogenicity screening tests are reported in the criteria documents, but they are not used either for derivation of numerical criteria nor to rule out the uncertainty factor approach.

Because of the high degree of judgment involved in the selection of a safety factor, the criterion derivation section of each document should provide a detailed discussion and justification for both the selection of the safety factor and the data to which it is applied. This discussion should reflect a critical review of the available data base. Factors to be considered include number of animals, species, and parameters tested; quality of controls; dose levels; route; and dosing schedules. An effort should be made to differentiate between results which constitute a toxicologically sufficient data base and data which may be spurious in nature.

2. Use of Acceptable Daily Intake (ADI). For carcinogens, the assumption of low dose linearity precludes the necessity for defining total exposure in the estimation of increased incremental risk. For non-carcinogens, ADIs and criteria derived therefrom are calculated from total exposure data that include contributions from the diet and air. The equation used to derive the criterion (C) is: $C = ADI - (DT + IN)/[2\ l + (0.0065\ kg \times R)]$ where 2 l is assumed daily water consumption, 0.0065 kg is assumed daily fish consumption, R is bioconcentration factor in units of l/kg, DT is estimated non-fish dietary intake, and IN is estimated daily intake by inhalation.

If estimates of IN and DT cannot be provided from experimental data, an assumption must be made concerning total exposure. It is recognized that either the inability to estimate DT and IN due to lack of data or the wide variability in DT and IN in different states may add an additional element of uncertainty to the criterion formulation process. In terms of scientific validity, the accurate estimate of the Acceptable Daily Intake is the major factor in satisfactory derivation of water quality criteria.

3. Use of Threshold Limit Values or Animal Inhalation Studies. Threshold Limit Values (TLVs) are established by the American Conference of Governmental and Industrial Hygienists (ACGIH) and represent 8-hour time-weighted average concentrations in air that are intended to protect workers from various adverse health effects over a normal working lifetime. Similar values are set by NIOSH (criteria) and OSHA (standards) for 10- and 8-hour exposures, respectively. To the extent that these values are based on sound toxicologic assessments and have been protective in the work environment, they provide useful information for deriving or evaluating water quality criteria. However, each TLV must be carefully examined to determine if the basis of the TLV contains data which can be used directly to derive a water quality criterion using the uncertainty factor approach. In addition, the history of each TLV must be examined to assess the extent to which it has assured worker safety. In each case, the types of effects against which TLVs are designed to protect are examined in terms of their relevance to exposure from water. It must be demonstrated that the chemical is not a localized irritant and that there is no significant effect at the site of entry irrespective of the routes of exposure (i.e., oral or inhalation).

If the TLV or similar value is recommended as the basis of the criterion, consideration of the above points is explicitly stated in the criterion derivation section of the document. Particular emphasis is placed on the quality of the TLV relative to the available toxicity data that normally is given priority over TLVs or similar established values. If the TLV can be justified as the basis for the cirterion, then the problems associated with the estimation of acceptable oral doses from inhalation data must be addressed.

Estimating equivalencies of dose-response relationships from one route of exposure to another introduces an additional element of uncertainty in the derivation of criteria. Consequently, whenever possible, ambient water quality criteria should be based on data involving oral exposures. if oral data are insufficient, data from other routes of exposure may be useful in the criterion derivation process.

Inhalation data, including TLVs or similar values, are the most common alternatives to oral data. Estimates of equivalent doses can be based upon: (1) available pharmacokinetic data for oral and inhalation routes, (2) measurements of absorption efficiency from ingested or inhaled chemicals, or (3) comparative excretion data when the associated metabolic pathways are equivalent to those following oral ingestion or inhalation. Given that sufficient pharmacokinetic data are available, the use of accepted pharmacokinetic models provides the most satisfactory approach for dose conversions. However, if available pharmacokinetic data are marginal or of questionable quality, pharmacokinetic modeling is inappropriate.

The Stokinger and Woodward (1958) approach, or similar models based on assumptions of breathing rate and absorption efficiency, represents possible alternatives when data are not sufficient to justify pharmacokinetic modeling. Such alternative approaches, however, provide less satisfactory approximations because they are not based on pharmacokinetic data. Consequently, in using the Stokinger and Woodward or related models, the uncertainties inherent in each of the assumptions and the basis of each assumption must be clearly stated in the derivation of the criterion.

The use of data pertaining to other routes of exposure to derive water quality criteria may also be considered. As with inhalation data, an attempt is made to use accepted toxicologic and pharmacokinetic principles to estimate equivalent oral doses. If simplifying assumptions are used, their bases and limitations must be clearly specified.

Because of the uncertainties involved in extrapolating from one route of exposure to another and the consequent limitations that this may place on the derived criterion, the decision to disallow such extrapolation and recommend no criterion is highly judgmental and must be made on a case-by-case basis. A decision for or against criteria derivation must balance the quantity and quality of the available data against a perceived risk to the human population.

If the Stokinger and Woodward (1958) approach is used to calculate an ADI from a TLV, the general equation is: $ADI = TLV \times BR \times DE \times d \times A_A/(A_O \times SF)$ where:

ADI = Acceptable daily intake in mg
TLV = Concentration in air in mg/m^3
DE = Duration of exposure in hours per day
$d = 5$ days/7 days
A_A = Efficiency of absorption from air
A_O = Efficiency of absorption from oral exposure
SF = Safety factor following guidelines given above
BR = Amount of air breathed per day; assume 10 m^3

For deriving an ADI from animal toxicity data, the equation is: $ADI = C_A \times D_E \times d \times A_A \times BR \times 70\ kg/(BW_A \times A_O \times SF)$ where:

ADI = Acceptable daily intake in mg
C_A = Concentration in air in mg/m^3
D_E = Duration of exposure in hours per day
d = Number of days exposed/number of days observed
A_A = Efficiency of absorption from air
BR = Volume of air breathed per day in m^3
70 kg = Assumed human body weight
BW_A = Body weight of experimental animals in kg

A_o = Efficiency of absorption from oral exposure
SF = Safety factor following guidelines given above

More formal pharmacokinetic models must be developed on a compound-by-compound basis.

It should be noted that the safety factors used in the above formulae are intended to account for species variability. Consequently, the mg/surface area/day conversion factor is not used in the derivation of toxicity based criterion.

C. Organoleptic Criteria

Organoleptic criteria define concentrations of materials which impart undesirable taste and/or odor to water. In developing and utilizing such criteria two factors must be appreciated: the limitations of most organoleptic data and the human health significance of organoleptic properties.

The publications which report taste and odor thresholds are, with very few exceptions, cryptic in their descriptions of test methodologies, number of subjects tested, concentration: response relationships, and sensory characteristics at specific concentrations above threshold. Thus, the quality of organoleptic data is often significantly less than that of toxicologic data used in establishing other criteria. Consequently, a critical evaluation of the available organoleptic data must be made and the selection of the most appropriate data base for the criterion must be based on sound scientific judgment.

Organoleptic criteria are not based on toxicologic information and have no direct relationship to potential adverse human health effects. Although sufficiently intense organoleptic characteristics could result in depressed fluid intake which, in turn, might aggravate a variety of functional disease states (i.e., kidney and circulatory diseases), such effects are not used in the derivation process of organoleptic criteria unless available data would indicate an indirect human health effect via decreased fluid consumption, criteria derived solely from organoleptic data are based upon aesthetic qualities only.

Since organoleptic and human health effects criteria are based on different endpoints, a distinction must be made between these two sets of information. In criteria summaries involving both types of data, the following format is used:

> For comparison purposes, two approaches were used to derive criterion levels for ———. Based on available toxicity data, for the protection of public health the derived level is ——. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water the estimated level is ——. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have no demonstrated relationship to potential adverse human health effects.

In those instances where a level to limit toxicity cannot be derived, the following statement is to be appropriately inserted.

> Sufficient data are not available for ——— to derive a level which would protect against the potential toxicity of this compound.

D. Criteria for Chemical Classes

A chemical class is broadly defined as any group of chemical compounds which are reviewed in a single risk assessment document. In criterion derivation, isomers should be regarded as a part of a chemical class rather than as a single compound. A class criterion is an estimate of risk/safety which applies to more than one member of a class. It involves the use of available data on one or more chemicals of a class to derive criteria for other compounds of the same class in the event that there are insufficient data available to derive compound-specific criteria.

A class criterion usually applies to each member of a class rather than to the sum of the compounds within the class. While the potential hazards of multiple toxicant exposure are not to be minimized, a criterion, by definition, most often applies to an individual compound. Exceptions may be made for complex mixtures which are produced, released, and toxicologically tested as mixtures (e.g., toxaphene and PCBs). For such exceptions, some attempt is made to assess the effects of environmental partitioning (i.e., different patterns of environmental transport and degradation) on the validity of the criterion. If these effects cannot be assessed, an appropriate statement of uncertainty should accompany the criterion.

Since relatively minor structural changes within a class of compounds can have pronounced effects on their biological activities, reliance on class criteria should be minimized. Whenever sufficient toxicologic data are available on a chemical within a class, a compound-specific criterion should be derived. Nonetheless, for some chemical classes, scientific judgment may suggest a sufficient degree of similarity among chemicals within a class to justify a class criterion applicable to some of all members of a class.

The development of a class criterion takes into consideration the following:

1. A detailed review of the chemical and physical properties of chemicals within the group should be made. A close relationship within the class with respect to chemical acitivity would suggest a similar potential to reach common biological sites within tissues. Likewise, similar lipid solubilities would suggest the possibility of comparable absorption and tissue distribution.

2. Qualitative and quantitative data for chemicals within the group are examined. Adequate toxicologic data on a number of compounds within a group provides a more reasonable basis for extrapolation to other chemicals of the same class than minimal data on one chemical or a few chemicals within the group.

3. Similarities in the nature of the toxicologic response to chemicals in the class provides additional support for the prediction that the response to other members of the class may be similar. In contrast, where the biological response has been shown to differ markedly on a qualitative and quantitative basis for chemicals within a class, the extrapolation of a criterion to other members of that class is not appropriate.

4. Additional support for the validity of extrapolation of a criterion to other members of a class could be provided by evidence of similar metabolic and pharmacokinetic data for some members of the class.

Based on the above considerations, it may be reasonable in some cases to divide a chemical class into various subclasses. Such divisions could be based on biological endpoints (e.g., carcinogens/non-carcinogens), potency, and/or sufficiency of data (e.g., a criterion for some members of a class but no criterion for others). While no *a priori* limits can be placed on the extent of subclassification, each subclassification must be explicitly justified by the available data.

Class criteria, if properly derived and supported, can constitute valid scientific assessments of potential risk/safety. Conversely, the development of a class criterion from an insufficient data base can lead to serious errors in underestimating or overestimating risk/safety and should be rigorously avoided. Although scientific judgment has a proper role in the development of class criteria, such criteria are useful and defensible only if they are based on adequate data and scientific reasoning. The definition of sufficient data on similarities in physical, chemical, pharmacokinetic, or toxicologic properties to justify a class criterion may vary markedly depending on the degree of structural similarity and the gravity of the perceived risk. Consequently, it is imperative that the criterion derivation section of each document in which a class criterion is recommended explicity address each of the key issues discussed above, and define, as clearly as possible, the limitations of the proposed criterion as well as the type of data needed to generate a compound-specific criterion.

A class criterion should be abandoned when there is sufficient data availabe to derive a compound-specific criterion which protects against the biological effect of primary concern; e.g., the availability of a good subchronic study would not necessarily result in the abandonment of a class criterion based on potential carcinogenicity.

The inability to derive a valid class criterion does not, and should not, preclude regulation of a compound or group of compounds based on concern for potential human health effects. The failure to recommend a criterion is simply a statement that the degree of concern cannot be quantified based on the available data and risk assessment methodology.

E. Essential Elements

Some chemicals, particularly certain metals, are essential to biological organisms at low levels but may be toxic and/ or carcinogenic at high levels. Because of potential toxic effects, it is legitimate to establish criteria for such essential elements. However, criteria must consider essentiality and cannot be established at levels which would result in deficiency of the element in the human population.

Elements are accepted as essential if listed by NAS Food and Nutrition Board or a comparably qualified panel. Elements not yet determined to be essential but for which supportive data on essentiality exists need to be further reviewed by such a panel.

To modify the toxicity and carcinogenicity based criteria, essentiality must be quantified either as a "recommended daily allowance" (RDA) or "minimum daily requirement" (MDR). These levels are then compared to estimated daily doses associated with the adverse effect of primary concern. The difference between the RDA or MDR and the daily doses causing a specified risk level for carcinogens or ADIs for non-carcinogens defines the spread of daily doses from which the criterion may be derived. Because errors are inherent in defining both essential and maximum tolerable levels, the criterion is derived from dose levels near the center of such a dose range. The decision to use either the MDR or RDA is guided by the spread of the doses and the quality of the essentiality and toxicity estimates.

The modification of criteria by consideration of essentiality must take into account all routes of exposure. If water is a significant source of the MDR or RDA, the criterion must allow for attainment of essential intake. Conversely, even when essentiality may be attained from nonwater sources, standard criteria derivation methods may be adjusted if the derived criterion represents a small fraction of the ADI or MDR. On a case-by-case basis, the modification in the use of the guidelines may include the use of different safety factors for non-carcinogens or other modifications which can be explicitly justified.

F. Use of Existing Standards

For some chemicals for which criteria are to be established, drinking water standards already exist. These standards represent not only a critical assessment of literature, but also a body of human experience since their promulgation. Therefore, it is valid to accept the existing standard unless there is compelling evidence to the contrary. This decision should be made after considering the existing standards vs. new scientific evidence which has accumulated since the standards have been established. There are several instances where the peer review process recommended usage of the present drinking water standards.

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Appendix D—Response to Comments on Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses

Introduction

Two versions of the Guidelines were published in the **Federal Register** for comment. The first version (43 FR 21506, May 18, 1978 and 43 FR 29028, July 5, 1979) was simply published for comment. The second (44 FR 15926, March 15, 1979) was published as part of the request for comments on the water quality criteria for 27 of the 65 pollutants. The second version was meant to be clearer and more detailed than the first, but very similar technically. Since the two versions were so similar, comments on both will be dealt with simultaneously.

Many comments were received that no draft water quality criteria for any of the 65 pollutants should have been issued for public comment until the comments on the first version of the Guidelines had been dealt with adequately and the Guidelines changed appropriately. The comments on the first version were read and the Guidelines were revised in an attempt to make the second version clearer and more detailed than the first. However, an extensive revision of the technical content of the Guidelines was not attempted between the first and second versions because the Agency was preparing water quality criteria based on the Guidelines. The Agency could have avoided this criticism simply by not publishing any version of the Guidelines for comment until March 15, 1979, but this would have greatly reduced the length of time available for people to consider the Guidelines and comment on them. As it was, some people commented that the comment period announced on March 15, 1979, was too short.

1. Comment—The procedures used to derive criteria in the "Red Book" were upheld in court and probably should still be used.

Response—The procedures used in the Guidelines are similar to some of the procedures used to develop criteria in the "Green Book", "Blue Book", and "Red Book". The Guidelines are designed to be more objective and systematic, to deal more adequately with residues, and to incorporate the concept of a minimum data base.

2. Comment—Criteria should be compilations of critically reviewed data with no synthesis or interpretation.

Response—Neither P.L. 92-500 nor the Consent Decree specify the form which a criterion must take. The Consent Decree (para. 11, p. 14) specifies that such criteria "shall state, *inter alia*, recommended maximum permissible concentrations". Adequate precedents have been set in the "Green Book", "Blue Book", and "Red Book" for the form of criteria used in the Guidelines.

3. Comment—The Guidelines and criteria should be developed by a consensus of aquatic toxicologists rather than by EPA personnel only.

Response—EPA certainly wants the Guidelines and the criteria to be as good as possible and as acceptable to as many interested people as possible. To this end, EPA has widely distributed draft versions of the Guidelines and the criteria documents, discussed them with many people, considered the comments received, and made many significant technical changes and editorial revisions. It is questionable whether or not a true consensus could have been reached by any means within the time available. In addition, EPA has a legislative responsibility which it should not delegate to someone else.

4. Comment—The Guidelines should be updated regularly.

Response—The Guidelines are not being promulgated as a regulation or directive. The purpose of presenting these Guidelines is to show how the water quality criteria for aquatic life were derived for the 65 pollutants. If EPA uses these Guidelines again, they will be revised to take into account new data, concepts, and ideas.

5. Comment—The objectives, purpose, and limitations of the Guidelines should be stated.

Response—The introductory portion of the Guidelines has been expanded to address these subjects more fully.

6. Comment—The Guidelines are too ambiguous.

Response—The Guidelines have been revised and rewritten, partly to improve clarity and provide additional details. It is not possible to provide explicit details on all items; in some areas only general guidance can be provided at this time.

EPA attempted to clearly and concisely deal with all issues which might significantly affect the resulting criteria without going into extreme detail on every potential problem. Because numerous judgments must be made, a reasonable amount of experience in aquatic toxicology will be necessary for a person to utilize the Guidelines effectively.

7. Comment—The Guidelines are too complex.

Response—Deriving a water quality criterion is a complex exercise because several different kinds of data and a wide variety of organisms need to be considered. In addition, because data have been generated using various procedures, numerous individual decisions need to be made and the Guidelines attempt to provide guidance concerning decisions that seem to need to be made frequently. The Guidelines are more complex than initially envisioned to help insure that criteria for different pollutants are derived in a reasonably comparable manner. Although the process of deriving a water quality criterion for aquatic life is complex, the Guidelines help organize the process into logical components and steps.

8. Comment—The Guidelines should be more flexible.

Response—The Guidelines are meant to provide guidance and at the same time allow reasonable flexibility. They have been used with quite a variety of pollutants for which the requirements of the minimum data base are satisfied, and they seem to be reasonably appropriate in all cases because the experiences with these substances were a major part of the basis for the Guidelines. If sound scientific evidence indicates that a particular aspect of the Guidelines is not appropriate for a specific substance, then some other more appropriate procedure should be used. However, the Guidelines should not be changed based on individual whim or personal preference.

9. Comment—The Guidelines should take into account synergism and antagonism by a wide variety of factors and the effect of the pollutant on important ecological relationships.

Response—Very little practically useful information is available on these factors in connection with the effects of pollutants on aquatic organisms. Synergism and antagonism are possible between numerous combination of two or more pollutants, and some data indicate that such interactions are not only species specific, but also vary with the ratios and absolute concentrations of the pollutants and the life stage of the species. Pollutants may affect the structure and function of aquatic ecosystems separate from their effects on individual species, but practical applications of such ideas seem very tenuous at this time. Little information is available concerning such effects, and the significance of the available data is questionable. An obviously important ecological relationship is the dependence of higher organisms on lower organisms for food. Even here, the existence of numerous lower species and their adaptability reduces the importance of any individual food species.

10. Comment—The Guidelines should take into account all identifiable effects—beneficial as well as harmful.

Response—Few tests have been conducted to identify beneficial effects of individual pollutants on aquatic organisms. However, beneficial effects are sometimes observed in chronic toxicity tests at concentrations below those that cause adverse effects. Usually in such cases the organisms in low concentrations of the pollutant are longer or heavier or reproduce more that do the controls. Even if such effects are statistically significant, they are not judged as adverse or harmful. On the other hand, a beneficial effect on one species may ultimately be to the detriment of a community if a balance between species is disturbed. Also, a concentration that benefits one species may harm a more sensitive species.

11. Comment—The Guidelines should take into account analytical methodology.

Response—The Guidelines do take into account analytical methodology in the definition of the substance, when necessary, but not in deriving the numerical value of the criterion. Concentrations which cannot be routinely measured accurately can often be measured accurately by nonroutine methods and, more importantly, do sometimes adversely affect aquatic organisms. When aquatic organisms are more sensitive than routine analytical methods, the proper solution is to develop better analytical methods, not to underprotect aquatic life. One use of criteria should be to identify needs in analytical chemistry.

12. Comment—The Guidelines should take into account (a) production and usage patterns, (b) chemical, physical and biological factors pertaining to degradation and fate of pollutants, including properties such as solubility in water, decay rate, persistence, and transformation pathways, and (c) whether or not a criterion is needed for the substance.

Response—Items included in (a) and (b) may be important in deciding whether a criterion is needed for a substance, but the Guidelines are intended to be used after the decision has been made that a criterion is needed. EPA is presently developing principles that can be used to decide whether or not a criterion is needed for a substance and items such as those listed above are probably some of the factors that should be considered when deciding whether or not a criterion is needed. If the toxicity of the chemical is used to evaluate the need for a criterion, the Guidelines may be useful in the collection and interpretation of the available toxicity data.

13. Comment—The Guidelines should take into account costs to states and industries, technological feasibility, and such characteristics of bodies of water as assimilative capacity, dispersal, dissipative factors, dilution, hydrology, mixing zones, and sediment.

Response—Factors such as these should be considered in developing standards, but not in deriving criteria. EPA is presently developing an implementation policy which will describe which of the above factors and which characteristics of the pollutant should be used, and how they should be used, in developing standards.

14. Comment—The Guidelines are not appropriate for establishing a concentration which may be present in an effluent.

Response—The Guidelines are for deriving water quality criteria, not effluent standards nor mixing zone standards nor water quality standards. Water quality criteria will probably be one factor taken into account in the development of water quality standards and toxicity-based effluent standards, but not technology-based effluent standards. EPA is presently developing policies concerning proper use of water quality criteria in various regulatory activities.

15. Comment—The derivation of criteria should be fundamentally a scientific exercise and should not employ subjective judgments.

Response—No exercise which involves the use and interpretation of data can avoid subjective judgment. Indeed, even the generation of scientific data requires subjective judgment, such as how many test organisms to use, what temperature to use, etc. One may decide to accept the recommendations of experts, but this is usually still a subjective decision. In statistics the subjective decisions are made on the basis of probability statements but the final decisions are still subjective judgments. Although the development of the Guidelines and the derivation of criteria cannot avoid subjective decisions, gross extrapolations, wild assumptions, and novel judgments can be avoided. One can also avoid using large safety factors to "make up" for insufficient data. When some agreement exists between experts, such as on test temperature and duration of tests, the collective opinion can usually be used. EPA feels that the Guidelines do not go too far beyond the state-of-the-art and do not produce criteria by extrapolating far beyond the usefulness of the data.

16. Comment—The Guidelines should not use unproven extrapolations.

Response—EPA feels that the extrapolations used in the Guidelines are reasonable for most pollutants. Probably the most questionable extrapolation is the acute-chronic ratio, but even here an arbitrary ratio is not used. Indeed, the ratio used is usually a mean of experimentally determined acute-chronic ratios for at least three, not just one, species. In addition, the species must include at least one fish and one invertebrate. Even this amount of data does not "prove" the validity of the extrapolation, but it should provide reasonable evidence for or against the use of the ratio with any particular substance. To achieve reasonable criteria without using any extrapolations would require acute and chronic tests with many more species. This would be a high price to pay for disallowing any use of scientific inference in deriving criteria.

The early versions of the Guidelines used adjustment factors and sensitivity factors which were averages derived from data for a wide variety of substances and thus were attempts to make some extrapolations across all substances. The present version of the Guidelines is based on a minimum data base for each individual pollutant and the calculations are essentially pollutant-specific. Thus no extrapolations are made from one pollutant to another.

17. Comment—Laboratory tests overestimate the toxicity of materials because the test organisms are stressed by the artifical conditions.

Response—Laboratory conditions certainly are artificial, but they do not necessarily stress the test organisms. Organisms which survive, grow, and reproduce well in the laboratory cannot be stressed too much. Organisms in a laboratory might be considered pampered because they do not have to compete for food and are not subject to stress due to predators and changing and extreme conditions of turbidity, temperature, flow, and water quality. Also, laboratory organisms are rarely subject to stress from pollutants. Some species probably have longer average life spans in laboratories than they do in field situations.

18. Comment—Laboratory tests underestimate the toxicity of materials because the tests are usually conducted with species which are hardy, adaptable, and insensitive.

Response—Species which are readily adaptable to laboratory conditions are not necessarily insensitive as evidenced by the great range of sensitivities obtained in laboratory tests for some individual pollutants with different species. In fact, once the the proper techniques are developed, a wide variety of species can survive, grow, and reproduce well in laboratories. When the proper techniques are discovered and a species changes form "difficult" to "easy", its sensitivity does not change. Also, some species and life stages which are fragile and must be handled with great care are not particularly sensitive. On the other hand, because so few species have actually been tested in laboratories, species which are more sensitive than any of those tested in laboratories, species which are more sensitive than any of those tested probably exist for most substances.

19. Comment—Laboratory tests are artificial and contrived and do not represent the real world.

Response—Laboratory tests are indeed artificial but they are not contrived to give results that are unnecessarily high or low. Organisms in a laboratory are generally acclimated to water and conditions of constant and desirable quality, whereas in the field they are often subjected to fluctuations and extremes. Organisms in a laboratory do not have to compete for food and are not subject to predators or pollution. Organisms in the field are often exposed to more than one pollutant at a time, with the combinations and concentrations changing often.

It is true that aquatic organisms are usually exposed to instantaneous high concentrations in laboratory tests, but in field situations organisms are often not given much chance to acclimate to spills or short-term discharges. Also, some ameliorating effects occur in field, but not laboratory, situations, but such effects are not always dependable over long periods of time. The concentrations of mitigating anions, suspended solids, and complexing agents are relatively constant in some bodies of water, but not in others. Suspended solids probably do sorb and detoxify significant amounts of some pollutants, but high concentrations of suspended solids also stress some aquatic organisms. In addition, organisms are usually fed in chronic tests, so the test solution contains suspended solids and dissolved organic carbon from the food and fecal matter. Degradation and other transformations are more likely in field situations than in laboratory situations, but degradation products are not always less toxic than the undegraded material. On the other hand, many of these kinds of considerations will probably be taken into account when site-specific criteria and standards are developed under the implementation policy which is being developed by EPA.

20. Comment—Laboratory tests are poor predictors of what will happen in field situations.

Response—If conditions are comparable, laboratory toxicity tests are useful predictors of what will happen in field situations. The usefulness of such predictions will depend on how carefully one accounts for differences between species, water quality, and the form of the pollutant. Extrapolations are much more difficult for some pollutants than for others. Water quality affects the toxicity of some pollutants much more than others, and species differences, even within families, are much greater for some pollutants than for others. If such factors are taken into account, useful predictions are possible. In what is probably the most extensive comparison available of laboratory and field data (Geckler, J. R., et al. 1976. Validity of Laboratory Tests for Predicting Copper Toxicity in Streams. EPA-600/3-76-116. U.S. EPA. Duluth, MN 208 pp.), it was found that effects observed in laboratory exposures were also observed in field exposures. However, avoidance, which was not studied in laboratory exposures, was observed in the field exposures. Laboratory to field comparisons are not simple because several factors must be taken into account, the laboratory test must be conducted well and the field observations and measurements must be extensive. Although adverse effects observed in laboratory tests will usually occur in similar field situations, a problem exists with the bioaccumulation of some persistent substances. For example, PCB's seem to bioaccumulate to much higher levels in some bodies of water than they do in laboratory tests.

21. Comment—The Guidelines should place more emphasis on field information than on laboratory information.

Response—Field information on effects of pollutants on natural populations is acceptable, but the collection of definitive information of this type is high risk and costly. Few studies on the effects of pollution on natural populations provide definitive information because of the multitude of variables that need to be taken into account. The major advantage of field studies is that conditions are natural (i.e., conditions are not controlled), but this is also the major problem with field studies. With uncontrolled conditions, numerous variables must be taken into account, because any individual variable or combination of variables may affect the results or indeed may be the cause of the results. Therefore, field studies on natural populations usually must last over several seasons and possibly over more than one year to be reasonably sure that proposed cause-and-effect relationships are real.

Another problem with field studies that are based on statistically significant differences is the power of the test. Because natural biological, spacial, and temporal variability is often rather great, a large number of samples is usually required to detect even a moderate change. A field study which purports to show that no change occurred is of no value if the power of the test calculated from the experimental design and observed variability was not high enough.

Because field studies are high cost-high risk ventures, well-designed laboratory tests are usually much more cost-effective for obtaining data on (1) the toxicity of substances to a variety of species and (2) the effect of various water quality characteristics on toxicity. Laboratory tests have been shown to generally be useful predictors of what happens in a field situation, and so it makes little sense to conduct high risk, high cost field studies rather than laboratory tests. Even definitive field studies rarely provide enough information to allow extrapolation of results to other situations, so field studies are more useful in reviewing criteria than in deriving criteria.

22. Comment—Field verification of laboratory tests and of the Guidelines are needed.

Response—Field verification of laboratory tests end of the Guidelines are certainly desirable and provide information that cannot be obtained in a laboratory. Field verification studies do not need to be as risky or as costly as studies on the effects of a pollutant on natural populations because verification studies can be designed (1) as a side-by-side comparison of the results of laboratory tests and field tests or (2) based on existing results of laboratory tests.

23. Comment—EPA should allow criteria to be derived using on-site acute toxicity tests and an application factor.

Response—This approach is usually suggested for developing effluent standards but may be just as applicable to deriving water quality criteria under certain conditions. This approach cannot be used with pollutants whose most sensitive adverse effect is due to residues. Also, it can only be used when the application factor has already been acceptably determined. Finally, acute tests must be determined with either an appropriate range of species or with an appropriate sensitive species. The implementation policy presently being developed by EPA will probably allow the use of appropriate on-site toxicity tests in the development of site-specific criteria and standards.

24. Comment—It is not clear what level of protection is intended.

Response—EPA feels that it is not possible to specify a minimum level of protection that is necessary to "protect aquatic life" or even to protect a particular species for such reasons as:

a. There are so many untested species.

b. Little practically useful information is available concerning synergism, antagonism, ecological relationships, and avoidance.

c. The effect of factors such as temperature on toxicity seems to be species-specific for at least some substances.

d. Information is not available concerning what amount of any effect would be ecologically significant and whether the amount is species-specific.

One possible conclusion is that to protect aquatic life, all species must be adequately protected. A possible extension of this would be that all criteria should be zero because any amount of any pollutant may affect some aquatic organism. Indeed, the assimilative capacity of body of water largely depends on the ability of aquatic life to "process" pollutants and to some extent, any organism which "processes" a pollutant is in some way affected by it.

The apparent level of protection is different for each kind of effect (acute toxicity to animals, chronic toxicity to animals, toxicity to plants, and bioaccumulation) because of the quality and quantity of the available information. An attempt was made to take into account such things as the importance of the effect, the quality of the available data, and the probable ecological relevance of the test methods. Thus it was felt that with regards to toxicity to animals it was probably not necessary to protect all of the species all of the time, but it certainly seems appropriate to protect most of the species most of the time and to protect important species.

On the other hand, the data base on toxicity to aquatic plants is usually very small and a variety of tests and endpoints have been used, especially with algae. Also, little information is available concerning the ecological relevance of the results of any toxicity test with algae in a concentrated test medium, especially because so many species of algae exist in each body of water.

The results of bioconcentration tests with organic chemicals, but not with inorganic chemicals, can apparently be extrapolated reasonably well based on percent lipids from one aquatic animal species to another, at least within commercially and recreationally important species. In addition, the limits on acceptable concentrations in tissue are reasonably well defined in some cases.

These kinds of considerations merely illustrate the complexity of the problem and the necessity for making decisions about each kind of effect individually. In addition, it is important to distinguish between the apparent level of protection provided by the Guidelines and the actual level of protection which will result in a field situation from the use of the implementation policy.

No attempt was made to develop Guidelines which would achieve a predetermined numerical level of protection. For each effect much desirable information is not available, and so it would be misleading to imply a level of sophistication that is not currently possible. EPA believes that the present state-of-the-art in aquatic toxicology does allow some useful conclusions about the ability of a substance to adversely affect aquatic organisms and their uses whenever the requirements of the minimum data base are satisfied, with the full realization that the resulting criterion may be somewhat overprotective or underprotective.

In almost all cases more data would be desirable and so an attempt to reach the "golden mean" will sometimes result incriteria being to high and sometimes too low. One alternative is to derive no criteria until all desirable data are available; this is unacceptable because it will almost always result in no criteria and no protection. The other alternative is to apply safety or uncertainty factors that are inversely proportional to the adequacy of the data base. In the long run this approach would encourage the generation of useful data where it was most needed, but in the short run would require many significant subjective decisions beyond the current state-of-the-art.

25. Comment—The Guidelines should not base criteria on "worst case" assumptions.

Response—The phrase "worst case assumptions" usually refers to the assumption that both the worst water quality and the most sensitive life stage occur at all times. These two assumptions are a natural result of the two concepts that criteria should be constant throughout the year and that aquatic life is not adequatley protected if it is not adequately protected throughout the year. The implementation policy being developed by EPA will determine whether site-specific criteria must be constant throughout the year. If not, then the "worst case assumptions" will not apply. Although the Guidelines might be viewed as making the "worst case assumptions", the implementation policy will determine whether the site-specific water quality criteria and standards will be based on these assumptions.

26. Comment—Safety factors should be used to protect against such things as potential subtle, but important, long term effects.

Response—Pollutants may cause many direct and indirect adverse effects which have not been studied adequately. For instance, some substances may make aquatic organisms more susceptible to disease or other stresses. in spite of such possibilities, the available information indicates that the major possible adverse effects are covered in the Guidelines and that adequate protection will usually be achieved without the use of safety factors. Safety factors would certainly offer additional protection, but the available information does not show that significant additional protection is needed.

Safety factors of from 10 to 1000 are often used to protect people mainly because people feel that people are more important than aquatic organisms and because humans are usually protected on the basis of tests with other species of animals, thus resulting in a greater uncertainty in the applicability of the results. Complete protection can only be achieved by setting all criteria at zero. Unfortunately, even "Mother Nature" sometimes seriously harms large groups of aquatic organisms, such as during droughts or severe winter freezes. EPA feels that complete protection is neither feasible, desirable, nor possible. In addition, aquatic ecosystems can recover from some adverse effects.

27. Comment—The Guidelines do not provide for an adequate margin of safety.

Response—If "margin of safety" is interpreted to mean "safety factor", then the Guidelines do not provide a margin of safety. If the Guidelines are viewed as deriving criteria for a constant quality water, then they provide a margin of safety during those portions of the year during which the most sensitive life stage does not occur. Although some species may occasionally be adversely affected, EPA feels that the Guidelines provide adequate safety because aquatic communities and their uses should not incur any substantial or permanent damage. Whether or not site-specific criteria will have a margin of safety will depend on how they are derived.

28. Comment—Criteria should be set at the least restrictive concentration and states can then apply more restrictive concentrations when necessary.

Response—It is unclear what is meant by the "least restrictive concentration" but presumably it would be a concentration which would not protect very many aquatic communities and their uses. This is contradictory to the concept that criteria are to protect aquatic life and its uses. The implementation policy being developed by EPA will allow site-specific criteria to be higher or lower than the criteria derived using the Guidelines, when adequate information is available.

29. Comment—The Guidelines should produce criteria in the form of a concentration-risk curve with appropriate confidence limits for each kind of effect.

Response—EPA feels that a risk analysis approach is certainly desirable, but far beyond the state-of-the-art at this time. When dealing with safety to humans, only one species is being protected and extrapolations are made far outside the limits of the actual test results, such as to 1 death in 100,000 people. With aquatic life, numerous species need to be protected and extrapolation far beyond the actual data is not readily accepted. In addition, safety or uncertainty factors are more readily accepted when protecting people than when protecting aquatic organisms.

Most aquatic toxicologists are not willing to let criteria for the protection of aquatic life be as dependent on mathematical models, assumptions, and manipulations as on the actual test results. Most people with experience in aquatic toxicology have an intuitive "feel" about how data should be interpreted and the Guidelines are merely an attempt to formalize a reasonable approach. The Guidelines could be written as mathematical algorithms and some approach such as error models could be developed in order to derive confidence limits. However, the algorithms and models would contain many unproven assumptions and, to be worthwhile, would undoubtedly require more data than are usually available. Although such models and algorithms would be acceptable to many statisticians and may be an appropriate future goal, the current Guidelines need to be useable by and comprehensible to current aquatic toxicologists. Most experienced aquatic toxicologists will judge the reasonableness of any set of Guidelines by comparing the resulting criteria for various pollutants with the data available for those pollutants using a "common sense" interpretation of data.

30. Comment—The Guidelines should not use unsound statistical procedures or misuse sound statistical procedures.

Response—EPA has tried to make sure that no statistical procedures are misused in the Guidelines, that no unsound statistical procedures are used, and that the purposes of the calculations are explained adequately.

31. Comment—It appears that geometric means were used instead of arithmetic means in the Guidelines to obtain lower values.

Response—Decisions such as this were made throughout the Guidelines on a case-by-case basis, and none were based on whether the resulting criteria would be higher or lower. The selection of the procedure used to calculate the mean could be based on the distribution of the values in the individual data set. Unfortunately, with small data sets rarely is it possible to reject many possible distributions and with large data sets all possible distributions are often rejected. Because many of the data sets of interest in the Guidelines are small, a reasonable approach is to base the selection of a procedure for calculating the mean on some general principles such as:

a. Sets of ratios and quotients are likely to be closer to lognormal than normal distributions. Thus geometric means, rather than arithmetic means, are used for acute-chronic ratios and for bioconcentration factors.

b. When there are numerous independent possible sources of error for each datum in a set, the error tends to be multiplicative rather than additive. Thus when the acute or chronic toxicity of a substance to a particular species is determined in different laboratories using different batches of organisms, different waters, etc, the geometric means should be used to calculate the species mean value rather than the arithmetic mean.

c. If a set of numbers approximates a lognormal distribution, the logarithms of the numbers will approximate a normal distribution.

d. The distribution of the sensitivities of individual organisms in a toxicity test is likely to be closer to a lognormal distribution than a normal distribution. Thus the geometric mean, rather than the arithemetic mean, of the upper and lower chronic limits is used.

32. Comment—There should not be any criteria which apply to all bodies of water. Criteria should be specific for individual states, regions, other geographic areas, or bodies of water.

Response—The Guidelines are designed to provide guidance in the collection and interpretation of data concerning the effects of pollutants on aquatic life and its uses. The uses of the resulting criteria will be described by EPA in various regulations. If desired, the Guidelines can be appropriately modified and used to derive a criterion specific to one or more bodies of water or geographic areas if an appropriate data base is available. The critical literature reviews on which the criteria are based will be available for use in the derivation of local, state, or regional criteria. The latitude allowed for deriving local, state, or regional criteria and standards will be determined by the implementation policy presently being developed by EPA.

33. Comment—The Guidelines should result in criteria that are specific for individual species or groups of species (e.g., warmwater and coldwater).

Response—If the necessary data were available, criteria could be derived for any particular species or group of species. It was impractical for EPA to derive criteria for many such groups, but a relatively simple division is freshwater and saltwater organisms because these two groups rarely coexist. Most other possible general divisions of species are faced with the problem that species coexist in various combinations unless the groups are very narrow. In addition, toxicity data are rarely available for very many individual species and so data for representative species must be used, unless appropriate new data are generated. Also, the available data sometimes show wide differences within families so extrapolations from one species to another are often tenuous. Because of these problems, deriving criteria for individual species or groups of species was deemed impractical.

34. Comment—A criterion should be one number, not two.

Response—The two-number criterion is an acknowledgement that aquatic organisms can tolerate short exposures to concentrations that are higher than those they can tolerate continuously. In a two-number criterion, the higher number can assure that short-term fluctuations above the average are not too high, whereas the lower number can assure that the long-term average is not too high. A one-number criterion could be derived by using the existing 24-hour average as an instantaneous maximum. This would certainly provide additional protection, but would provide unnecessary overprotection in most cases. Because a one-number criterion would be more of an approximation than a two-number criterion, one-number criteria would be too high or too low more often and to a greater degree then two-number criteria.

35. Comment—The criteria should not specify sampling schemes.

Response—Criteria should state numerical concentration limits in terms of exposure durations because, everything else being constant, the amount of adverse effect depends on both the concentration of the pollutant and the duration of exposure. Criteria in the Green Book, Blue Book, and Red Book were usually stated as single numbers with no duration expressly stated. The implication was that the criteria were never to be exceeded at any time. Each criterion was apparently and instantaneous maximum. In practice, however, standards derived from these criteria were usually enforced on the basis of 24-hour composite samples. To avoid any ambiguity, the Guidelines specify that a criterion should be explicitly stated in terms of two time frames: an instantaneous maximum and a 24-hour average. However, this is not a specification for a sampling scheme. Standards developed from such a criterion should probably specify a sampling scheme for compliance monitoring, but it would not necessarily be in terms of point measurements and 24-hour averages.

Any sampling scheme used to determine whether or not an ambient concentration exceeds a water quality criterion or a comparable water quality standard should take into account such things as the ratio of the instantaneous maximum and the 24-hour average and the retention time of the body of water because these will primarily determine which portion of the criterion is most limiting in any specific situation. The sampling scheme should probably also take into account the cost of the analyses and results of any past analyses.

36. Comment—The criteria should be stated in terms of time frames longer that an instantaneous maximum and a 24-hour average.

Response—These two time frames were chosen because they would allow the derivation of a criterion which would be less restrictive than, but just as protective as, the previous one-number criterion. These two specific time frames were chosen because they match two kinds of samples that are commonly collected: grab samples and 24-hour composite samples. These specific time frames could probably be changed somewhat without much practical effect, but EPA saw no particular advantage to anyone to introducing novel time periods. For example, for all practical purposes in most situations a 10-minutes average is probably about the same as an instantaneous maximum.

Large increases in the time frames, however, would not provide the same amount of protection. If the instantaneous maximum were changed to a 24- or 96-hour average, and the 24-hour average were changed to a 7- or 30-day average with no change in the numerical limits, the amount of protection afforded aquatic life would fall to an unacceptable level. The longer the time span for the average, the higher the instantaneous concentration could be for short periods of time within that span. Although most chronic tests last for 28-days or longer, some chronic effects may be caused by short exposures of sensitive life stages. If the acute-chronic ratio is small, fluctuations in the instantaneous concentration may even cause acute toxicity, especially for cumulative pollutants, because for some substances the 24-, 48-, and 96-hour acute values do not differ too much.

37. Comment—A two-number criterion will be difficult to enforce.

Response—Criteria are not enforceable. Standards are enforceable. When standards to protect aquatic life are developed, they may or may not be in the same format as the criteria for aquatic life. Few standards are adequately enforced because of the high cost of continuous monitoring. The real value of many criteria and standards is in the design of waste treatment facilities; a two-number criterion should be a better basis for design than a one-number criterion.

38. Comment—The criteria should be expressed to one significant figure, not two.

Response—EPA acknowledges that there is much variability in some of the data and that the range of sensitivities is often great. When the requirements of the minimum data base are satisfied and the data agree reasonably well, two significant figures are not unreasonable. Rounding off to one significant figure could arbitrarily raise or lower the criterion by up to forty percent with no apparent consistent benefits to dischargers, regulators, or aquatic life.

39. Comment—The Guidelines should only use data for species that ought to be protected.

Response—In order to protect commercially and recreationally important species, a wide variety of "unimportant" species must also be protected. Such so-called "unimportant" species include the food organisms all the way to the bottom of the food chain. The "important" species in an aquatic community cannot maintain themselves without the help of primary producers, primary consumers, nitrifiers, dentrifiers, detritivores and saprophytes.

40. Comment—Criteria should not be based on sensitive, short-lived invertebrates.

Response—Many species of invertebrates are short-lived and are not widely distributed. However, these numerous short-lived, local species do serve important functions and should be represented in the data base. This group of organisms needs to be protected even if no one species can be considered important.

41. Comment—Criteria should protect endangered species.

Response—EPA agrees that criteria should protect endangered aquatic species. However, very few toxicity tests have been conducted with endangered species, and it does not appear feasible to require tests with such species. Endangered species are some of the many untested species which should be protected by criteria derived from available data using the Guidelines.

42. Comment—Migratory species are a special problem.

Response—Migratory species should usually be protected by criteria derived using the Guidelines unless such species are unusually sensitive. Migratory species may be especially susceptible to avoidance, but few data are available to compare species on this basis. Avoidance may be a serious latent problem because it might apply to all motile species, rather than just migratory species, and it has not been studied very much.

43. Comment—Estuarine species were ignored.

Response—The term "saltwater organisms" is meant to include estuarine species as well as true marine species.

44. Comment—The classification "invertebrates" includes species that are too dissimilar to be grouped together. These species should be separated into phyla or classes.

Response—The never-ending arguments between the "lumpers" and the "splitters" can only be resolved by considering the advantages and disadvantages of each approach in each situation. The "splitters" can usually argue that obvious differences should be taken into account and it is certainly true that shrimp are different from insects and both are different from worms. It can also be argued that there are significant differences within phyla, classes, and families. Each species could be considered a separate group, if differences between stains are arbitrarily ignored. After the species are split into separate groups, the problem then would be whether to recombine the data to derive one criterion for all species or to derive one criterion for each group. If numerous criteria are derived for a pollutant, how are these to be used to develop standards? Another problem is that unless more data are generated, the greater the number of groups, the less information there is available per group.

The basic question is "What are the important differences that need to be taken into account and how should this be done?" Because there are differences between taxonomic groups, the Guidelines require data on a number of species from a varitety of taxonomic groups. The information of each separate species is treated individually. This approach preserves the differences between species and allows all species to be considered in the development of the criterion. The number of data points is increased and the range of the data is readily apparent. Because "invertebrates" is already a large diverse group and because the range of sensitivities of fish usually overlaps that of invertebrates, little justification exists for not combining all aquatic animals.

45. Comment—Do not extrapolate from freshwater organisms to saltwater organisms or vice versa.

Response—Criteria and absolute toxicity values were not extrapolated from fresh water to salt water, but some relative data were, when it did not appear that factors such as salinity affected the data. The toxicity of some substances apparently is significantly affected by salinity, but most substances seem to have overlapping ranges of toxicity to freshwater and saltwater organisms. However, because these two kinds of organisms rarely inhabit the same body of water simultaneously, separate criteria were derived for each. Even though these two kinds of organisms are physiologically different, they do not seem to be too different toxicologically. Bioconcentration factors and acute-chronic ratios seem to be fairly similar for many freshwater and saltwater species for many pollutants, particularly organic chemicals.

46. Comment—The Guidelines base the criteria only on sensitive species and do not take into account insensitive species.

Response—The Guidelines do not necessarily base the criteria on the data for the most sensitive species. However, an aquatic ecosystem cannot be protected by protecting only the species which are insensitive. Protecting half the species will probably not protect the community. To offer reasonable protection to aquatic life and its uses, each major kind of organism and each major use must be given reasonable protection. In some cases it may in fact be necessary to protect the most sensitive species if it is a highly desirable species.

47. Comment—Species should be tested at their environmental extremes.

Response—Toxicity tests with each pollutant could indeeed be conducted with some or all species under a variety of extreme conditions and the lowest result obtained with a species could be used instead of a mean result. On the other hand, differences between results with different species seem to be much greater, and therefore more important, than the differences between results obtained with one species under different conditions. Furthermore, criteria need not necessarily protect species from all stress under the most extreme conditions, because aquatic communities and populations of individual species can recover from some perturbations.

48. Comment—Only data for species that are widely distributed, representative, critical, indigenous, important, ecologically relevant and sensitive should be used.

Response—Few species would satisfy all of the requirements that have been suggested. As more and more data are obtained with a wider variety of species for any one pollutant, it becomes more obvious that few if any species are atypically sensitive, although that may not be true for aquatic communities which contain very few species. No data exist to show that species in any one key role are toxicologically more sensitive than other kinds of species. Ecologically relevant species and species that have key roles or are relevant to the overall functioning of viable ecosystems are not necessarily toxicologically different from other species. EPA feels that if the available data cover an adequate number and variety of species, it is not necessary to try to identify and conduct tests with all important, sensitive species. In addition, the derivation of a criterion should not be based only on sensitive species, because a knowledge of the range of sensitivities may be useful. For instance, elevated concentrations of a pollutant that produces a narow range of species sensitivities are likely to cause more damage than elevated concentrations of a pollutant that produces a wide range of species sensitivities.

49. Comment—The distinction between ionizable and unionizable compounds is not very good because some chemicals ionize and reach chemical equilibrium very slowly and others very rapidly.

Response—Most chemicals can readily be classified into one of three groups:

A. Chemicals that ionize, including hydrolyze, at least 90% and reach 90% of equilibrium in less than 8 hours in most surface waters.

B. Chemicals that ionize, including hydrolyze, less than 10% in 30 days in most surface waters.

C. Chemicals that do not fit into either one of the above categories.

For the purpose of the Guidelines, chemicals in the A group should be considered ionizable, chemicals in the B group should be considered non-ionizable, and chemicals in the C group should be classified on a case-by-case basis. Although the distinction between ionizable and unionizable may not be perfect, it is very useful for most chemicals.

50. Comment—Each individual organic compound should be considered separately.

Response—The vast majority of organic chemicals will be considered separately according to the Guidelines except for structurally similar organic compounds that meet all three specifications given in the Guidelines, such as polychlorinated biphenyls and toxaphene.

51. Comment—In-stream water quality criteria are meaningless for substances that are highly insoluble.

Response—The concentration of some substances in sediment may be important separate from the concentration of the substance in the ambient water and for these compounds a sediment quality criterion may be necessary. Generally such compounds can also cause adverse effects if the concentration in the ambient water is too high even if the concentration in the sediment is low. Thus for such compounds both kinds of criteria may be necessary rather than just one or the other.

52. Comment—If a substance is not dissolved, it is not biologically or toxicologically available.

Response—Although this may usually be true, it certainly does not apply to elemental mercury which can be oxidized and methylated to form a very toxic compound. Some organic acids and phenols and hydroxide and carbonate salts of metals have solubilities which differ substantially from one body of water to another.

53. Comment—Criteria for metals should not be for total metal.

Response—Criteria for metals will generally not be based on total metal. Most will be based on total recoverable metal because forms of metals that are not measured in the total recoverable procedure probably are not, and will not become, toxic. A major problem is that some people use a procedure for total recorverable, but report the results as total, metal. In many situations the two results are about the same, but in some cases the results are quite different.

54. Comment—The Guidelines should give more guidance for distinguishing between acceptable and unacceptable data.

Response—The Guidelines contain as much detail on this subject as EPA believes is currently feasible. Items such as the maximum acceptable control mortality and minimum number of test organisms are based on what many aquatic toxicologists generally feel are acceptable, as expressed in published methods. No data should be used in the derivation of a criteria until their quality and acceptability had been reviewed by a competent person. Competent people will occasionally disagree, but that is a fundamental property of subjective decisions.

55. Comment—Only published data should be used.

Response—Peer review is one of many concepts that is better in theory than in practice. Some poor quality data are published and some high quality data are rejected. In addition, publication is not a particularly rapid process. Whether or not data are used should depend on the applicability and quality of the data, not on whether they have been published. Data that are not published should be made readily available if they are used to derive water quality criteria.

56. Comment—All static test are unacceptable

Response—In general, high quality flow-through acute tests are preferable to high quality static acute tests, but static tests are by no means unacceptable. Few data are available to show whether static tests consistently produce acute values lower or higher or different than flow-through tests. Whereas degradation, violatilization, and buildup of metabolic products are more likely to be a problem in static tests, operator and mechanical errors are more likely in flow-through tests. Static acute tests are certainly not unacceptable for most pollutants, but static chronic tests generally are unacceptable because of changes in the toxicant concentrations and the quality of the dilution water during the test.

57. Comment—Data obtained using test organisms that were previously exposed to the pollutant should be used.

Response—Comparisons of results obtained with unexposed and previously exposed organisms should indicate whether or not acclimation has occurred. Generally, data obtained with acclimated organisms should not be used in deriving criteria because acclimated organisms are the exception rather than the norm. Rarely, if ever, can acclimation be depended on to protect organisms in a field situation because concentrations often fluctuate and motile organisms do not stay in one location very long. Data obtained with acclimated organisms may be acceptable for use in deriving some site-specific criteria.

58. Comment—Foreign species should be used to expand the data base.

Response—Foreign species may be representative of indigenous species, but some of them are quite unusual. Data obtained with foreign species may give good indications of indigenous speceis that should be used in tests on some pollutants and may identify some potential problems that should be investigated.

59. Comment—If data for brine shrimp are not used, the criteria should not apply to saline waters.

Response—Data obtained using brine shrimp are not used because these organisms are atypical. Although they may not be usually sensitive or insensitive to various pollutants, the species found in North America and used for testing only survive in the Great Salt Lake and in salt ponds near San Francisco Bay. These two habitats are unlike any others in the United States. If criteria were to be derived specifically for the Great Salt Lake or for salt ponds, then data for brine shrimp should be used.

60. Comment—Structure-activity relationships should not be used unless proven.

Response—No provision is made in the Guidelines for the use of structure-activity relationships. Such relationships may soon be well enough understood that they can be used in deriving water quality criteria.

61. Comment—A criterion should not be derived for a pollutant until data are available for a broad range of commercially, recreationally, and ecologically important species. Each species should be acutely and chronically tested under a variety of conditions in a number of different waters.

Response—Except for those people who merely want to stop EPA from deriving any water quality criteria, most people will admit that there must be some reasonable limit as to how much information is necessary concerning any regulatory action. This is as true for deriving water quality criteria, as it is for issuing NPDES permits, submitting PMNs, registering pesticides, etc. All of these regulatory activities deal with potentially significant adverse effects on aquatic organisms and should take into account many of the same possible kinds of adverse effects. Therefore, the data needs for these various activities should probably be somewhat similar, but for each regulatory activity the minimum data requirements also need to take into account the special aspects of the program and practical considerations. Unrealistic data requirements will benefit no one. It is not necessary that all questions be answered before any action is taken. It is only necessary that enough data be available to allow reasonable confidence that the water quality criteria will generally not be too high or too low.

EPA has developed minimum data requirements that describe the amounts and kinds of information that should usually be available if a criterion is to be derived using the Guidelines. When the minimum data requirements are satisfied, it should usually be possible to derive a useful criterion. The requirements take into account many things such as:

a. The existence of some species which are commerically or recreationally important and generally sensitive to some broad classes of pollutants;

b. The range of species for which data are available;

c. The cost of obtaining additional data and the usefulness of the data; and

d. The reasonableness of extrapolations from one species to another within and between groups.

The requirements set forth in the minimum data base are indeed minimal, considering the great varitey of species which exist in most aquatic ecosystems. However, EPA feels that based on the availavble information the routine requirement of more data would probably not improve criteria enough to justify the additional cost.

62. Comment—The mimimum data requirements should depend on the nature of the pollutant.

Response—EPA feels that such an approach may be feasible some time in the future, but would be an unwarrented level of sophistication at this time. For a few pollutants, it may be possible to relax some of the data requirements, but in general this can only be determined after enough data are available to indicate that a special case exists. In other cases the minimum data may indicate that additional data are highly desirable.

63. Comment—Criteria should not be derived if enough data are not available. The alternative procedures which were proposed should not be used.

Response—EPA agrees that a numerical criterion should not be derived if enough appropriate data are not available, except in some special cases. EPA also agrees that the alternative procedures which were proposed should not be used to develop numerical criteria at the present time. However, EPA feels that when a numerical criterion is not derived, a descriptive criterion can be used to accurately reflect the latest scientific knowledge.

64. Comment—The guidelines should give more guidance on relating a criterion to a water quality characteristic.

Response—More detail on this subject has been written into the Guidelines.

65. Comment—If data on the relation of toxicity and water quality are not available, no criterion should be derived.

Response—The purpose of a criterion is to present the best available information, not to ensure that all desirable information is available. Any water quality characteristic may affect the toxicity of each pollutant to some degree and it is never going to be possible to investigate all such interactions for even a few species and pollutants. EPA has adopted a minimum data base requirement for deriving a criterion, but there must be practical limits or no criterion will ever be possible. When the minimum data base requirements are satisfied, a criterion should be derived regardless of speculation that some unstudied relationship exist. When enough good data demonstrate a relation between toxicity and a water quality characteristic, an attempt should be made to use this information in the derivation of a criterion. A major purpose of site-specific criteria is to take into account the effect of local water quality conditions on toxicity.

66. Comment—Do not specify the form that a relationship between toxicity and water quality must take.

Response—The Guidelines allow the use of any set of transformations that fit the data well. The log-log model is given as an example because it seems to fit most of the available data concerning the relationship between hardness and toxicity of metals (the only such relationship for which much quantitative data are available) reasonably well.

67. Comment—The toxicity of metals should not be related to "hardness".

Response—EPA has tried to derive criteria in a form that will (a) adequately protect aquatic organisms and (b) be practically useful. Hardness is used as an easily measured surrogate for a number of interrelated water quality characteristics, such as pH, alkalinity, calcium, and magnesium. Various combinations of these probably affect individual metals differently, but these are all reasonably well correlated with hardness in a wide variety of natural waters. Some waters, such as those impacted by acid mine drainage, obviously are special cases, but they have special problems of their own.

68. Comment—Do not extrapolate slopes for toxicity vs. water quality from fish to invertebrates or from acute values to chronic values.

Response—The Guidelines do not now assume that the acute slope and the chronic slope are similar for a pollutant. On the other hand, there is no reason to believe that invertebrates are more similar than are fish and invertebrates. As explained earlier, the group "invertebrates" does not consist of a collection of species that are similar taxonomically or toxicologically. Some water quality characteristics apparently affect the toxicity of the pollutant, rather than the sensitivity of the organisms. For these kinds of factors, slopes should be the same for different species. Even factors that affect such things as the permeability of membranes may produce similar slopes for a wide variety of species. If each species must be treated separately, no criteria will ever be possible.

69. Comment—Relationships based on only two points should not be used.

Response—Two points certainly do not provide very much information about the shape, slope and position of a line. However, if other information or a reasonable assumption is available concerning the shape of the line, two good data points, spaced at a reasonable interval, can provide very useful information concerning the slope and position of the line. Three appropriately spaced points would certainly be better, and four points would be an ideal minimum.

70. Comment—Do not combine relationships that are and are not statistically significant.

Response—The Guidelines do now specify that relationships should be tested for statistical signficance. A test for statistical significance may be one indication of whether or not a slope is useful, but such a test cannot be used with just two points and does not take into account such things as the comparability of the data, the quality of the test, and the range of the independent variable. A relationship based on six points may not be as significant as it seems if five of the points are tightly grouped.

71. Comment—The Guidelines should not combine 96-hr LC50 values and 48-hr EC50 values.

Response—Both LC50 values and EC50 values are used to measure acute toxicity of a substance to aquatic organisms. In general, an EC50 can be based on a wide variety of effects, but the Guidelines specify that the only effects to be used for deriving criteria are incomplete shell development, immobilization, and loss of equilibrium. All of these are certainly drastic effects. In a field situation these effects probably often lead to death. Just as the endpoint may be specific for the species, so may be the length of the test. The generally accepted length of an acute test with daphnids is 48 hours, whereas for most species of fish, it is 96 hours. Thus the Guidelines use both 48-hr EC50 values and 96-hr LC50 values because they are the widely accepted durations and endpoints used to measure acute toxicity to specific species.

72. Comment—Shell deposition tests are chronic tests and should not be equated with lethality tests.

Response—"Acute" implies "short" not "death". Many acute toxicity tests do use death for the effect, but many also use non-lethal effects. The shell deposition test is one of many non-lethal acute tests and is generally accepted as a short test compared to the average life span of oysters.

73. Comment—Adjustment factors should not be used to adjust for the length of the test, the technique, and unmeasured concentrations.

Response—All three kinds of adjustment factors have been deleted from the Guidelines. The factor for the length of the test was found to be unnecessary because most tests had been conducted for the standard times usually specified for the individual species. Thus the Guidelines now specify that only data from tests conducted for the time specified for the species should be used to calculate the Final Acute Value.

EPA has found that on the average flow-through acute tests give results slightly lower than do static tests, but the relationship does not seem to be too consistent and may vary from species to species for some pollutants. In addition, on the average results based on measured concerntrations do not seem to be much different from those based on unmeasured concentrations.

However, the results of flow-through tests based on measured concentrations are generally accepted as being better measures of acute toxicity than the results of flow-through tests based on unmeasured concentrations or the results of any static or renewal tests. Therefore, whenever the results of flow-through acute tests in which the concentrations were measured are available, the results of all other kinds of acute tests with that species and pollutant are not used in the calculation of the species mean acute value.

74. Comment—Species sensitivity factors should be pollutant-specific; and average factor should not be calculated for a variety of substances.

Response—EPA agrees. The requirement for acute values for at least eight different species was developed in part to allow for a reasonably good calculation of a mean acute value and a species sensitivity factor for each individual pollutant. A better way of using the acute values for the individual species has been developed, but no extrapolations are made from one pollutant to another.

75. Comment—The distribution of species mean acute values for a pollutant will be truncated if the species cannot be killed or affected by concentrations above solubility.

Response—Some species are so resistant to some pollutants that they cannot be killed or affected in acute tests even by concentrations which are much above solubility. Such "greater than" values cannot be used in the calculation of means and variances for pollutants. When the "greater than" values are for insensitive species and are at or above solubility, the values can be used in the calculation of the Final Acute Value by adjusting the cumulative proportions for all the speices with quantitative values. The shape of the curve at the high end cannot be determined, but the Final Acute Value is more dependent on the species mean acute values and the cumulative probabilities at the low end.

76. Comment—Early life-stage tests with fish should be used interchangeably with life-cycle and partial life-cycle tests with fish.

Response—EPA agrees that early life-stage tests with fish generally give about the same results as comparable life-cycle and partial life-cycle tests. However, because the shorter test is merely a predictor of the longer tests, whenever both kinds of results are available, the results of life-cycle and partial life-cycle tests should be used instead of the results of early life-stage tests.

77. Comment—Appropriate measures of chronic toxicity and appropriate lengths of exposure should be defined.

Response—The descriptions of appropriate chronic tests have been clarified.

78. Comment—The factor of 0.44 should not be used.

Response—It is not now used.

79. Comment—The Final Chronic Value should not be lower than the lowest measured species chronic value, even if chronic data are not available for sensitive species.

Response—Aquatic ecosystems cannot be protected from chronic toxicity by protecting only the insensitive species from chronic toxicity. In the past both arbitrary and experimentally determined application factors have been used to relate acute and chronic toxicity. For a variety of reasons the Guidelines do not use an application factor, but instead use the acute-chronic ratio, which is similar to the inverse of an application factor. Thus the acute-chronic ratio should normally be greater than one. The acute-chronic ratio is to be used with invertebrates as well as fish and is to be an experimentally determined value for each individual pollutant. The acute-chronic ratio should also avoid the confusion as to whether a large application factor is one that is close to unity or one that has a denominator that is much larger than the numerator. The acute-chronic ratio is calculated by dividing the appropriate measure of acute toxicity for the species (as specified in the Guidelines) by the appropriate measure of chronic toxicity for the same species (as specified in the Guidelines).

Some people have confused application factors and safety factors and use of the term "acute-chronic ratio" should help avoid this problem. Acute-chronic ratios are a way of estimating the chronic sensitivity of a species for which no chronic toxicity data are available. Safety factors would provide an extra margin of safety beyond the sensitivity of the species. Safety or uncertainty factors are intended to reduce the possibility of underprotection, whereas acute-chronic ratios are intended to estimate the actual chronic sensitivity of the species to the pollutant. This estimate is just as likely to be too high as it is to be too low. A mean acute-chronic ratio will in fact be too high for half the species and too low for the other half.

When three or more acute-chronic ratios have been determined for a pollutant with both fish and invertebrates, three patterns have been observed when the individual species are listed in order of their species mean acute values:

a. The ratios randomly differ by a factor of ten or more.

b. The ratio appears to be about the same (within a factor of ten) for all species.

c. Species with higher acute values also have higher acute-chronic ratios.

The available data indicate that fish and invertebrates do not consistently have different acute-chronic ratios and that for some pollutants freshwater and saltwater species have similar acute-chronic ratios.

80. Comment—No application factor should be used unless it is specific for the pollutant, species, and water.

Response—There is no point in using an application factor or acute-chronic ratio or any concept if it does not allow some generalization or extrapolation from one species to another or from one water to another. Not allowing any generalizations or extrapolations would require that much data be generated for each species and each pollutant in each water in which a criterion is necessary. When enough supporting data are available, extrapolations using such things as acute-chronic ratios are cost-effective and scientifically sound.

81. Comment—Additional development of methodology for toxicity tests with aquatic plants is needed.

Response—This is most certainly true. Much other research also is needed, and generally is considered higher priority. EPA hopes that someday all of the additional research that needs to be done will be done. Few pollutants seem to affect aquatic plants at concentrations which do not chronically affect aquatic animals, and it is hoped that this is not an artifact of the test methods currently used.

82. Comment—Data on toxicity to plants should not be used for deriving criteria because plants are more site-specific than animals.

Response—Numerous species of plants, especially algae, exist in most bodies of water. On the other hand, EPA knows of no data to support the contention that the sensitivities of aquatic plants are any more site-specific than those of aquatic animals, or that the range of sensitivities between plants is as great as that for animals. One species may or may not be representative of other species. After the methodology for toxicity tests with aquatic plants is better developed, tests with a wider variety of species would certainly be desirable.

83. Comment—The Final Plant Value should not be the lowest available plant value based on measured concentrations.

Response—EPA adopted the procedure described in the Guidelines for obtaining the Final Plant Value for several reasons including:

a. The methodology for toxicity tests with aquatic plants is not well developed.

b. For only a few pollutants have toxicity tests been conducted with more than a very few species of plants.

c. Little is known about the range of sensitivities of various species of aquatic plants.

d. Based on available data, almost no pollutants are toxic to aquatic plants at the lowest concentrations which are chronically toxic to aquatic animals or cause unacceptable residues.

84. Comment—Residue accumulation in any part of an aquatic ecosystem should be prevented as much as possible.

Response—Accumulation of residues in aquatic organisms only becomes a problem if the concentration of residue is high enough to adversely affect either (a) the organism itself, (b) a consumer of the organism, or (c) the marketability of the organism. Adverse effects on the aquatic organism itself will be detected in acute and chronic toxicity tests. The use of FDA action levels and chronic feeding studies with wildlife are designed to protect the uses and consumers of aquatic organisms.

85. Comment—Bioconcentration factors (BCFs) derived from field data should not be used.

Response—EPA feels that BCFs derived from adequate data, whether they be laboratory data or field data, should be used. More data are necessary to document a BCF from a field exposure than a laboratory exposure, as specified in the Guidelines, but if enough data are available, field BCFs should be used.

86. Comment—Kinetically derived bioconcentration factors (BCFs) should be used.

Response—Kinetically derived BCFs should be used if the bioconcentration test lasted long enough, *i.e.*, to apparent steady-state, to verify that the model (assumptions) used in the calculations actually fits the data for the individual pollutant.

87. Comment—Bioconcentration factors (BCFs) should not be estimated from octanol-water partition coefficients.

Response—The available data seem to indicate a reasonably good relationship for lipid-soluble substances between steady-state BCFs and octanol-water partition coefficients. BCFs estimated from partition coefficients are not used in the Guidelines because measured BCFs are available for all pollutants for which a maximum permissible tissue concentration is available.

88. Comment—Bioconcentration factors (BCFs) are dependent on temperature, food, salinity, stress, and other things.

Response—Many things such as these probably do affect BCFs. Until data are available to show that such effects are important and are not species-specific, little needs to be, or can be, done to take such factors into account when deriving water quality criteria.

89. Comment—Bioconcentration factors (BCFs) should be based only on tissues that are actually eaten.

Response—Although people usually only eat muscle tissue of fish, wildlife usually eat the whole body of fish. The tissues used in the determination of BCFs must be appropriate to the kind of consumer organism or regulatory action. On the other hand, since the BCF for a lipid-soluble substance seems to be proportional to percent lipids, extrapolations can be made on the basis of percent lipids regardless of the tissue.

90. Comment—Chronic toxicity tests with rats and mice should not be used as representative of tests on mammalian wildlife.

Response—Because results of tests on a variety of species are extrapolated to man, it should be just as reasonable to extrapolate from one mammalian species to another mammalian species within certain limits. However, such extrapolations are not now used in the Guidelines; only the results of chronic toxicity tests with wildlife are used to protect wildlife consumers of aquatic life.

91. Comment—Information concerning bioconcentration should only be used if such information is used to protect aquatic organisms, not to protect the marketability of aquatic organisms.

Response—Protection of aquatic organisms must include not only the protection of the existence of aquatic organisms, but also protection of the common uses of aquatic organisms. Commercially important aquatic organisms cannot be considered adequately protected if they cannot be sold. The Guidelines do not use any data pertaining to safety to humans in an attempt to protect human consumers of aquatic organisms. Instead, the Guidelines merely attempt to ensure that residues in aquatic organisms do not exceed FDA action levels so that the uses of commercially and recreationally important species are not restricted by the Food and Drug Administration.

92. Comment—A Final Residue Value calculated from an FDA action level is actually a concentration that will result in the average concentration in some species being at the FDA action level.

Response—This is a good point. A similar situation exists when the calculation is based on a concentration which caused an adverse effect in a chronic wildlife feeding study. In all such cases, the Final Residue Value should be lower, but EPA knows of no non-arbitrary way to determine how much lower the value should be.

93. Comment—The FDA action levels for finished animal feed should not be used.

Response—They are not now used.

94. Comment—Flavor impairment should not be used to derive water quality criteria for aquatic life.

Response—Many of the commercially and recreationally important aquatic orgnaisms are consumted by people. If the flavor is significantly impaired, the use of these species will be adversely affected. Flavor impairment should be considered an effect that can adversely affect the use of aquatic organisms.

95. Comment—The instructions for using the other data are not very detailed and are not mathematical.

Response—EPA has tried to include as much detail in the instructions for using the other data as are currently justified. Extensive detail and mathematical treatment are not deemed realistic at this time because so little information is available concerning the various kinds of other data.

96. Comment—The final review of the criteria should allow revision up or down based on sound scientific evidence.

Response—The Guidelines always have allowed revision up or down, but this is now stated explicitly in the Guidelines.

97. Comment—Some bodies of water, such as some USGS benchmark streams and the Houston ship channel, contain concentrations above the criteria for some pollutants and still contain aquatic communities that are diverse, healthy, and productive. Such information should be used in the review of the criteria because it indicates that some criteria are too low.

Response—Rarely are there enough data available to accurately identify the concentrations of pollutants to which aquatic organisms in bodies of water are actually exposed. The sampling scheme should provide a good estimate of the mean and variance of the concentration; a few grab or composite samples cannot provide enough information to characterize the concentrations of pollutants in most bodies of water. The concentrations vary not only with time but also with location at each time, so the samples must be taken where the organisms of interest are located at that time.

A more serious problem concerns the definition of an acceptable aquatic ecosystem. How does one determine if an aquatic ecosystem is healthy or productive? If a diverse system is, by definition, healthy, is it also, by definition, productive? What is the minimum acceptable diversity? What is the minimum acceptable productivity? Should the acceptable levels of diversity and productivity be site-specific? Is a body of water acceptable just because no dead fish are observed. How many pounds of trout should a trout stream produce each year to be considered healthy and productive? How does one treat motile species that may avoid some periodic increases in pollution levels? Is an aquatic ecosystem healthy and productive if the normally edible portion of a consumed species tastes bad or contains excessive residues? Questions such as these indicate the difficulty of quantitatively judging the quality of aquatic ecosystems on the basis of their acceptability or usefulness to man or on any other basis. Although judging bodies of water would be a difficult job, it certainly could be done by a competent group of trained professionals. The point is that it is not as easy a job as some people would like to think. There are also people who feel that various pristine bodies of water should be managed because they are not as productive as they could be.

As mentioned earlier, the criteria documents derive criteria which may be too high or too low for some specific bodies of water. With appropriate modifications the Guidelines can be used to derive criteria for any specific body of water or geographic area. In addition, it is certainly possible that one or more factors which affect the toxicity of one or more pollutants may not have been studied very throughly or even identified yet. The criteria are based on the best available information and the state-of-the-art of aquatic toxicology, but it is always possible that something important has not been adequately studied by regulators, discharges or academia.

Appendix E.—Responses to Public Comments on the Human Health Effects Methodology for Deriving Ambient Water Quality Criteria

I. Introduction

On March 15, 1979, the U.S. Environmental Protection Agency (EPA) announced the availability for public comment of the proposed methodology for the derivation of ambient water quality criteria for the protection of human health. The public comments were resolved in three phases.

First, comments relating to policy issues were resolved in an initial screening/disposition by Agency personnel. Second, a peer review workshop was conducted and involved Agency personnel, contractors, and recognized scientists. The group evaluated all issues pertaining to the derivation of criteria for non-carcinogens, and third, a similar workshop was held to review all issues relating to the derivation of criteria for carcinogens.

The following report presents the resolutions of the public comments by the EPA after considering the advice of the meeting attendees. While the EPA greatly appreciates the contribution of these individuals and acknowledges their substantial assistance in resolving many difficult questions, the EPA accepts full responsibility for the positions outlined in this document. (Note: Comments addressing similar issues were appropriately compiled and summarized under each issue.)

Comments Resolved in Initial Screening

Issue 1

Comment summary: The water quality criteria documents should provide information and/or guidelines for deriving standards from criteria.

Response: The water quality criteria documents contain information which will be useful in developing standards (e.g., current levels of exposure). However, in developing standards, many additional factors not directly related to criteria must be considered. It would be more appropriate to compile and to analyze this information as part of the standard-setting process rather than to include it in the criteria documents. Guidelines will be issued separately since the development of the standard includes use designation with a commensurate criteria value.

Issue 2

Comment summary: Water quality criteria should consider or be limited by technological achievability, cost/benefit analysis, limits of detection, and environmental fate.

Response: The distinction between criteria and standards must be recognized. For non-carcinogens, ambient water quality criteria are estimates of concentrations in water which will not result in either adverse human health effects (criteria based on toxicity) or unpleasant taste or odor (organoleptic criteria). For carcinogens, criteria are estimates of concentrations of individual compounds in water which will result in specified increases in the lifetime risk of developing cancer. By definition, these criteria exclude considerations of technological achievability, cost/benefit analysis, limits of detection, and environmental fate, as appropriate within the authority of The Clean Water Act [33 U.S.C. 1314(a)]. These factors are more properly considered in the standard-setting process.

Issue 3

Comment summary: The validity of a single criteria for all bodies of water is questionable. Criteria should be site specific an/or use specific.

Response: In the standard-setting process, criteria may be modified based upon site specific or use specific considerations.

Issue 4

Comment summary: Even if there is insufficient data, some criteria must still be developed for "highly hazardous compounds."

Response: If there is sufficient information to indicate that a compound is "highly hazardous," there should be sufficient information to derive a criteria. Conversely, if insufficient data are available, by definition no criteria can be derived.

Issue 5

Comment summary: Criteria should be derived only for persistent compounds or for compounds which present a clear hazard to humans.

Response: Criteria can be derived for any compound on which sufficient information is available. By definition, criteria are independent of persistence or current levels of exposure.

Issue 6

Comment summary: Criteria should be developed to protect terrestrial wildlife as well as humans and aquatic organisms.

Response: Because of the great number of diverse wildlife species and differences in their habitat, diet, and behavior, it is unlikely that a single criteria could be developed to protect all wildlife species from a given contaminant. The EPA is currently assessing possible approaches to developing a valid methodology for deriving wildlife criteria. Until a specific wildlife criteria methodology is developed, the proposed aquatic life and human health effects criteria should serve as interim levels for the protection of wildlife.

Issue 7

Comment summary: Criteria should be derived by an independent scientific panel and not by the EPA.

Response: The EPA has a legislative mandate to derive ambient water quality criteria and must accept the final responsibility for this process. However, the EPA has solicited the advice of many independent scientists in this effort. It should be noted that the consensus of the peer review committees has been considered and generally followed by the EPA. Nonetheless, the responsibility for the criteria rests solely with the Agency.

Issue 8

Comment summary: The ambient water quality criteria are not sufficiently protective of special groups at risk.

Response: In most cases, each document contains a specific section on special groups at risk. This is intended to serve as a notice to individuals or agencies using the criteria; that the derived criteria may not be sufficiently protective in all applications. If sufficient data are available, information in the section on special groups at risk could be used to modify the criteria during the standard-setting process.

Issue 9

Comment summary: Comments express concern with the failure of the criteria to specifically address possible toxicant interactions.

Response: The importance of toxicant interactions in the environment cannot be disregarded. Each document attempts to summarize the available data on such interactions. However, since the composition of toxicants is likely to vary substantially in different areas, a general approach modifying criteria based upon toxicant interactions is not available at this time. Further, the limitations of valid approaches for dealing with interactions in multi-toxicant mixtures should be recognized.

Issue 10

Comment summary: Because of the uncertainties involved in deriving criteria, the criteria should be limited to only one significant figure.

Response: The number of significant figures used to express the criteria is an admittedly arbitrary decision. The EPA recognizes the inexactitude of these numbers.

III. Comments on Non-Carcinogens

A. Criteria for Chemical Classes

Issue 1

Comment summary: Two basic approaches were taken in the documents on chemical classes when sufficient data were not available on all members in a class:

(a) Criteria were derived for individual chemicals on which sufficient data were available and no criteria were recommended for other chemicals in the class.

(b) A criteria was derived for all or some chemicals in the class based on toxicity data on one or a few members of the class.

Alternative "a" can be criticized for "allowing" contamination by "probably hazardous compounds" (reasoning by chemical analogy). Alternative "b" can be criticized for applying a general criteria to a specific compound for which data are not available.

What guidelines with justifications can be given for selecting either alternative? What other alternatives might be considered?

Response: The initial methodology did not adequately address the problems associated with deriving class criteria. The following section has been added to the methodology and serves as a useful guide in the criteria derivation process.

A chemical class is broadly defined as any group of compounds which are considered in a single risk assessment document. In criteria derivation, isomers are regarded as a chemical class rather than as a single compound. A class criteria is an estimate of risk/safety which applies to more than one member of a class, and involves varying degrees of extrapolation from available data on some members of the class to other class members on which sufficient data are not available to derive a compound-specific criteria (i.e., a criteria based on data solely on the specific chemical for which the criteria is derived).

A class criteria usually applies to each member within the class rather than to the sum of the compounds within the class. While the potential hazards of multiple toxicant exposure are not to be minimized, a criteria, by definition, most often applies to an individual compound. Exceptions may be made of complex mixtures which are produced, released, and toxicologically tested as mixtures (e.g., toxaphene and PCBs). For such exceptions, some attempt should be made to assess the effects of environmental partitioning different patterns of environmental transport and degradation on the validity of the criteria. If these effects cannot be assessed, an appropriate statement of uncertainty should accompany the criteria.

Because relatively minor structural changes within a class of compounds can have pronounced effects on their biological activities, class criteria should be avoided. Whenever sufficient toxicologic data are available on a chemical within a class, a compound specific criteria for that chemical should be developed. Nonetheless, for some chemical classes, scientific judgment may suggest a sufficient degree of similarity among chemicals within a class to justify a class criteria applicable to some or all members within a class. Such a judgment should be influenced by a perceived risk to the human population if a class criteria was not derived.

The development of a class criteria should take into consideration the following:

(a) A detailed review of the chemical and physical properties of chemicals within the group should be available. A close relationship within the class with respect to chemical activity would suggest a similar potential to reach common biological sites within tissues. Likewise, similar lipid solubilities would suggest the possibility of comparable absorption and tissue distribution.

(b) The amount of qualitative and quantitative data for chemicals within the group should be examined. Obviously adequate toxicological data on a number of compounds within a group would provide a more reasonable basis for extrapolation than minimal data on one or two chemicals within a group.

(c) Similarities in the nature of the toxicological response to chemicals in the class provides additional support for the prediction that the response to other members of the class may be similar. In contrast, where the biological response has been shown to differ markedly on a qualitative and quantitative basis for chemicals within a class, extrapolation of a criteria to other members of that class may not be appropriate.

(d) Additional support for the validity of extrapolation of a criteria to other members of a class could be provided by evidence of similar metabolic and pharmacokinetic data, if available, for some members of the class.

Based on the above considerations, it may be reasonable to divide a chemical class into various subclasses. Such divisions could be based on biological endpoints (e.g., carcinogens/non-carcinogens), potency, and/or sufficiency of data (e.g., a criteria for some members of a class but no criterion for others). While no *a priori* limits can be placed on the extent of subclassification, each must be explicitly justified by the available data.

Class criteria, if properly derived and supported, can constitute valid scientific assessments of potential risk/safety and can be used in establishing appropriate standards. Conversely, the development of a class criteria from an insufficient data base can lead to serious errors in underestimating or overestimating risk/safety and should be rigorously avoided. Although scientific judgment has a proper if not totally explicable role in the development of class criteria, such criteria will be useful and defensible only if they are based on adequate data and scientific reasoning rather than intuition. The lack of data on dissimilarity cannot be used as the basis of a class criteria. Further, the definition of sufficient data on similarities in physical, chemical, pharmacokinetic, or toxicologic properties to justify a class criteria may vary remarkably depending on the degree of superficial structural similarity and the gravity of the perceived risk. Consequently, it is imperative that the criterion derivation section of each document in which a class criterion is recommended explicitly address each of the key issues discussed above and define, as clearly as possible, the limitations of the proposed criteria and the type of data necessary to generate a compound-specific criterion.

Class criteria should be corrected when sufficient data become available to derive a compound-specific criterion that protects against the biological effect of primary concern. The availability of a good subchroni study would not result necessarily in the abandonment of a class criteria based upon potential carcinogenicity.

The inability to derive a valid class criteria does not and should not preclude regulation of a compound or group of compounds based upon concern for potential human health effects. The failure to recommend a criterion is simply a statement that the degree of concern cannot be quantified from the available data and risk assessment methodology.

Issue 2

Comment summary: To what extent can "guilt by association" be used to derive a cancer-based criteria for a compound which has been tested for carcinogenicity with negative results [e.g., bis(2-chloroisopropyl) ether in the Chloroalkyl Ethers Ambient Water Quality Criteria Document].

Response: As stated in the response to Issue 1, "guilt by association" is only an extremely limited role in criteria derivation process.

B. Organoleptic Criteria

Issue 3

Comment summary: Whenever organoleptic criteria are derived, corresponding toxicity based criteria should be derived if possible.

Response: The Agency agrees. Since organoleptic criteria are not based on toxicologic information and have no direct relationship to potential adverse human health effects, both organoleptic and toxicity based criteria are provided whenever possible.

Issue 4

Comment summary: The quality of organoleptic criteria should be assessed in terms of experimental design and statistical analysis.

Response: The revised methodology recognizes the limitations of most organoleptic data:

> With very few exceptions, the publications which report taste and odor thresholds are cryptic in their descriptions of test methodologies, number of subjects tested, concentration/response relationships, and sensory characteristics at specific concentrations above the threshold. Thus the quality of the data is usually worse than the toxicological data used for the setting of other criteria. Consequently, a clear critical evaluator of the available data on a compound's organoleptic characteristics should appear in the criteria document.

Issue 5

Comment summary: Criteria based on organoleptic properties should not be considered equal to criteria based on toxicologic effects.

Response: The revised methodology makes a clear distinction between organoleptic and toxicity based criteria. The use of the criteria in the regulatory process should reflect an appreciation of this distinction.

C. Naturally Occurring Compounds

Issue 6

Comment summary: Background levels should be defined in terms of the quality of the data base and geographical/seasonal variations.

Response: The documents summarize data on background levels of naturally occurring compounds and include information on seasonal and/or geographical variation when available.

Issue 7

Comment summary: A distinction should be made between natural and anthropogenic background.

Response: An attempt is made, with extreme difficulty, in the exposure section of the documents to differentiate between natural and anthropogenic background. However, background levels cannot be used directly to modify the criteria. By definition, criteria should not consider current levels of exposure but are estimates of safe level or incremental risk level exposures. Background levels, both natural and anthropogenic, should be considered if the criteria are used to promulgate standards.

Issue 8

Comment summary: What is the minimum data base needed to define a compound as essential?

Response: As indicated in the revised methodology, elements will be accepted as essential if the National Academy of Sciences (NAS) Food and Nutrition Board or a comparably qualified group declares them as such. Elements not yet determined to be essential, but for which supportive data on "essentiality" exists, were recommended to be reviewed by a joint EPA/NAS committee.

Issue 9

Comment summary: How can essentiality be used to modify a criteria?

Response: The following additions have been made to the revised methodology in response to this question:

In order to be useful in modifying toxicity/carcinogenicity based criteria, essentiality must be quantified either as a recommended daily allowance (RDA) or minimum daily requirement (MDR). These levels must be compared to estimated daily doses associated with the adverse effect of primary concern. The difference between the RDA or MDR and the daily doses causing a specified risk level for carcinogens or acceptable daily intake (ADI) for non-carcinogens defines the "window" of daily doses from which the criteria should be derived.

Because errors are inherent in defining both essential and maximum tolerable levels, the criteria should be derived from dose levels near the center of such a dose range. The decision to use either the MDR or RDA will be guided by the size of the window and the quality of the essentiality and toxicity estimates.

The modification of criteria by consideration of essentiality must include all routes of exposure. If water is a significant source of the MDR or RDA, the criteria must allow for attainment of essential intake. Conversely, even when essentiality may be attained from non-water sources, standard criteria derivation methods may be adjusted if the derived criterion represents a small fraction of the ADI or MDR. On a case-by-case basis, the modification in the use of the guidelines may include the use of different safety factors for non-carcinogens or other modifications which can be explicitly justified.

D. Use of NOAELs/NOELs

Issue 10

Comment summary: NOELs and related effect terms should be defined more clearly in the methodology.

Response: In the revised methodology, the following additions have been made to clarify the use of these terms:

In developing guidelines for deriving criteria based on non-carcinogenic responses, five types of response levels are considered:

NOEL—No-Observed-Effect-Level
LOEL—Lowest-Observed-Effect-Level
NOAEL—No-Observed-Adverse-Effect-Level
LOAEL—Lowest-Observed-Adverse-Effect-Level
FEL—Frank-Effect-Level

In the above terms, adverse effects are defined as any effect resulting in functional impairment and/or pathological lesions that may affect the performance of the whole organism, or which contributes to a reduced ability to respond to an additional challenge. The word lowest refers to the incidence of the effect in the tested population. It should be noted that LOELs, NOAELs, and LOAELs refer to exposure levels or dosage zones which are experimentally defined by upper and lower exposure levels. NOELs and FELs, however, are not defined at the lower and upper exposure levels, respectively.

Issue 11

Comment summary: Considerations of experimental design should be more explicitly/quantitatively considered in the criteria derivation process.

Response: The development of a rigid system for considering experimental design in criteria derivation would limit the use of scientific judgment. The section of the methodology dealing with the derivation of toxicity based criteria has been extensively revised to allow for the maximum use of scientific judgment in selecting safety factors based on both the quality of the individual study and the weight of the supporting scientific data.

E. Safety or Uncertainty Factors

Issue 12

Comment summary: Can the guidelines for applying safety factors be clarified or developed in greater detail to minimize inconsistencies without impairing scientific judgment?

Response: The following additions have been included in the methodology to allow for the use of greater judgment in the application of safety factors, while also requiring more explicit justification for the use of any uncertainty factor:

The justifications for the various safety factors can become very restrictive if they are not employed with care and judgment. This is the case especially in those instances where the data do not completely fulfill the conditions for one category of uncertainty factor and appear to be intermediate between two categories. Given the uncertainties in the entire process, it is more appropriate to set the operative uncertainty factor at some intermediate value on a logarithmic scale (e.g., 32, being halfway between 10 and 100 on a logarithmic scale). If intermediate values for uncertainty factors are more representative of actual conditions, then they are used.

In the selection of the uncertainty factor approach, "no indication of carcinogenicity" is interpreted as the absence of carcinogenic data from animal studies or human epidemiology. Short-term carcinogenicity screening tests are considered in the criteria documents, and are used in the derivation of numerical criteria and are used to rule out the uncertainty factor approach.

Because of the high degree of judgment involved in the selection of a safety factor, the criteria derivation section of each document must provide a detailed discussion and justification for both the selection of the safety factor and the data for which it is applied. This discussion should reflect a critical review of the total data base. Factors to be considered include: number of animals tested, parameters tested, species tested, quality of controls, dose levels, route, dosing schedules, etc. An effort should be made to differentiate between coherent results which form a toxicologically valid data base and data which may be spurious in nature.

Issue 13

Comment summary: What, if any, safety factor should be used when deriving criteria from a threshold limit value (TLV).

Response: The safety factor used when deriving criteria from a TLV must depend on the quality of the data base on which the TLV is based. considerations of uncertainties involved in extrapolating data from inhalation to oral exposures, and the quality of the additional supporting data.

F. Related NOAEL Issues

Issue 14

Comment summary: Can/should concentration response curves representing a "full range of effects" be used in deriving criteria?

Response: No available system for utilizing concentration response curves in representing a full range of effects for deriving criteria has been developed. If such a system does become available, it will be assessed by the Agency.

Issue 15

Comment summary: When more than one method is available to derive a non-carcinogen criteria (e.g., 2-year chronic, 90-day, TLV), can guidelines be given for selecting the most appropriate method?

Response: As indicated in the revised methodology, criteria can be based on several different types of data (e.g., studies on humans or experimental animals, subchronic or chronic exposure periods, oral or inhalation exposure routes, TLVs or similar standards). Specific guidelines for selecting a particular study or approach have not been recommended because of the many judgmental factors which are involved. As indicated in the methodology, the criteria derivation section must specifically state the reasons for selecting the approach and study used to derive the criteria.

Issue 16

Comment summary: The approach used to derive criteria for non-carcinogens may not adequately address the question of whether children are at greater risk than adults.

Response: When specific data are available on women or children as groups at increased risk, it should be stated in the document and discussed in the criteria derivation section, but should be used to modify the criteria only if sufficient specific data are available. This is a highly judgmental decision which must be made on an individual case.

Issue 17

Comment summary: Criteria based on carcinogenic effects might not be adequate to protect humans from mutagenic, teratogenic, or other toxic effects.

Response: With very few exceptions, criteria based on carcinogenicity are probably protective for other toxic effects. However, alternative criteria can be derived based on non-carcinogenic effects on a case-by-case basis if there is any doubt of the level of protection offered by the cancer based criteria.

G. Alternative Approaches to the Development of Criteria for Non-Carcinogens

Issue 18

Comment summary: Is there a reasonable way to use multiple NOEL/NOAELs to derive criteria?

Response: The revised methodology clearly indicates that all toxicity must be considered in deriving criteria and multiple NOELS/NOAELs are used. A detailed mathematical approach using multiple NOEL/NOAEL data has not been developed or accepted by the scientific community.

Issue 19

Comment summary: Is there a reasonable way to use dose/response data to derive criteria?

Response: Mathematical models for deriving non-cancer based criteria are available. However, they have not gained wide acceptance in human risk assessment. Until various models have been reviewed in greater detail, the Agency uses the current approach, based on that recommended by the National Academy of Sciences, as the most appropriate.

Issue 20

Comment summary: Confidence intervals or a range should be used in deriving criteria.

Response: A workable method for using confidence intervals in deriving non-cancer based criteria has not been developed. Given the many uncertainties involved in this process, the use of confidence intervals could be misleading in simply considering problems in statistical variation without considering problems in species to species conversion. Safety factors are an accepted procedure and are used to consider both problems in statistical variability as well as problems in species to species conversions and individual susceptibility.

H. Exposure

Issue 21

Comment summary: Should non-cancer criteria be based on all sources of exposure because they are derived from estimates of ADIs (acceptable daily intake) which define *total* daily acceptable doses for man?

Response: The methodology has been revised so that estimates of total exposure can be considered in deriving criteria. Estimates of water and fish consumption are used to derive the criteria. However, the criteria levels can be modified by considering all routes of exposure in the standard-setting process. This approach may be particularly desirable because exposure conditions will probably vary markedly on a regional basis.

Issue 22

Comment Summary: If sufficient data are not available on all sources of exposure, can any reasonable assumptions be made to factor in all sources of exposure or can/should an additional "uncertainty" factor be used?

Response: When no reasonable estimate can be made of contributions from non-fish diet and from air, it can be assumed that one-half of the exposure comes from water and fish and one-half comes from other sources. This is equivalent to using an additional safety factor of 2. It is recognized that the inability to quantify all sources of exposure adds an additional element of uncertainty to the criteria.

I. General Issues

Issue 23

Comment Summary: With the exception of recommending "good scientific judgment," can specific guidelines be given for accepting or rejecting a study or set of studies as a data base for criteria derivation?

Response: Specific guidelines cannot be given for accepting or rejecting studies. Scientific judgment must be exercised in view of the magnitude of the total evidence on the chemical or chemicals under consideration. Chronic data and appropriate exposure routes are most desirable.

Issue 24

Comment Summary: Is there a need to individualize the criteria derivation process so that the "nature of the toxic agent and its mechanism of action" can be more explicitly considered? If so, how can this be accomplished?

Response: The criteria derivation process does consider as specifically as possible the nature of the toxic agent and, when known, the mechanism of action.

Issue 25

Comment Summary: Is the Stokinger-Woodward model adequate for converting inhalation dose data to "equivalent oral doses," or should a more sophisticated approach be used?

Response: The derivation of water quality criteria from inhalation data is an admittedly tenuous process. The following guidelines have been added to the methodology:

Estimating equivalencies of dose/response relationships from one route of exposure to another introduces an additional uncertainty in the derivation of criteria. Consequently, whenever possible, ambient water quality criteria should be based on data involving oral exposures. Even with oral data, differences in dosing schedules and vehicles can be problematic. If oral data are insufficient, data from other routes of exposure may be used in deriving water quality criteria.

Inhalation data, including TLVs or similar values, are the most common alternative to oral data. Estimates of equivalent doses can be made on the basis of extensive pharmacokinetic data for oral and inhalation routes, on the basis of measurements of absorption efficiency from ingested or inhaled chemical, or on the basis of comparative excretion data when the metabolic pathways can be established to be equivalent after oral or inhalation dosing. When sufficient pharmacokinetic data are available, the use of accepted pharmacokinetic models provides the most satisfactory approach for dose conversions. However, if the pharmacokinetic data are marginal or of questionable quality, pharmacokinetic modeling is inappropriate and may result in an artifical sense of exactitude.

The Stokinger and Woodward (1958) approach, or similar models which are based on assumptions of breathing rate and absorption efficiency, can be used as alternatives when data are not sufficient to justify pharmacokinetic principles. Consequently, in using the Stokinger and Woodward or related models, the uncertainties inherent in each of the assumptions and the basis of each assumption should be clearly stated in the derivation of the criteria.

The use of data involving other routes of exposure to derive water quality criteria should not be ruled out. However, as with inhalation data, an attempt should be made to use accepted toxicologic and pharmacokinetic principles to estimate equivalent oral doses. If simplifying assumptions are used, their bases and limitations must be clearly specified.

Because of the uncertainties involved in extrapolating from one route of exposure to another and the consequent limitations that this may place on the derived criteria, the decision to disallow such extrapolation and recommend no criterion is highly judgmental and must be made on a case-by-case basis. Such a decision should balance the quantity and quality of the available data against a perceived risk to the human population if no criteria is derived.

Issue 28

Comment Summary: Can/should criteria be qualitatively or quantitatively ranked in terms of their scientific strength of validity? How could such a ranking system be developed?

Response: The Agency is presently assessing the quality of the data base supporting individual criteria. This will eventually result in the development of a ranking system of all the priority pollutants.

IV. Response to Public Comments on Methodology to Derive Water Quality Criteria

The Carcinogen Assessement Group (CAG) and the Environmental Criteria and Assessment Office-Cincinnati (ECAO-Cin.) of the U.S. Environmental Protection Agency (EPA) has reviewed in detail the public comments on EPA's methodology to derive water quality criteria for carcinogens. Since the majority of the comments are concerned with the low-dose extrapolation procedure and since they are closely related to each other, an appendix is presented which summarizes our new procedure to derive water quality criteria and the rationale for selecting the procedure and compares the new with the old procedure. Much of the criticism has been directed toward utilization of the one-hit linear model for estimation of the risk. After considerable input by a peer review of outside scientists, the multistage model developed by Kenneth Crump has been adopted in place of the one-hit model extrapolation. The Appendix describes the new multistage hit model. Further responses to the individual comments are being presented below.

A. The One-Hit Model

Issue 1

Comment summary: Several comments criticize the one-hit model as arbitrary, inappropriate, simplistic, unrealistic, inaccurate, not universally accepted, and/or overly conservative.

Response: The Agency has adopted a new procedure for deriving water quality criteria which is conceptually similar to, but operationally more systematic than the one-hit procedure used previously by the Agency. Although the criteria calculated by the new procedure are not appreciably different than those calculated by the old procedure as demonstrated in the appendix, most of the general criticisms do not apply to the new procedure.

Issue 2

Comment summary: Comments pointed out that the EPA has declined to use the one-hit model under the federal pesticide laws for heptachlor and chlordane.

Response: The commentor is correct that the one-hit model was not used in the chlordane-heptachlor suspension hearings in 1975. However, in the cancellation hearings, which were held after the formation of the Carcinogen Assessment Group and the adoption by the Agency of the Interim Cancer Assessment Guidelines and in the proposed water quality criteria, one-hit extrapolation model was used for risk estimation. In the current final water quality documents the "linearized" multistage model is used; the comparison between these two approaches in the appendix to those comments shows that the chlordane and heptachlor data have the largest upward curvature in the dose-response curve of all the carcinogens in the water quality list. For this reason the new approach reduces the risk for chlordane and heptachlor more than for the other compounds. This example shows how the new extrapolation procedure compensates for the "overly conservative" results of the one-hit approach in cases where the dose-response data is sharply concave upward at low doses.

Issue 3

Comment summary: The EPA's choice of this model because ". . . it gives greater risk estimates than other plausible models" (page 15978 of March 15, **Federal Register**) was criticized as being a policy/political/social statement rather than a scientific defense.

Response: See the appendix for reasons for selecting linear, non-threshold models.

Issue 4

Comment summary: The statement that this model was endorsed by IRLG (1979) was felt to have limited meaning because this document has not yet been reviewed and because the document is merely a reiteration of policy.

Response: The model was not selected on the endorsement of IRLG. See appendix.

Issue 5

Comment summary: In the methodology (page 15978, column 1, first full paragraph of the March 15, **Federal Register**), this model is scientifically defended as being consistent with three basic concepts in chemical carcinogenesis:

a. The Linearity of the Dose-response Curve for Mutagens—This is challenged on the following points:

The shape of the dose response curve in the low dose region cannot be determined.

Not all assay systems give linear dose-response patterns.

Some Ames tests are linear because the liver microsomes are added in a fixed amount and thus ". . . the laws of first order kinetics require a linear response to the variation in concentrations of the test substance as it is mediated by the activator."

b. Chemicals which are Mutagens are Likely to Induce Cancer—This is challenged on the basis that not all mutagens cause cancer.

c. Epidemiology Studies on Radiation, Cigarettes, and Aflatoxin show a Linear Dose-response Pattern—This is challenged on the following points:

Radiation carcinogenicity cannot be applied to chemical carcinogenicity because they act by different mechanisms.

Not all radiation dose-response data is linear.

Smoking data are compounded by difficulties with cocarcinogens and other exposures.

Aflatoxin data rely purely on estimated exposures.

Response: (a) The commentor points out that even in mutagenesis test systems there is a level of mutagenic response that is too small to be detected and that below this level the shape of the dose-response curve cannot be measured. While this is true, the Agency's point is that the mutagenesis data available are fundamentally consistent with a linear no-threshold mechanism of action. Another commentor has misinterpreted the mutagenesis dose-response data. As presented by the original authors, the data show some residual mutagenic activity at zero dose. This is interpreted erroneously as being a threshold below which no response occurs. Another commentor supports the Agency's contention that the mutagenesis dose-response relationship is linear by giving a possible explanation for the linearity.

(b) The fact that chemicals which are mutagenic are "likely" to induce cancers does not imply that "all" mutagens cause cancer. Furthermore, those mutagens which were not shown experimentally to be carcinogenic could not be accepted unequivocally as non-carcinogenic because of the uncertainty in the study outcome.

(c) Both chemicals and radiation cause DNA damage and subsequent interference with the normal functioning of DNA, although the mechanisms for causing this damage are different for radiation and chemicals.

Issue 6

Comment summary: Several comments stated that the possibility of thresholds for at least some chemical carcinogens is not unreasonable, should be addressed in greater detail and/or cannot be resolved at this time. The possibility of assuming a threshold was recommended for the following compounds: chloroform, PCBs, acrylonitrile, hexachlorocyclohexane, chlorinated benzenes, and chlorinated ethanes.

Response: Currently there is no satisfactory method for estimating the low-dose carcinogenic risk to "epigenetic" chemicals. Until the mechanisms for such action are understood on a case-by-case basis to the point of being able to justify a specific extrapolation procedure, the linear, no-threshold concept will be assumed to be valid. The "linearized" multistage approach now used result in lower risks than the older "one-hit" approach for compounds having a sharp upward curvature.

For the specific chemicals referred to in Issue 6, no evidence was presented in support of a carcinogenic threshold dose except for chloroform. Commentors state that chloroform induces an increased rate of cell proliferation, which they implicitly equate with carcinogenesis, at high doses because of a cytotoxic response which is unrelated to direct DNA interaction and which therefore is not expected to occur at low doses. Three pieces of evidence are cited in support of that position: (a) chloroform is not mutagenic in the Ames tests; (b) at doses below 15 mg/kg/day, mice show no excess rate of DNA synthesis in kidney and liver tissue. This excess is expected for a cytotoxic response leading to cell proliferation; (c) Roe et al (1979) on the basis of responses in four strains of mice, has established a no-carcinogenic effect level of 17 mg/kg/day, whereas the positive NCI experiment used by EPA for the water criterion was carried out at 200–400 mg/kg/day.

Before the existence of a threshold for chloroform can be established several issues need to be resolved: (a) are the no-effect levels in the DNA synthesis studies and in Roe's observations real phenomona or only artifacts occurring simply because the limit of detection in these studies was being reached? (b) The relation between the cellular proliferation, which is alleged to be manifested by increased DNA synthesis, and carcinogenesis is unclear, since in the mouse strains used by NCI kidney tumors do not occur and liver tumors do, whereas in the experiments cited by a commentor both liver and kidney exhibit DNA synthesis.

Issue 7

Comment summary: A distinction should be made between genetoxic and epigenetic carcinogens based on mutagenicity data. These comments imply that a threshold model would be more appropriate for epigenetic carcinogens.

Response. While it is true that most carcinogens do interact with DNA, there are some compounds, such as phorbol esters in mouse skin studies and phenobarbital in rat liver, which are incomplete carcinogens by themselves, but require another substance to initiate or promote their action. In these studies the effects are unrelated to DNA interactions and apparently involve important recovery processes. This newly-developing field is not yet well enough understood to justify the use of a particular dose-response extrapolation model.

Issue 8

Comment summary: Another group of comments vigorously opposed the non-threshold assumption used in the one-hit model. Criticism of the non-threshold assumption were most extensively articulated by commentors which contended that the non-threshold assumption is:

Contrary to experience and logic, to what is known of biological systems, and to existing scientific data and is a product of the desire to obtain a simple and easy-to-use method for criteria derivation.

A related comment contended that thresholds are apparent for mutagens and therefore—given the presumed relationship of carcinogenicity to mutagenicity—thresholds should be postulated for carcinogens.

Response: Commentors state that the linear non-threshold model is: (a) contrary to experience and logic; (b) contrary to what is known about biological systems; (c) contrary to existing scientific data and (d) an approach based on faith that could not be disproved by any facts.

(a) The linear non-threshold model does not imply, as suggested by a commentor, either that (a) cancer is inevitable in the general public or in heavily exposed industrial workers or that (b) all substances are carcinogenic. It simply states that the probability of a person getting cancer is proportional to the amount of carcinogen to which he is exposed.

(b) First order reaction processes are common in biological systems especially in mutagenesis.

(c) Dr. Bingham's article did not advocate a sigmoid dose-response curve in preference to a linear curve, as stated by the commentor. She stated that several environmental factors can alter the dose-response relationship, and would thereby change the curve whichever way it was described. In fact, her main point was that "until we understand more about the primary carcinogenic insult and its progression, predicting or estimating thresholds is risky." The Agency agrees with this conclusion.

(d) The Agency agrees that it would be extremely difficult to use negative epidemiology data as proof that a carcinogenic threshold exists for a compound having positive animal results.

Issue 9

Comment summary: Several comments critized the one-hit model because it does not fit some experimental data as well as other models. This was illustrated for heptachlor, chlordane, and aflatoxin and chlorinated ethanes.

Response: The new extrapolation method overcomes the difficulty in fitting the model to the data because the multistage model has enough flexibility to fit any montonically increasing dose-response relationship. See also the response to Issue 16.

Issue 10

Comment summary: The application of the model was also criticized because it disregards data at all but one dose level and fails to consider the results of other experiments.

Response: The new procedure does not have these shortcomings. See appendix.

Issue 11

Comment summary: The highest potency factor to the exclusion of all other data should not be used in generating criteria because this process does not involve maximum-likely risk estimates.

Response: In judging which of several animal studies to use as the basis for the quantitative risk estimate, the quality of each study is considered as well as the numerical slope factor. As explained in the preamble, an experiment with a small number of animals is rejected in favor of a larger experiment if the two have a similar dose-response relationship. A similar rejection is also made if an experiment is judged to be unreliable for other reasons. Because of the strain, species, and sex differences, it is considered improper to calculate an average response across all animal species and designate this average as the carcinogenic potency for animals in general.

Issue 12

Comment summary: ". . . no experiment, however large and well run, could ever reduce these estimates (criteria)."

Response: In judging which of several animal studies to use as the basis for the quantitative risk estimate, the quality of each study is considered as well as the numerical slope factor. As explained in the preamble, an experiment with a small number of animals is rejected in favor of a larger experiment if the two have a similar dose-response relationship. A similar rejection is also made if an experiment is judged to be unreliable for other reasons. Because of the strain, species, and sex differences it is considered improper to calculate an average response across all animal species and designate this average as the carcinogenicity potency for animals in general.

Issue 13

Comment summary: The EPA method is insensitive to reproducibility of the results, results at lower doses, and the number of animals per dose group.

Response: In judging which of several animal studies to use as the basis for the quantitative risk estimate, the quality of each study is considered as well as the numerical slope factor. As explained in the preamble, an experiment with a small number of animals is rejected in favor of a larger experiment if the two have a similar dose-response relationship. A similar rejection is also made if an experiment is judged to be unreliable for other reasons. Because of the strain, species, and sex differences, it is considered improper to calculate an average response across all animal species and designate this average as the carcinogenic potency for animals in general.

Issue 14

Comment summary: Several examples are given of the failure of the one-hit model to predict cancer rates in humans based on epidemiologic studies:

Analyses of data on: chloroform, carbon tetrachloride, tetracholoroethylene, aflatoxin, chlordane, arsenic, and beryllium.

In a summary of analyses of DDT, dieldrin, and aflatoxin, it is indicated that the one-hit model predicts an incidence of 153,000 liver cancers per year but that the observed response rate from all chemicals in only 3,000 to 4,000 per year. A similar analysis is made of pollution exposure-cancer rates in the Sacramento River area.

Response: For chloroform, carbon, tetrachloride, and tetrachloroethylene the analysis assumed that all of the workers were exposed at the TLV levels for their entire lifetime. In reality most workers are not exposed continuously to levels as high as the TLV and most work for only a few years at these jobs. This procedure overestimates the average lifetime exposure by at least a factor of 10 and the risk estimates for the workers are too high because of exposure assumptions used by the commentor rather than solely because of an overestimated slope factor.

For aflatoxin the commentor showed that the multistage model fits the observed human data more closely than the one-hit model. Therefore, that analysis partially justifies the revised procedure, although this compound is not on the water quality list.

The criterion for arsenic was based on human data, which was linear with dose. However, none of the negative epidemiology studies in areas with high drinking water levels of arsenic was inconsistent with the model developed on the basis of the Taiwan skin cancer data.

Commentors estimated that the annual number of cancer cases caused by beryllium intake is about 14,000. They gave no reason why this number is considered excessive considering that 400,000 cases per year are observed from all causes.

Issue 15

Comment summary: Based on the above types of analyses, several comments recommended that epidemiologic data be used to test and/or modify risk estimates.

Response: The Agency agrees that good epidemiological data should be used to estimate or modify risk estimates. The Agency always preferred using epidemiological data to the animal data in deriving water quality criteria.

Issue 16

Comment summary: Some comments suggest that selection of a particular model should be left open and subject to the nature of the experimental data and epidemiologic or metabolic information.

Response: The Agency does not agree that the selection of a particular model should be left open and subject to the nature of the experimental data for the following reasons. When behavior of the dose-response curve at low doses is not sufficiently understood, it is more appropriate to predetermine the low-dose extrapolation model. Considering the fact that all the mathematically analytic functions such as most of the parametric dose-response curves could be approximated by polynomials which are dominated by the higher order terms in the high-dose range, whereas they are vanishingly small in the low-dose regions it is not surprising to see that different dose-response models could fit well a set of high dose data while their low-dose extrapolations differ drastically. Therefore, the selection of the extrapolation model should be based on knowledge of carcinogenic mechanisms (even though limited and debatable) rather than being determined solely by the high dose behavior of the dose-response curve.

Issue 17

Comment summary: Other comments suggested that the one-hit model should be used only in the absence of data suggesting that other models give a better fit.

Response: See response to Issue 16 and the appendix.

Issue 18

Comment summary: Two comments recommend that several models used for analysis along with appropriate confidence intervals would more objectively reflect the state of scientific knowledge.

Response: The inclusion of several arbitrary models in order to get a range of risk estimates would add no additional scientific information while at the same time would create confusion and thereby undermine the utility of risk estimates. The model chosen by the Agency is regarded as giving a plausible upper limit to the risk.

Issue 19

Comment summary: A general discussion of alternative models is given in some of the comments. Specific models recommended include: Logistic; Probit; Multi-hit; Mantel and Bryan; Weibull; and Pharmacokinetic.

Response: The inclusion of several arbitery models in order to get a range of risk estimates would add no additional scientific information while at the same time would create confusion and thereby undermine the utility of risk estimates. The model chosen by the Agency is regarded as giving a plausible upper limit to the risk.

B. Use of Confidence Intervals

Issue 20

Comment summary: Confidence intervals or a range should be used in deriving criteria.

Response: The Agency feels that the statistical confidence intervals should not be used to express the range of uncertainty of the criteria because this range does not include major uncertainties which are not quantifiable, such as species differences in metabolism, diet, target organ specificity, and other biological variables.

C. Species Conversion Factor $(W_H - W_A)$⅓

Issue 21

Comment summary: Comments suggested that this factor may not be appropriate for carcinogens because: (a) DNA repair rates appear to be inversely proportional to body weight, and (b) mixed-function oxidase activity, which may activate carcinogens, is higher in rodents than in man. Examples were given indicating that man is less sensitive than experimental mammals to chloroform, aflatoxin, and vinyl chloride.

Response: Although some commentors discussed reasons why the species conversion factor, (70/W)⅓, may not be appropriate for particular compounds, no suggestion was made for an alternative method which would be valid in general. Commentors suggested that mixed-function oxidase activity is lower in humans than in rodents and that humans metabolize chloroform less completely than animals, but facts like these, even if quantified would have uncertain implications to carcinogenic potency in general because increased metabolic activity could both enhance carcinogenic potency by "inactivating" the agent.

The fraction of a compound (e.g. chloroform) unmetabolized may have no relation at all to the amount of active metablite formed. In the general method, the cube root factor is intended to account only for the body size difference between animal species as it relates to the availability of the chemical to the body tissues. Any specific knowledge available on metabolism differences would have to be incorporated as an additional factor if it could be directly related to cancer incidence. In general, mixed-function oxidase activity has no clear relation to cancer occurrence, therefore, cannot be included in the general approach.

The best apporach for checking the validity of the species conversion factor is to correlate carcinogenic potency of agents in animals with that in humans where suitable information is available. This was done in a preliminary fashion by Messelson (quoted by one commentor) and is currently being investigated by the Agency.

Issue 22

Comment summary: Data on comparative metabolism should be used, whenever possible, to modify the risk estimate.

Response: See Issue 21 for response to chloroform metabolism issue. The Agency acknowledges that species differences in metabolism should be considered in all cases where the data can be interpreted as being relevant to carcinogenicity. In the methodology description the appropriate place to incorporate this information is in the factor r, called the absorption fraction.

D. Time-To-Tumor Data

Issue 23

Comment summary: Some comments stated that the EPA's modification of the one-hit model does not consider the time-to-tumor concept.

Response: The time-to-tumor concept was incorporated in the one-hit procedure by using the model $P = 1 - \exp(-bd\,t^3)$ where t is the average fraction of a lifetime the tumor was observed and is also incorported into the current approach. If sufficiently well defined time-to-tumor data are available, a more refined model would be used.

Issue 24

Comment summary: Other comments contended that, because of the relationship between dose and latency, even potential carcinogens will not induce tumors in a normal lifespan. Examples were given for beryllium and arsenic.

Response: The arguments given in this comment do not invalidate the criteria which are associated with a lifetime risk of 10^{-6}. The arguments given in the comment proceed as follows:

Let F(d,t) be the probability of cancer by age t at exposure d. F is a monotonic increasing function of both variables t and d. Let d_e be the exposure associated with the lifetime risk of cancer 10^{-6} obtained by solving for d from the equation $F(d,t) = 10^{-6}$ and $t = 70$ which is taken as the average lifespan. Based on the arsenic risk assessment by the CAG the comment argues that at exposure $d_e = 0.002$ µg/liter (where d_e is associated with a lifetime risk of 10^{-6}), the median age would be 2,636 years before cancer can occur, where 2,636 is obtained by solving for t from the equation $F(d_e,t) = 0.5$.

Therefore, if the water concentration is 0.002 µg/l the risk at 70 years is 10^{-6} and at 2,636 years it would be 0.5. These are not inconsistent statements, as implied by the commentor.

Issue 25

Comment summary: One comment suggested that studies in which t is less than 0.75 may not be useful because of insufficient time for tumor development.

Response: The t^3 factor is necessary when the carcinogenic response is so strong that the animals die prematurely of tumors. This is true regardless of whether the median time of death from tumors is greater or less than three-fourths of their natural lifespan.

Issue 26

Comment summary: Another comment raised questions about the experimental difficulties of Druckery's work in precisely determining time-to-tumor development and the need to correct time-to-tumor data for the degree of malignacy of the tumor.

Response: The time-to-tumor data is used only when there was an early terminal sacrifice. In this case the full spectrum of tumor development is observable histologically and the difficulty of observing the precise time of tumor development is not encountered.

E. Mutagenicity Data

Issue 27

Comment summary: Mutagenicity data should be given greater weight to determine potential carcinogenicity especially when mammalian bioassays or epidemiology data are lacking.

Response: See response to Issue 28.

Issue 28

Comment summary: The commentor describes a method for using results from short-term tests, such as the Ames test or the hamster embryo *in vitro* transformation test, to perform quantitative carcinogenicity risk assessment.

Response: The Agency does not regard results from short term mutagenicity tests, even those from a test battery using several organisms, equivalent to chronic whole animal bioassays for carcinogenicity because of the inherent differences between the test systems utilized (i.e., bacteria and cell cultures versus whole animals, in which all the metabolic, distribution and excretion systems of the body are intact). Until correlations between the carcinogenicity and mutagenicity of agents become better understood and more widely accepted, and until the data base is more extensive, the Agency is not justified in making quantitative assessments of carcinogenicity based solely on mutagenicity test results and structure activity relationships. In decisions regarding the carcinogenicity of agents, the Agency currently uses short term bioassay tests only to support equivocal findings of long term animal bioassays and human studies.

F. Epidemiology Data

Issue 29

Comment summary: Epidemiology data on societies other than the U.S.A. should not be used because of "dissimilar and possibly controlling variables."

Response: The CAG feels that epidemiological data on societies other than the U.S.A. population can be used as long as care is taken in interpreting and using the data.

Issue 30

Comment summary: Citing criteria for arsenic and cadmium, the comment states that: "Valid epidemiological studies exploring a cause-and-effect relationship between exposure to a substance and disease must avoid a number of flaws: bias, confounding factors, and the confusion of chance associations with casual relationships. The epidemiological studies used by the Agency in criterion formulation fail to avoid these flaws."

Response: While nearly every epidemiologic study contains flaws in the scientific sense, a regulatory agency must interpret all data available, making judgments as to whether the studies were too flawed for proper conclusions. In determining the carcinogenicity of a substance, the Agency is sensitive to the need to find human populations who have been exposed to other agents also. The appearance of rare types of cancers and/or a dose-response trend, however, often provide(s) very positive evidence of carcinogenicity. Such is the case with the Taiwan drinking water survey. Here, where artesian well water with a high concentration of arsenic has been used for more than 60 years, a high correlation between amount of arsenic and skin cancer was found. In addition, the pre-cancerous skin conditions were pathonomic of arsenic exposure, so that there was little chance that the cancers were caused by another agent. Furthermore, the skin cancer is of a rare form that was virtually unknown in parts of Taiwan where the drinking water arsenic content was small. In addition, a positive association between arsenic level in drinking water and the prevalence of skin cancer has been reported in at least three other areas in the world.

Cadmium is an unusual situation in that five independent populations showed an excess of prostate cancer. Even though each study is inconclusive by itself for the reasons cited, a chance occurrence of this finding is exceedingly unlikely.

While the effect of many possible confounding factors, especially concomitant exposure to unknown chemicals, cannot be accurately determined, the Agency has the responsibility of estimating criteria levels with the best information available.

G. Qualitative Determination of Carcinogenicity

Issue 31

Comment summary: The commentor states that: "A substance is currently considered to be carcinogenic if it produces a statistically significantly higher than normal incidence of tumors in treated animals in a single test. Such a result is inconclusive, because of the problems of false positives.

Response: In establishing a false negative rate of $P<0.05$ the commentors correctly point out that the false positive rate is rather high. However, the careful review of other information about the compound reduces the effective false positive rate.

Issue 32

Comment summary: The decision to label a compound "a suspect human carcinogen and therefore a potential human carcinogen" based on tumorigenicity in experimental mammals has not been validated.

Several comments from the initial publication of the methodology made a similer criticism.

Response: Among public health authorities it is widely accepted that the positive results in chronic animal bioassays indicate that the agent poses a potential risk for human carcinogenicity. This attitude is thoroughly summarized in the IRLG report (Jour. Natl. Cancer Inst. 63: 241, 1979). In addition, a review by Tomatis (Am. Rev. Pharmacol. Toxicol. 79: 511, 1979) emphasized the value of rodent bioassays in predicting human carcinogenic risk.

Since 1976, (41 FR 21402) the EPA has been following the same regulatory philosophy in evaluating carcinogenic hazards. Therefore, contrary to the comments, the EPA has not been acting unilaterally without adequate public notice.

Issue 33

Comment summary: The commentor quotes a WHO publication: "It would be unwise to classify a substance as a carcinogen solely on the basis of a species or strain-specific increased incidence of tumors of a kind that occur spontaneously with high frequency."

Response: The CAG agrees partially with the comment. However, even if the spontaneous incidence is high, a statistically significant enhancement of the tumor incidence is of concern, and other evidence for the compound should be evaluated for consistency with that finding.

Issue 34

Comment summary: Citing PAH as an example, commentors state that the documents have been inconsistent in qualitative determinations of carcinogenicity.

Response: In cases such as PAH where one criterion has to be set for an entire class of compounds, the Agency does not state that each chemical in the class is a carcinogen, as implied by the commentor. Therefore, the PAH example cited by the commentor does not show that the Agency is inconsistent in classifying compounds as carcinogenic.

The intended interpretation of the criterion is that the risk is less than 10^{-5} whenever the total concentration of all PAH compounds in water is less than the criterion. In a hypothetical case where all of the PAH compounds in a sample are non-carcinogenic, the criterion would be too strict; however, this situation seldom occurs. In most cases where PAH is detected, a mixture of compounds occurs and in calculating the criterion the assumption is made that all components have the same carcinogenic potency as benzo-α-pyrene.

Issue 35

Comment summary: Bis(2-chloroisopropyl)ether (BCIE) yielded negative results in an NCI bioassay. Nonetheless, the cancer based criteria based on an upper limit of the true response rate was calculated because of the structural similarity of BCIE to other carcinogenic chloroalkyl ethers. The commentor states that this is inappropriate.

Response: As a response to the public comments, the Agency has changed its interpretation of data on bis(2-chloroisopropyl)ether (BCIE). It is no longer considered to be carcinogenic and, therefore, a criterion based on carcinogenic data is not calculated.

Issue 36

Comment summary: Using the criteria for chromium and asbestos as examples, the commentors state that data on inhalation carcinogenicity should not be used to derive criteria if oral carcinogenicity tests are negative.

Response: The criteria for chromium (Cr) was derived on a marginally significant digestive cancer incidence which occurred from inhalation exposure (Enterline epidemiology study). The digestive system cancer is assumed to be caused by chromium (Cr) which is removed from the respiratory system by mucociliary action and then swallowed. This is comparable to exposure to chromium in drinking water. Cr (VI) has not been adequately tested for its carcinogenic potential in animals; therefore, further studies to assess the carcinogenicity of Cr (VI) by the oral route would be desirable and necessary before it is concluded that oral tests are negative.

Asbestos has been shown to cause peritoneal mesothelioma in humans and is also associated with a significant increase in human gastrointestinal cancers. These are caused by inhaled asbestos. Since up to 99 percent of the inhaled asbestos is eventually swallowed, the Agency feels that asbestos-contaminated water could cause the same type of gastrointestinal cancers as inhaled asbestos.

H. Joint Action/Cocarcinogenicity

Issue 37

Comment summary: The commentors emphasized the potential importance of cocarcinogenicity and possible synergistic effects among carcinogens.

Response: The potential importance of cocarcinogenicity and possible synergistic effects among carcinogens has not been addressed by the CAG in deriving water quality criteria, since sufficient data is not available at this time to make decisive judgments related to these issues.

I. Site Specific vs. Total Tumors

Issue 38

Comment summary: No public comments specifically addressed this issue. However, the methodology committee should discuss the appropriateness of using data on total tumors for quantitative risk assessment.

Response: Since chemicals generally exert their carcinogenic effects at specific organ sites, the incidence of tumors at the responding sites is the most relevant information to consider in making either qualitative or quantitative evaluations of hazard. The instances where the tumor incidence at all sites combined is elevated, but no one site or group of sites is significantly increased, are regarded as weak evidence of carcinogenicity.

J. General Issues

Issue 39

Comment summary: Single unverified bioassays should not be used for establishing criteria for dichlorobenzene.

Response: The comment does not refer to carcinogenicity data since no carcinogenic information was available on dichlorobenzene.

Issue 40

Comment summary: Some comments expressed concern with the types of studies used to derive criteria. Another comment implies that only data published in referenced journals should be used. Two commentors recommend that explicit reasons be developed for accepting or rejecting studies.

Response: The evaluations of bioassay studies for carcinogenicity by the CAG is sufficiently detailed to be equivalent to that given in peer reviewed journals.

Issue 41

Comment summary: The EPA "has used animal studies without adequately considering the nature of the toxic agent and its mechanism of action, the conditions of exposure, or the physiological characteristics of the test organism."

Response: The Agency routinely considers all of the available toxicological data cited by the commentor and agrees that these factors are important.

Issue 42

Comment summary: Several comments questioned the appropriateness of using studies from one route of exposure—particularly inhalation—to establish criteria for ingestion.

Response: If a given chemical induced a carcinogenic effect by inhalation at a distant site, it is likely that the compound could also produce a carcinogenic effect by other routes of administration. Therefore, the Agency considers it appropriate to use inhalation data to derive criteria for ingestion, recognizing the difficulty of determining the dose.

Issue 43

Comment summary: Treating all of the proposed criteria as if they were based upon equally valid data is not scientifically sound. EPA must make explicit the nature, extent, and quality of the data utilized to estimate criteria.

Response: The Agency has indicated which criteria should be regarded as marginal based on the nature of the available data.

Issue 44

Comment summary: Criteria based on carcinogenic effects might not be adquate to protect humans from mutagenic, teratogenic, or other toxic effects.

Response: The U.S. EPA Office of Health and Environmental Assessment is currently developing guidelines for estimating the human risk to substances producing mutagenic, teratogenic, and reproductive effects.

Appendix

I. An Improved Procedure for Deriving Water Quality Criteria

As discussed in the methodology document (1981)[*] the Carcinogen Assessment Group (CAG) has adopted a new prodedure which is more systematic than the one-hit procedure used previously by the CAG for calculating the water quality criteria. The model selected for the low dose extrapolation is given by

$P(d) = 1 - \exp[-(q_0 + q_1 d + \ldots + q_k d^k)]$

At low doses, the upper confidence limit for the extra risk

$$A(d) = \frac{P(d) - P(o)}{1 - P(o)}$$

has the form

$$A_u(d) = 1 - \exp(-q_1^* d) \simeq q_1^* d.$$

That is, the risk $A_u(d)$ is always linerarly related to d at low doses. The constant q_1^* corresponding to the 95 percent upper confidence limit for A(d) is taken as the carcinogenic potency for calculating the water quality criteria.

Instead of extrapolating with the one-hit model based on the lowest dose group showing statistically significant response as previously used by the CAG, the new procedure is employed because of the following reasons: (1) the procedure is more systematic; (2) it invokes fewer arbitrary assumptions; (3) the assumption of the low-dose linearity is not essential in the use of the model, and, (4) it incorporates data from all of the dose groups which are consistent with the multistage model. At the same time, it is conceptually consistent with the linear non-threshold concept on which the one-hit procedure was based.

The Agency recognizes that there is no really solid scientific basis for any mathematical extrapolation model which relates carcinogen exposure to cancer risks at the extremely low level of concentration that must be dealt with in evaluating the environmental hazards. For practical reasons such low levels of risk cannot be measured directly either by animal experiments or by epidemiologic studies. We must, therefore, depend on our current understanding of the mechanisms of carcinogenesis for guidance as to which risk model to use. At the present time the dominat view of the carcinogenic process involves the concept that most agents that cause cancer also cause irreversible damage to DNA. This position is reflected by the fact that a very large proportion of agents that cause cancer are also mutagenic.

There is reason to expect the quantal type of biological response that is characteristic of mutagenesis is associated with a linear non-threshold dose-response relationship. Indeed, there is substantial evidence from mutagenesis studies with both ionizing radiation and a wide variety of chemicals that this type of dose-response model is the appropriate one to use. This is particularly true at the lower end of the dose-response curve; at higher doses, there can be an upward curvature probably reflecting non-threshold dose-response relationships. The linear non-threshold model is also consistent with the relatively few epidemiological studies of cancer responses to specific agents that contain enough information to make the evaluation possible (e.g., radiation induced leukemia, breast and thyroid cancer, and skin cancer induced by arsenic in drinking water, and liver cancer, induced by aflatoxin in the diet). There is also some evidence from animal experiments that is consistent with the linear non-threshold model (e.g., liver tumors induced in mice by 2-acetylaminofluorene in the large scale ED_{01} study at the National Center of Toxicological Research and the initiation stage of the two-stage carcinogenesis model in the rat liver and the mouse skin).

Because it has the best, albeit limited, scientific basis of any of the current mathematical extrapolation models, the linear non-threshold model has been adopted as the primary basis for risk extrapolation to low levels of the dose-relationship. The risk estimates made with this model should be regarded as conservative, representing the most plausible upper limit for risk, i.e., the true risk is not likely to be higher than the estimate but it could be smaller.

II. Comparison of the new Procedure with the Old (One-Hit) Procedure

The Agency had previously calculated the slope b based on the one-hit model P = 1-exp-bd, using only the data from the lowest dose group where the incidence rate is statistically significantly different from the control group (see Federal Register, Part V, Thursday, March 15, 1979). The point estimate b was taken as the carcinogenic potency for the compound. Unlike the new procedure, the upper confidence limit was not used because the CAG recognized that the one-hit model is usually conservative at low doses and thus the point estimate b of the slope was considered as an upper limit of the true carcinogenic potency. This ad hoc approach was used because it is simple and easy to understand.

Since b was considered an upper limit in an ad hoc sense, it would be interesting to compare the new procedure with the one-hit procedure by calculating the ratio of two carcinogenic potencies b/q_1^* for 21 chemical compounds in the Proposed Water Quality Criteria Documents which have data from at least three dose groups. Except for chlordane and heptachlor the new procedure agrees with the one-hit procedure within a factor of 2. When the one-hit procedure is modified (Table 1) the two procedures become comparable. Therefore, the old procedure could be used as a simple and quick way of estimating the carcinogenic potency.

Table 1.—*Ratio of Carcinogenic Potencies*

Chemicals	b/q_1^*	
Chlordane	3.33	[1] (0.83)
Heptachlor	2.50	[1] (0.78)
Carbon tetrachloride	1.47	
PAH	1.36	
HCB	1.19	
1,2-Dichloroethane	1.18	
Acrylonitrile	1.10	
Hexachloroethane	1.06	
2,4,6-Trichlorophenol	.96	
Trichloroethylene	.95	
Hexachlorobutadiene	.88	
Chloroform	.81	
1,1,2,2-Tetrachloroethane	.75	
1,1,2-Trichloroethane	.70	
TCDD	.66	
2,4-Dinitrotoluene	.63	
Vinyl chloride	.63	
Toxaphene	.56	
Aldrin	.56	
Bis(chloromethyl)ether	.53	
Hydrazobenzene	.50	

[1] The parenthesized value is the ratio b/q_1^* when b is calculated based on the 95 percent upper confidence limit (one sided) for the incidence rate in the next lowest dose group, instead of using the incidence rate of the lowest dose group showing statistically significantly different from the control group. This modification was recommended when the tumorigenic responses exhibit sharp upward curvature with low doses.

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[*] Carcinogen Assessment Group's Procedure for Calculating Water Quality Criteria, Updated 1981.

July 12, 1983

AMERICAN INSURANCE ASSOCIATION:

PRELIMINARY PAPER ON PROPOSED HAZARDOUS SUBSTANCE VICTIM COMPENSATION LEGISLATION

I. INTRODUCTION

The American Insurance Association is a trade association composed of more than 160 publicly owned insurance companies providing property and casualty coverages. The Association's members provide the majority of general commercial liability insurance in the United States. It is likely that the members of the American Insurance Association will be asked to provide insurance for those subject to liability for hazardous substance compensation. A market for such insurance can develop only if the compensation liabilities to be insured are both reasonably predictable and affordable. The members of the American Insurance Association, and other insurers, have responded in the past to the emerging environmental liabilities; new coverages have been developed and competitively marketed. Thus, the American Insurance Association has an interest in seeing that the liability system as it develops is reasonable, equitable and insurable.

This Paper expresses the position of the American Insurance Association in response to state and federal legislative proposals concerning "hazardous substance" liability claims. Many of these bills are based in part on recommendations made by a majority of the

"Superfund Section 301(e) Study Group" (the "Study Group majority") in a Report dated September 1982 (the "Report") entitled Injuries and Damages from Hazardous Wastes -- Analysis and Improvements of Legal Remedies, submitted to the United States Congress pursuant to Section 301(e) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 ("CERCLA"), P.L. 96-510.

II. EXECUTIVE SUMMARY

The law of torts, as implemented through our nation's courts, is the primary medium for resolving disputes as to compensation for claimants alleging injury from exposure to hazardous substances. The tort liability system as it has evolved over the centuries -- and as it continues to evolve today -- is designed to serve three functions: justice, compensation, and incentives for proper conduct. Ideally, a plaintiff will receive compensation in the courts only if it is just to require the defendant to pay, and if requiring such payments overall will tend to provide proper incentives for prudent behavior and to minimize the incidence and seriousness of such damage in the future.

Under traditional principles, a defendant whose fault or abnormally dangerous activity is determined to have caused an injury must compensate an injured claimant; such a determination is consistent with our notions of justice and fair play, and also serves as an incentive to that defendant and others, to do better in the future. Moreover, an injured plaintiff may not obtain compensation from parties randomly selected out of the general population who have not caused the injuries at issue and who have not engaged in any improper or abnormally dangerous activity; compensation is not provided in such a case because it would not be consistent with our notion of justice, would not penalize the parties actually at fault or encourage them and others to be more careful in the future.

The Study Group majority, and the sponsors of various bills subsequently introduced, have advocated radical departures from the present common law tort system to ease plaintiffs' burden of obtaining damages for personal injury or property loss allegedly resulting from exposure to certain hazardous substances. These proposals include devices such as:

1. A presumption that hazardous substances caused a plaintiff's injuries or disease where plaintiff cannot show that this is the case.

2. Absolute liability, without any regard to the presence or absence of fault on the part of the defendant.
3. The imposition of liability on a particular defendant far in excess of the harm that defendant caused, through the use of presumptions that a particular defendant's substance caused harm when no proof of that fact is available, and the use of joint and several liability.
4. The use of administrative remedies partially or completely funded by a tax on certain industries, which falls on good and bad actors equally and removes any incentives for proper conduct.
5. The imposition of retroactive liability.
6. The use of an administrative "compensation" scheme as an add-on to potentially unlimited tort liability, thus providing both widespread recoveries under relaxed standards of proof, and no limitation on the amount of recoveries that are available.

The American Insurance Association opposes such radical proposals because they detract from the combined ideals of justice, compensation and incentives for proper conduct that are the basis of the present tort system, and because it has not been demonstrated that such legislation is necessary or will improve the present system.

The proposals made thus far have the potential of providing awards for every case of cancer, tumor, genetic damage, and other diseases of the type claimed to result from "exposure" to hazardous substances. The cost of the proposed legislation would run into tens of billions of dollars per year for American industries -- a cost far in excess of any harm that has been demonstrated to be caused by hazardous substances, as opposed to the many other factors that affect our everyday lives and health.

Enactment of the specific proposals recommended by the Study Group majority and others, in addition to providing costly and unfair presumptions of causation not warranted by scientific or other evidence, would remove all incentives for industry to use due care in handling hazardous substances, since everyone would be penalized (through absolute, joint and several liability and through taxes) without any regard to fault or lack of fault. Thus the goal of preventing future harm would be abandoned in favor of an overbroad system of after-the-fact compensation. Moreover, the use of an add-on system of administrative recovery in addition to a system of unlimited courtroom recovery would constitute perhaps the most expensive and cumbersome compensation system that could possibly be devised.

The American Insurance Association believes that the proposals, which combine staggering costs, unlimited and unpredictable liability, and the abandonment of the traditional requirements of fault and causation, would be uninsurable, unaffordable, unjust and unwise.

III. THE STUDY GROUP MAJORITY'S RECOMMENDATIONS

Because most of the proposed legislation suffers from the same generic problems that are inherent in the Study Group majority's Report, a brief summary of the Report is included here.

The Study Group was comprised of twelve lawyers, three each appointed by the Presidents of the American Bar Association, American Law Institute, American Trial Lawyers Association, and the National Association of (State) Attorneys General. No medical, scientific, epidemiological, economics, industrial or underwriting experts were included in the Study Group.

The Study Group majority recommended that a two-tier recovery system be established. Tier One would provide an administrative remedy administered by the states but governed by federal law. (Report at 206).* Tier Two -- which would also be available even if a claimant first received a Tier One administrative award --

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* Citations in this form refer to the pagination of Volume One of the Report as printed by the Government Printing Office, Serial No. 97-12. Volume Two contains lettered appendices to the Report.

would consist of the judicial tort-claim process, modified to make recovery of damages (including damages for pain and suffering, and punitive damages) far easier for plaintiffs.* Vehement dissents from these proposals were recorded in the individual comments of certain Study Group members, especially Chief Judge (ret.) Charles D. Breitel and George Clemon Freeman, Jr.**

Tier One

Tier One administrative remedies as recommended by the Study Group majority would permit recovery of full medical costs (without limit) and 2/3 of lost wages (up to a fixed monthly limit). Death benefits would also be paid to family members who had relied on the decedent for support. (Report at 206, 234).

Tier One administrative recoveries would be based in many cases upon a presumption of causation:

> "If the claimant in an administrative compensation proceeding proves:

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* Through the use of joint and several liability, broader strict liability principles, liberal joinder rules and a more liberal statute of limitations. (Report at 255-60).

** Comments by Charles D. Breitel, Study Group Member (Report at 295); Comments of George Clemon Freeman, Jr. (Report at 305)

> 1) that the source involved in the claim was engaged at the time of exposure in the production, transportation or disposal of hazardous wastes; and
>
> 2) that the claimant was exposed to such hazardous waste by proximity, contact, ingestion, inhalation, or by some other form of exposure; and
>
> 3) that the claimant suffered death or the kind of injury or disease which is known to result from such exposure
>
> then the following rebuttable presumptions should arise:
>
> (a) that the exposure to the hazardous waste generated, transported or disposed of, by or in the source, contributed to the death, injury or disease of the claimant; and
>
> (b) that the source by or in which such hazardous wastes were generated, transported or disposed of was responsible for the claimant's exposure and for the resulting death, injury or disease." (Report at 213-14 (indentation added)).

A claimant would not be required to demonstrate any fault on anyone's part in order to recover. (Report at 196, 209).

The Study Group majority made no study of the expected or potential costs of its proposed program. It did recommend that the fund out of which these administrative recoveries would be paid be provided initially through a tax on industry. (Report at 245). Further funding would be provided by subrogated agency claims against the owner, operator, generator, transporter, or other "alleged responsible party" after the agency had paid a claimant. (Report at 246). Such subrogated claims by the agency would be made on the same basis as a claimant's original claim -- *i.e.*, including the presumption of causation and lack of any required proof of fault; subrogated claims would not require proof that an owner, operator, generator, or transporter, etc., knew or should have known of the hazard. (Report at 246-47). Thus, a defendant's liability would be nearly absolute.

Tier Two

A Tier One administrative award would *not* bar a claimant from pursuing further damages in court under Tier Two. The agency granting a Tier One award would be reimbursed from any court award. The Study Group majority recommended that the courts be empowered to assess costs against a plaintiff who had previously obtained an administrative award, if the plaintiff failed to receive an award in court at least 25% larger than that granted by the agency. Such cost assessment, however, would not be mandatory. (Report at 197-98).

The Study Group majority recommended that the judicial process adopt a very liberal statute of limitations (based not only on discovery of the injury or disease but also on discovery of the cause of the injury or disease), liberal joinder rules, and joint and several liability for each defendant who has "contributed" to the injury, above some de minimis level. (Report at 255-58).

IV. DEFECTS IN THE PROPOSALS

A. Proponents' Failure to Demonstrate a Need for Radical Change

The proposals of the Study Group majority and others have been put forward despite the complete absence of evidence that large groups of plaintiffs exist with injuries caused by hazardous substances who will not be compensated by the present common law tort system. In the absence of any such showing, the American Insurance Association opposes legislation that would so radically alter traditional common law remedies and would render uninsurable those who would be subject to the new liabilities.

The lack of any reliable scientific basis underlying these calls for legislative reform is admitted in the Report:

> "The discussion of legal issues relating to injuries from hazardous wastes must proceed in a setting of factual and scientific uncertainty because, at this time, in mid-1982, it is impossible to determine the number of persons who

> have been injured or who could be injured by exposure to such sites." (Report at 21, footnote omitted).

Study Group Member George C. Freeman, Jr. in a letter dated March 2, 1982 (included in Volume Two of the Report) noted:

> "We have very little reliable information about how much truly toxic wastes are out there and how many persons may or may not have been exposed to them in circumstances that have caused, or are likely to cause, death, serious bodily injury or disease.
>
> * * *
>
> If relatively few people have in fact been injured by (or even exposed to) toxics, the fact that few cases for damages have been successfully brought should not be regarded as evidence that the current law is inadequate or unfair." (Id.)

Premature public outcry is not unprecedented in the field of hazardous substance legislation. In 1980 the Environmental Protection Agency released a study purporting to show chromosome defects among residents near the Love Canal. Based in large part on this study, more than 700 people were evacuated from the area by the federal government; a public outcry was launched and CERCLA was passed later the same year. Subsequent reports, however, reveal that this study "was so poorly designed that it should never have been undertaken."[*] A subsequent study released on May 17, 1983, by the Centers for Disease Control found no increase in chromosomal abnormalities among Love Canal residents (including 17 such residents who had participated in the original study) when compared to a comparable control group.[**] This newest study also noted that other "[e]xtensive studies at the Love Canal dump site in Niagara Falls, New York, giving particular attention to reproductive abnormalities and to cancer, have thus far defined no firm evidence of increased disease incidence or mortality . . . ."[***]

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[*] Boffey, "Study of Love Canal Finds No Direct Link to Genetic Disease", *New York Times*, May 18, 1983, at A1, col. 6. No comparable control group was used in the original study.

[**] Centers for Disease Control, Brookhaven National Laboratory & Oak Ridge National Laboratory, *A Study of Cytogenic Patterns in Persons Living Near the Love Canal* (hereinafter "CDC Study"); *see* "Study of Love Canal Finds No Direct Link to Genetic Disease", *New York Times*, May 18, 1983, at A1, col. 6. *See also* Dionne, "U.S. Finds Love Canal Neighborhood Is Habitable," *New York Times*, July 15, 1982, at A1, col. 1.

[***] CDC Study at 1. Similarly, there is no evidence that the tort system has failed in the oft-cited Environmental Law Institute study entitled *Six Case Studies of Compensation for Toxic Substances Pollution: Alabama, California, Michigan, Missouri, New Jersey, and Texas* (Senate Comm. on Environ. and
(Footnote continued)

Given this background, legislatures should be cautious and not rush headlong into enacting overbroad "reforms." For example, in September 1981 the State of California established a Hazardous Substance Compensation Account (Cal. Health & Safety Code §§ 25300-95) to provide compensation "for loss caused by a release, in California, of a hazardous substance" if the release was from an unknown source or an unknown liable party, or if the liable party could not be found, or was judgment proof. As of March 1983, not a single direct claim had been asserted against the fund.* Despite publicity concerning the lack of claims made against the fund,** at

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(Footnote *** continued from previous page) Public Works, 96th Cong., 2d Sess., Serial No. 96-13, Comm. Print, June 1980) (hereinafter "Six Case Studies"). Of the six cases studied -- which six were chosen specifically because they were believed to demonstrate "substantiality of harm" and not on any random basis -- only two included any evidence of serious personal injuries due to chemical exposures, and certain injured persons received compensation in each of those two instances. This two-month study heavily reflected the personal views of the authors, but failed to afford those views an evidentiary support.

* See Hazardous Waste Management Council, Compensation: An Analysis of Hazardous Waste Facility Insurance Requirements and Alternatives 28 (January 1983).

** "California Cleanup Fund Has Money Begging," New York Times, Monday, March 21, 1983, at A12, col.6.

the present time less than a dozen claims have been filed.

In view of these facts, North Carolina has recently adopted a more cautious approach. The 1982 Annual Report and Special Reports of the North Carolina Governor's Waste Management Board, while recommending certain limited changes in the application of strict liability principles under the tort law system, rejected an administrative remedy or any presumption of causation. (Id. at 53-56). With respect to administrative remedies:

> "The Board concluded that it is premature at this time to assume that the volume or complexity of hazardous waste liability cases will be so great that the general court system cannot handle them adequately." (Id. at 56).

B. Proponents' Failure to Consider the Staggering Costs of Their Overinclusive Proposals

It would be disastrous to structure a radical new "solution" in such a way as to eradicate the traditional tort law requirements that a plaintiff show a defendant was at fault and caused the harm complained of.* Such a "solution" would, in effect, require the chemical and oil industries to pay the costs of cancers, tumors, genetic damage, joint pains, headaches and other diseases or symptoms every time a claim of "exposure" to toxic waste was lodged, even if (as would almost always be the case) the claimant's disease resulted from a combination of other causes such as normal environmental or genetic factors unrelated to exposure to toxic waste. (See further discussion of the specific legal principles below).

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* Moreover, any "solution" that abandons these elementary principles might have serious ramifications for tort law in other areas, which no one intended to "reform."

For example, consider the present cancer statistics - just one disease that might result from exposure to hazardous substances. In 1983, an estimated 440,000 Americans will die of cancer, and an estimated 855,000 people will be diagnosed as having cancer.* Statistics show that more than 20% of the total deaths among Americans resulted from some form of cancer -- the second highest cause of death, after heart disease.** Yet, it has been estimated that approximately 2% (and no more than 5%) of cancer deaths are caused by air, water and food pollution in all its forms;* 1% or less may result specifically from exposure to pollutants found in hazardous waste sites.** However, the Study Group's proposal, echoed in many of the pending legislative proposals, would permit recovery by anyone who can claim non-occupational "exposure" to hazardous waste dumps and whose ailment cannot be demonstrated not to have resulted from such exposure -- an impossible burden for a defendant to meet, as more fully discussed below. Such a skewed system would certainly involve many times the number of recoveries than would be warranted by the small percentage of diseases arguably "caused" by hazardous substances.

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* E. Silverberg & J. Lubera, Cancer Statistics, 1983 10 (reprinted from Ca-A Journal for Clinicians, Vol. 33, No. 1, 1983 (American Cancer Society).

** Figures for 1978 (the last available year). Cancer Statistics, 1983, supra, at 11.

In light of these facts, the cost of the Study Group majority's overinclusive "solution" would be staggering. Just as with other types of compensation programs begun in the 1960s and early 1970s the costs can be expected to balloon to truly massive proportions. See "Toxic-Liability Bills Plant a Time Bomb of Entitlement Costs," The Wall Street Journal, Friday, June 3, 1983, p. 20, col. 3; Office of Management and Budget, Compensation for Toxic Substances Exposure (May 19, 1983). The experience under the federal Black Lung Benefits Act provides an apt warning:

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* See R. Doll & R. Peto, The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today, 66 J. Nat'l Cancer Inst. 1192, 1245-50. 1256 (1981); Technology Assessment Board, 97th Cong., Assessment of Technologies for Determining Cancer Risks from the Environment 91 (June 1981).

** Comments of Jim J. Tozzi at a conference on May 7, 1983 concerning Office of Management and Budget-sponsored Inter-Agency Study of Toxic Torts.

> "The Black Lung Act provides an example of what can happen when a compensation system is not well thought out. A General Accounting Office report issued in January 1982 found that 84 percent of claims were paid without adequate medical evidence.
>
> The result: In 1969, Congress estimated that the total program would cost between $40 million and $385 million. By 1972, costs to the federal government alone amounted to $33 million per month. In 1979, such costs amounted to $1.5 billion and reached $2 billion in 1982. The total cost of the program, including benefits paid directly by industry, is unknown."*

As a result, in 1981 Congress eliminated many of the presumptions of causation in the Black Lung Act because they had "led to an excessive number of claims and vastly greater expenditures than had been anticipated." (Report at 228). Black Lung Act costs are expected to level off or slightly decrease over the next few years.* Nonetheless, one critical presumption remains -- a rebuttable presumption that certain identified diseases were work-related if the employee could show 10 years of employment. Consequently, costs will not decline very quickly, and in fact continued to rise in 1982.**

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* J.B. Browning (Chemical Manufacturers Association), Public Compensation: A Chemical Industry Perspective 9 (April 5, 1983); see Problems Relating to the Insolvency of the Black Lung Disability Trust Fund: Hearings Before the Subcomm. on Oversight of the Comm. on Ways and Means of the House of Representatives, 97th Cong., 1st Sess. 2-15, 159-88 (1981).

The Black Lung Act is very limited in scope; costs for a sweeping hazardous substance program would be enormous, given the broad presumptions, unlimited awards and huge administrative costs inherent in the pending proposals.

To obtain a rough concept of this cost, assume that the number of cases that would be filed for all the types of diseases which plaintiffs may claim could result from exposure to hazardous substances -- cancer, respiratory ailments, tumors, genetic defects, coronary disease, etc. -- might be so low as one-fourth of the diagnosed annual number of cases of cancer alone. Rounding downwards, such an assumption yields an incidence of approximately 200,000 claims filed per year.*

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* Office of Management and Budget, Compensation for Toxic Substances Exposure, Chart 2 (May 19, 1983).

** Id. Further, the use of a presumption that a disease is work-related in the context of the Black Lung Act or workers' compensation statutes is inherently a limited one; the list of employees on the roster is finite and easily ascertainable. Such a presumption relating to hazardous substance exposure is open-ended, uncontrollable and potentially limitless in scope.

At this conservatively estimated rate of claims, and given the same average recovery in Tier One that is estimated for current workers' compensation cancer cases of $130,000 per award,** the Study Group's program would result in an annual cost of $26 billion in Tier One claims payments alone.*** Assuming allocated loss adjustment expenses (i.e., defense legal fees, etc.) of approximately 10% added to the total cost of these cases,* the annual bill in Tier One alone might be on the order of approximately $28 billion.

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* In view of the claims of the Environmental Protection Agency ("EPA") and legislative proponents, this number of cases is entirely within the bounds of possibility. EPA has claimed there are over 80,000 sites in the nation with contaminated surface impoundments, 90% of which "are believed to pose at least a potential threat of ground water contamination." Technology Assessment Board, 98th Cong., Technologies and Management Strategies for Hazardous Waste Control 5 (March 1983). Under the Study Group majority's approach, residents around these sites showing "exposure" may be able to recover, even where such "exposure" did not actually cause their disease or injury.

** Source: National Council on Compensation Insurance. See Technical Appendix A.

*** I.e., 200,000 claims times $130,000 per award. To put this annual cost in perspective, it represents more than twice the total workers' compensation loss and loss expense incurred by private carriers and state funds in 1981 (almost $12 billion), and would

(Footnote continued)

Additional awards to plaintiffs in Tier Two would add massive costs such as pain and suffering awards, attorneys' fees and litigation expenses. To the extent this estimate is conservative in its assumptions, it follows that the actual cost of the program will run even higher.

To require that this extraordinary cost -- far in excess of any proven level of injuries caused -- be shouldered by a few American industries would seem very

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(Footnote *** continued from previous page) add 7.0% to the inflation rate of chemical and petroleum product prices (given 1982 sales of $371 billion), along with a similar effect on the prices of all other goods and services that incorporate these products, if an industry tax were passed through to consumers.

* The National Council on Compensation Insurance figures showed allocated loss adjustment expenses of 10%, but it is reasonable to expect that for cases proceeding on through Tier Two litigation, such expenses would be three to four times higher. The Study Group majority's proposal provides no abatement in these transaction costs, since plenary court lawsuits are not barred; indeed, the Study Group's proposal merely adds the administrative costs to the present system.

unwise, especially in the face of foreign competition which would not be burdened by similar costs. See Letter dated March 10, 1982 by Study Group Member George C. Freeman, Jr. (included in Volume 2 of the Report). In addition, as the cost of complying with the law rises, the incentive to engage in illicit, secret dumping or to employ other illegal means of waste disposal rises.* Further, to assume that such an extraordinarily costly scheme is insurable, or worse yet to dismiss the issue with the Study Group majority's assertion that:

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* Concern has already been expressed that organized crime may be involved in the garbage and waste disposal industry. See, e.g., "Corruption Cited in Waste Business," New York Times, Sunday, May 1, 1983, at A52, col. 4; "Attorney Denies Dumping, Mob Connection," UPI Regional News story, Friday, April 29, 1983; "Poison Dump Stirs Alarm in Poconos," New York Times, Sunday, April 24, 1983, at A20, col. 1. As the New York Times reported last year in connection with the conviction of Russell W. Mahler:

> "New York can handle only 60 percent of the 1.4 million tons of industrial waste that companies in the state produce annually, and the pressure to get rid of some of it by illegal means is tremendous, according to state officials. 'If you pay the price, you can dispose of hazardous waste, but the price can be prohibitive,' said Jeremiah B. McKenna, counsel to the State Senate's Select Committee on Crime."

Chavez, "Toxic Waste Entrepreneur," New York Times, Thursday, May 27, 1982, at D1, col. 1.

> "insurability is not the sole or even the dominant factor that ought to determine whom the law protects and how,"*

is to court disaster. If no insurance were written to cover these risks, or if industry is unable to afford the premiums, there would be no effective means of spreading the enormous cost of the proposed system while at the same time offering the incentives to careful conduct that are traditionally provided by variable commercial insurance rates. Without insurance, it is unclear that our vital industries would even be able to meet this level of risk without jeopardizing their very existence. Unfortunately, this is precisely the perilous course which the Study Group majority, and proposed state and federal legislation modelled on its recommendations, would have us adopt.

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* Report at 185. See Letter dated May 17, 1982 from George C. Freeman, Jr. (included in Volume Two of the Report), which questioned the availability of insurance to cover the Study Group's proposals.

C. Proponents' Failure to Consider the Ramifications of the Specifics of Their Proposals

1. The Presumption of Causation

The heart of both the Study Group's Report and many of the legislative proposals introduced is a presumption that diseases contracted by an "exposed" person were caused by that exposure. But in fact, such diseases have a multiplicity of possible causes, of which hazardous substance exposure may be one of the least significant when compared with diet and certain habits (such as smoking).* The Study Group majority's attempt to legislate science, without regard to the actual facts that are available, is echoed in the vast majority of the bills that have been submitted to state and federal legislatures.**

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* See R. Doll & R. Peto, The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today, 66 J. Nat'l Cancer Inst. (1981); Technology Assessment Board, 97th Cong., Assessment of Technologies for Determining Cancer Risks from the Environment (June 1981).

** See, e.g., Massachusetts H. 5179, § 7(a); S.917, § 5(c)(3); S.946, § 3(b); H.R. 2330, § 102(a); cf. Minn. Stat. § 115B.07. Indeed, Senator Mitchell's proposal in S.946, after providing a "rebuttable presumption", goes on to state:

> "Evidence as to the physical weakness or personal habits of the individual on whom

(Footnote continued)

It may be impossible for a defendant to prove lack of causation from an exposure, given the long latency period for many toxic waste related diseases. Plaintiff, and not defendant, is in possession of all the facts concerning the onset of disease and the symptoms involved, personal diet and habits that might be the cause of the disease, and all background information concerning plaintiff's history and exposure to this or other hazardous substances. None of this information is public, unlike much of the information about hazardous substances in dump sites collected by the federal government under CERCLA. See, e.g., 42 U.S.C. § 9603(c).

Given ordinary human sympathies, a burden to disprove causation would be much harder for a defendant to sustain than it would be for a claimant to prove causation affirmatively. Thus, industry members would become virtual guarantors for the costs of all diseases and

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(Footnote ** continued from previous page)

> the claim is based shall not be used to rebut the presumption created by this paragraph."

Thus, for example, a person who contracted lung cancer after an exposure to hazardous substances would presumably recover under this formulation on the basis of the exposure, no matter how heavy a smoker he is. Smoking, however, might be many times the more significant contributory factor.

genetic defects occurring in an indeterminate population that lives, works, and vacations in, and perhaps even passes through, the vicinity of every hazardous waste site, without any real regard for whether the harms claimed were actually caused by the hazardous substances, or whether they resulted from or were primarily contributed to by other causes.

Insurance industry representatives and members of the Study Group itself also expressed concern that, despite a lack of any recommendation so providing, the presumption of causation available to claimants in a Tier One administrative proceeding would likely be imported into Tier Two by the courts,* resulting in full recoveries (including pain and suffering damages) to a large number of plaintiffs without proof of causation. This concern is heightened by examples such as the Minnesota Environmental Response and Liability Act (the "Minnesota Act"), passed in May 1983, which provides in Minn. Stat. § 115B.07 for restrictions on directed verdicts -- a provision that amounts in practice to a presumption.* This *de facto* presumption is by its terms made applicable to actions brought under the Minnesota Act "*or any other law* to recover damages for death, personal injury, or disease arising out of the release of a hazardous substance . . . ." *Id*. (emphasis added).

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* Report at 175, 264-65; Comments of Study Group Member Charles D. Breitel (Report at 300); Comments of Study Group Member George Clemon Freeman, Jr. (Report at 318).

2. Liability Without Regard to the Defendant's Fault or Lack of Fault

The Study Group majority recommended that all consideration of fault be eliminated entirely in both Tier One and Tier Two proceedings.** Even the attenuated fault concepts embodied in the "strict liability" provisions of the Restatement (Second) of Torts § 519-52 would be disregarded by the Study Group majority.

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* Minn. Stat. § 115B.07 prohibits a directed verdict for the defendant on the issue of causation if plaintiff "produces evidence sufficient to enable a reasonable person to find that" (a) the defendant is responsible for the release of the hazardous substance; (b) the plaintiff was exposed; (c) "the release could reasonably have resulted" in the amount and duration of plaintiff's exposure; and (d) "the death, injury, or disease suffered by the plaintiff is caused *or significantly contributed to* by exposure to the hazardous substance in an amount and duration experienced by the plaintiff." The statute continues:

> "*Evidence to a reasonable medical certainty that exposure to the hazardous substance caused or significantly contributed to the death, injury or disease is not required* for the question of causation to be submitted to the trier of fact." *Id*. (Emphasis added).

** Report at 196, 209, 260, 265-66.

The Second Restatement applies a strict liability standard to abnormally dangerous activities. At the same time, it:

> "cautions against defining a type of activity as 'abnormally dangerous' in and of itself, however, and advocates considering the activity in light of surrounding circumstances on the facts of each case. This, in essence, shifts consideration from the nature of the activity to the nature and extent of the risk."

Clark-Aiken Co. v. Cromwell-Wright Co., 323 N.E.2d 876, 887 (Mass. 1975) (stated with respect to the Tentative Draft of the Second Restatement). Section 520 of the Second Restatement provides:

> "In determining whether an activity is abnormally dangerous [and thus gives rise to strict liability], the following factors are to be considered:
>
> (a) Whether the activity involves a high degree of risk of some harm to the person, land or chattels of others;
>
> (b) Whether the gravity of the harm which may result from it is likely to be great;
>
> (c) Whether the risk cannot be eliminated by the exercise of reasonable care;
>
> (d) Whether the activity is not a matter of common usage;

> (e) Whether the activity is inappropriate to the place where it is carried on; and
>
> (f) The value of the activity to the community."*

Thus, the Second Restatement requires a court to consider whether an activity is necessary to society and is being carried on in an appropriate location, among other things, before finding it to be "abnormally dangerous" and imposing "strict liability" for injuries that result from such an activity.

The Study Group majority, however, would impose strict liability without first considering all the circumstances. The Study Group majority stated:

> "Formulations of strict liability that balance the hazard of the activity against its utility and its appropriateness to the locale import considerations of negligence that entail significant barriers to the recovery of damages for injuries from exposure to hazardous wastes. To remove these barriers, the application of a theory of strict liability is needed that focuses on the nature of the hazardous waste activity itself, and on the magnitude of the risk." (Report at 260).

The trend in tort law today is not toward absolute liability such as was recommended by the Study Group majority, but rather toward more limited "strict liability" concepts based on the Second Restatement.* Today the overwhelming majority of states that have considered Section 520 of the Second Restatement have utilized its factors in determining cases.**

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* Under the case law, disposal of hazardous waste has not uniformly been found to be abnormally or inherently dangerous. See Ewell v. Petro Processors of Louisiana, 364 So.2d 604 (La. Ct. App. 1978).

This acceptance of the Second Restatement's factors is based upon sound considerations. These same factors are, in part, the criteria used by underwriters to determine insurance premium rates for a particular insured. Absolute liability, without any regard to proper conduct, on the other hand, leaves no room for consideration of fault, and provides no incentive for companies to behave well in handling their hazardous substances. An absolute liability system would penalize necessary industries conducted in a reasonable manner just as heavily as marginally useful, irresponsibly conducted activities; careful conduct would become irrelevant. The balanced approach of the Second Restatement should not now be abandoned in favor of an ill-considered stampede to an absolute liability standard.

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* See, e.g., Dye v. Burdick, 553 S.W.2d 833, 839-40 (Ark. 1977).

** Report at 120-24; see, e.g., Cropper v. Rego Distribution Center, Inc., 542 F. Supp. 1142, 1150 (D. Del. 1982); City of Northglenn v. Chevron U.S.A., Inc., 519 F. Supp. 515 (D. Colo. 1981); Indiana Harbor Belt Railroad Co. v. American Cyanamid Co., 517 F. Supp 314, 319 (N.D. Ill. 1981); Harper v. Regency Development Co., 399 So. 2d 248 (Ala. 1981); Cities Service Co. v. State, 312 So. 2d 799 (Fla. Dist. Ct. App. 1975); National Steel Service Center, Inc. v. Gibbons, 319 N.W.2d 269 (Iowa 1982); John T. Arnold Associates, Inc. v. City of Wichita, 5 Kan. App. 2d 301, 615 P.2d 814 (1980); Yommer v. McKenzie, 255 Md. 220, 257 A.2d 138 (1969); Locke v. Mach, 115 Mich. App. 191, 320 N.W.2d 70 (1982); Ferguson v. Northern States Power Co., 307 Minn. 26, 239 N.W.2d 190 (1976); Otero v. Burgess, 84 N.M. 575, 505 P.2d 1251, 1255 (Ct. App., 1973); Doundoulakis v. Town of Hempstead, 42 N.Y.2d 440, 368 N.E.2d 24, 398 N.Y.S.2d 401 (1977); Wirth v. Mayrath Industries, Inc., 278 N.W.2d 789 (N.D. 1979); McLane v. Northwest Natural Gas Co., 255 Or. 324, 467 P.2d 635 (1970); Fleege v. Cimpl, 305 N.W.2d 409 (S.D. 1981); Pacific Northwest Bell Tel. Co. v. Port of Seattle, 80 Wash. 2d 59, 491 P.2d 1037 (1971); Peneschi v. National Steel Corp., 295 S.E.2d 1 (W.Va. 1982). But see New Jersey v. Ventron Corp., 182 N.J. Super. 210, 440 A.2d 455 (App. Div. 1981).

3. Imposition of Excessive Liability on Non-Responsible Defendants

The use of absolute liability without regard to fault is particularly far-reaching in light of the Study Group majority's recommendation that defendants be jointly and severally liable in a court proceeding. (Report at 257-58). Thus, a very prudent manufacturer who merely had the misfortune to place his waste in the same site as another, who acted intentionally or even criminally, could be held liable for the entire judgment. If the negligent enterprise cannot be found, or is judgment proof, the prudent manufacturer will be forced to bear the entire liability without recourse despite careful and proper behavior.

This situation is exacerbated further by the presumption proposed by the Study Group majority. Under that presumption, for example, a claimant might simply prove his exposure to "Substance X" and that a particular manufacturer deposited "Substance X" in the dump site the claimant asserts is the source of the contamination affecting him. In a subrogated liability proceeding against the manufacturer, liability would be found unless the manufacturer meets a burden of showing that somebody else's "Substance X" was the source of the contamination. If there was a lack of historical evidence, as may often be the case, such a burden could not be maintained. Thus, a perfectly prudent, careful and non-responsible party who dealt in "Substance X" might ultimately bear a liability for harm it never caused, but which was instead caused by the irresponsible activities of another person. The unpredictability of this liability, coupled with its potentially huge amount, would make it extremely difficult to insure.

4. The Initial Funding Tax and Further Removal of Incentives for Careful Handling of Hazardous Substances

The proposal that a compensation program should be initially funded through taxes on industry* would place an impossibly high burden on domestic industries already faced with stiff foreign competition unburdened by such expenses. See Comments of Study Group Member George Clemon Freeman, Jr. (Report at 311-14). Funding by such a generic tax burden prevents an enterprise from reducing its cost by prudent behavior.

> "The Study Group claimed that the costs of injuries should be carried by the industry or enterprise that creates them 'because efforts to reduce the cost will result in greater care, more safety and protection of the public' . . . [see Report at 248]. Though this generalization is often true, it is an inappropriate justification of the proposed tax.
>
> The Study Group's rationale is wrong on at least two counts. First, a tax on industry output provides little or no incentive for any single enterprise to reduce its liability. A firm does not reduce its tax burden by taking due care to avoid hazardous waste damages; therefore, such a funding mechanism simply does not act as a deterrent. Secondly, the fund is only applied to exposures that have either happened in the past or for which there is no responsible party; obviously, it is impossible to deter this type of exposure and, consequently, the tax provides no appropriate incentives. . . ."**

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* Report at 245.

(Footnote(s) ** will appear on following pages)

Such a tax would eliminate the use of commercial insurance, thus further removing any incentive to handle hazardous substances properly, and eliminating a valuable social mechanism of industrial self-regulation.

Insurance is a method of assessing and spreading risk, which has proven to be efficient in determining proportionate potential liability. Each insurer, in competition with all other insurers, puts itself at risk based upon its determination of an insured's potential liability. Using underwriting criteria that include not only statistical data but also loss control and loss prevention expertise, an insurer makes a determination as to which facilities or activities it will insure. In so doing, insureds that may treat or deal with the same quantity and type of toxic substances may be charged different premiums for their coverages because of judgments made by underwriters that reflect tangible and intangible qualities ranging from record keeping, to attitude toward loss control, to the level of personnel dealing with loss control and prevention.

** National Economic Research Associates, Inc., The Role of Economics in Tort Law Reform, (Draft Memorandum by F. Dunbar), at 10-11 (November 8, 1982).

Myriad factors, together with the personal experience of the underwriters, will determine the effectiveness of the underwriting process. This process is totally unlike a tax system, which treats the facilities disposing of the same or similar quantity or type of toxic substance in an identical manner, as though they were identical. The insurance mechanism, operating in conjunction with a sound tort system, provides carefully honed financial incentives to proper behavior and injury prevention, which can never be duplicated by a tax-based system.

5. Retroactivity

> "[I]f regulation under RCRA is effective, there is hope that there will be few cases of injury from hazardous waste in years to come. The recommendation for the compensation remedy is based on the possibility and concern that past hazardous waste disposal practices may have created a backlog of injuries and exposures that the legal system will need to clean up." (Report at 195).

With these words, the Study Group majority focused its attention on the issue of retroactive liability, relying heavily upon the speculative "possibility" that past practices "may have created" an as yet unidentifiable "backlog" of cases for the legal system to "clean up."

The pitfalls for the insurance industry of creating new, massive, retroactive liability for past practices are well known. The imposition on insurers of new obligations beyond those contemplated by the parties to the insurance contracts could be devastating to the entire insurance industry. Insurers have collected their premiums and set aside reserves based upon the law as the parties understood it at the time of contract. It is neither just nor sound practice to expand these obligations by legislative fiat. Further, as to future underwriting, the idea that such a massive liability might be created at any future date would certainly chill the most courageous underwriter. This type of sudden liability expansion is more likely to deter the growth of insurance markets than it is to provide a financially sound system of injury compensation; it would therefore defeat its own objectives.

The Study Group majority itself was aware of the problem that an overtly retroactive liability scheme would present; it attempted to circumvent retroactivity to some extent. While its recommended scheme would allow a number of retroactive claims by claimants* against the

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* Claims could be filed within three years of discovery, whether discovery occurred before or after passage of the implementing legislation. (Report at 209). Further, since discovery of injury might take place many years after the initial exposure to (Footnote continued)

administrative agency (Report at 209-10), it would permit subrogated claims against industry members only for injuries or damages caused by exposure to "wastes disposed of, transported or spilled _after_ the adoption of this proposal. . . ." (Report at 246). However, this is not an effective remedy to the problem of retroactive liability. Industry would end up footing the bill retroactively anyway because, under the Study Group majority's recommendations, unsubrogated claims would be paid through a tax on industry. In all fairness, if society decides to pay such retroactive costs, society in general should foot the bill, rather than attempting to load it unfairly on a small segment of its members.

Further, as we have seen, "toxic torts" may often involve diseases that take as long as 20 or 30 years to develop; claimants may have been exposed to a combination of substances deposited by a number of different companies both before and after implementing legislation is passed. It might be virtually impossible to sort out injury resulting from substances deposited both

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(Footnote * continued from previous page)
toxic waste, many claims -- and certainly the most catastrophic ones -- would in effect be "retroactive."

before and after the cut-off date, or from substances deposited by different companies. Subrogation suits would thus become an expensive nightmare of claims and cross-claims.* Alternatively, legislative proposals such as the Minnesota Act have permitted suits retroactively unless there is proof that "the substance was placed . . . in or on the facility wholly before" a cutoff date -- in Minnesota the date is set at January 1, 1980.** But for activities that continued after the cutoff date, the Minnesota Act provides extensive retroactive liability as a practical matter, and also fails to solve the problem of cross-claims, thus creating the worst possible "solution."

6. Add-On Remedies

> "[F]ree access to the plenary tort action after the claimant had already had a compensation award on Tier One would be prohibitively expensive from the insurance point of view. A system which called for a binding election between the tort remedy and the compensation remedy

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* The continued or increased use of joint and several liability of defendants in Tier Two lawsuits would also presumably result in a tangle of cross-claims for contribution in the courts.

** Minn. Stat. § 115B.06(1)(b). The Minnesota Act also provides an additional defense if the substance was deposited prior to January 1, 1973 and defendant's activity was not an "abnormally dangerous" one. Minn. Stat. § 115B.06.

> would be more readily insurable." (View Expressed to the Study Group, Report at 174).

Administrative remedies, in order to be practical, have traditionally involved a trade-off: relaxed standards of proof and easier awards must be balanced by a limitation on the amount recovered. Despite its claims that Tier One is modelled on workers' compensation (Report at 195), the traditional quid pro quo associated with such a compensation system has not been adopted by the Study Group majority's recommendation. Instead, the Study Group majority:

> "adopted the worst of both worlds: an administrative compensation system with liberal evidentiary standards and generous benefits, as well as continued availability of traditional tort law." Office of Management and Budget, Compensation for Toxic Substances Exposure 13 (May 19, 1983).

By providing an administrative recovery with reduced evidentiary burdens for plaintiffs, while doing nothing to provide either a bar or a binding election against further tort claims in court, the Report has gone far beyond the workers' compensation model it claims to follow. Since plaintiffs will typically be seeking large "pain and suffering" awards, which they would not receive in Tier One, the incentive to "go for it" in court would be high. A plaintiff could use his Tier One administrative recovery to fund a subsequent lawsuit in court, thus (in effect) causing the likely defendants to pay the cost of suits against themselves.

The Study Group majority's sole attempt to limit the use of both Tiers by plaintiffs was to permit (but not require) a judge to impose court and expert witness costs on the plaintiff if the plaintiff fails to recover at least 25% more in court than he did in the Tier One administrative proceeding.*

> "Such an assessment, to be made at the discretion of the judge in the plenary court proceeding, is to take into account whether the plaintiff's action was brought in good faith, with a view of avoiding frivolous and duplicatory proceedings." (Report at 202).

In reality, it is doubtful that such costs would be assessed with any frequency. Plaintiffs will usually be individuals of limited means suing defendants who are large corporations; as a practical matter, it would be a rare case in which a judge would find plaintiff's pleading sufficiently frivolous to warrant the imposition of a cost sanction.

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* A Joint Statement of dissenting Study Group members proposed that once an administrative award was given, claimant should be required to submit to a hearing in court to determine if a lawsuit is warranted, before being allowed to proceed with the lawsuit itself. (Report at 286). Even that system, however, involving yet another hearing, would proliferate legal proceedings and transaction costs. In this regard, consider the view expressed by Study Group Member Jeffrey O'Connell (Report at 354-55) that the three-proceeding structure proposed by the Study Group (i.e., administrative claim, subrogated claim, claimant's lawsuit) was excessive. Mr. O'Connell nonetheless joined in the Joint Statement.

With much to gain and virtually nothing to lose, it is unlikely that many plaintiffs would be deterred from going forward in both Tiers. Thus the Study Group majority's proposal would simply increase the administrative costs of the system overall by adding the Tier One proceedings and administrative costs to the present proceedings and costs of court litigation continued in Tier Two. Such an expensive, cumbersome system lacks any logical support.

V. CONCLUSION

The recommendations of the Study Group majority, and the legislative proposals based on those recommendations, are both unfair and unwise. It makes little sense to rush into such a radical "solution," involving so many pitfalls and draw-backs, as well as staggering costs well in excess of the harm caused. Instead, concerted effort should be devoted to the prevention of such problems in the future, by rigorous enforcement of existing laws, such as the Clean Air Act, Clear Water Act, and RCRA, and by retention of the incentives for proper conduct inherent in the tort system.

In addition, funding should be increased for professionally-conducted studies to determine in a rigorous way what causal links exist between exposure to hazardous substances and the harms claimed to result from such exposure. Once science has established what causal connections exist, there will be little need for Congress or state legislatures artificially to ordain such connections by legislative fiat, or to require compensatory payments from those who are not at fault and not the responsible parties. The American Insurance Association suggests that dispassionate scientific inquiry, coupled with traditional tort law principles assigning liability to those at fault who caused the harm, is the best and most rational means of achieving a just, economic, insurable and workable compensation system for injuries caused by hazardous waste.

TECHNICAL APPENDIX A

Estimation of Average Claim Costs for Workers' Compensation Cancer Cases

The National Council on Compensation Insurance (NCCI) has estimated the current average cost of a worker's compensation cancer claim to be $130,000 based on data for claims which have already been filed, as explained below.

Worker Benefits

A review of reported workers' compensation cancer cases showed that the typical claimant was a fifty year old male who lived for three years after he became disabled and incurred medical expenses approximately equal to his wage loss benefits during that period. The costs of these claims were calculated using current wage rates so that no adjustment for inflation was made.

Survivor Benefits

Existing rules allow for the worker's widow to continue receiving benefits after his death for a duration of up to ten years unless she dies or remarries prior to that time, whereupon benefits cease. Widow's benefits were limited according to these rules using standard mortality tables and the results of an NCCI remarriage study which provided distributions, by age, of the number of women expected to die or remarry in each subsequent year.

November 21, 1983

NOV 23 1983
Committee on Inv.
and Oversight
on Public W.
Transportation

COMMENTS OF THE
AMERICAN INSURANCE ASSOCIATION
SUBMITTED BY
M. PATRICIA CASEY TO THE
SUBCOMMITTEE ON INVESTIGATIONS & OVERSIGHT
OF THE
COMMITTEE ON PUBLIC WORKS & TRANSPORTATION
HOUSE OF REPRESENTATIVES
CONCERNING
TOXIC WASTE VICTIM COMPENSATION
HEARINGS OF NOVEMBER 8 & 9, 1983

The American Insurance Association was pleased to be present at the testimony concerning the activities of states on hazardous waste legislation and concerning the scientific problems involved in the compensation issue. It is pleased to present its comments on the testimony heard by the Subcommittee.

The American Insurance Association is a trade association composed of 170 publicly owned insurance companies providing property and casualty coverages. The Association's members provide the majority of general commercial liability insurance in the United States. It is likely that members of the American Insurance Association will provide insurance for those subject to liability for hazardous substance compensation.

Our members have been instrumental in helping to shape the financial regulations under the Resource Conservation & Recovery Act and have been represented at the state legislative level also. A market for insurance to cover liabilities from pollution incidents can develop only if the liabilities to be insured are reasonably predictable and affordable. Since the members of the American Insurance Association have responded in the past to emerging liabilities, the Association is interested in proposals concerning new liabilities.

The American Insurance Association applauds the efforts of the Subcommittee on Investigations & Oversight to review the activities at the state level. In the past two years, our Association has seen that state legislative activity has expanded greatly in this area in response in part to the increased awareness of the public to the dangers of hazardous substances. The laws in Minnesota, California and North Carolina are representative of the significant interest and hard work by those state legislators.

Insurers can underwrite only liabilities which manifest some degree of predictability. They do not insure unlimited exposures. Yet currently, very little data on pollution claims, their frequency and severity, exists to assist underwriters in assessing the risk.

Given the problems involved in underwriting pollution coverages, the members of the American Insurance Association and other insurers strongly opposed the enactment of Minnesota's Environmental Response & Liability Act about which Representative Long testified on November 8. Our Association urged the Governor to veto Minnesota's Environmental Response & Liability Act in a letter dated May 2, 1983 (copy attached). T. Lawrence Jones, President of the American Insurance Association, in that letter wrote:

> "Our member companies, as well as the insurance industry as a whole, have responded to the need for coverage for environmental harms through the environmental impairment liability coverage. Our Association has been heavily involved with the consideration of environmental issues at state and federal levels. We are strongly supportive of enforcement of laws to

> protect our environment and our public's health. We believe absolutely that prevention of harm to the environment is essential. During the past two years, our member companies have responded to the new challenge; they have entered the environmental liability market, and developed new insurance policies, including a policy covering gradual contamination. They have aggressively marketed this coverage."

He objected to the extensive liability provisions which would severely curtail the developing insurance market for environmental harms. He noted:

> "Among other provisions, the Conference Committee proposals contain a presumption of causation so vague and sweeping that it would eliminate the predictability of claims against specific insureds needed by underwriters. The Conference Committee proposal would also retroactively apply a standard of strict, joint and several liability for unprecedented personal injury and property damage without limits. Even the federal Superfund, which contains extensive liability provisions for property damages, provides for maximum limits of liability."

Further, he informed the Governor that:

> "Our member companies have informed me that should the Conference Committee proposals become law, many of them will consider severely restricting their current policies in the State of Minnesota. Because of the effect of unpredictability and unlimited liability which this legislation would have on the market, companies at all willing to write this coverage may have to make it so expensive as to be prohibitive, especially for small and medium sized customers."

Since that time, some companies have already decided they will not provide this coverage. Other companies are reviewing their position. We do not know of any company that currently provides coverage under Minnesota's enactment.

The American Insurance Association feels that it is important to correct some of the misperceptions of our industry contained in Representative Long's testimony. Ms. Long quoted extensively from William Mahoney, Vice President of the brokerage company Marsh & McLennan, from a presentation he made on May 23, 1983. At that time, Mr. Mahoney stated that the capacity for coverage of pollution liabilities was not a problem and that it was indicated that the "entire insurance industry - from the primary placement up through the excess layers - will be ready and willing to accept pollution liability insurance risks." Mr. Mahoney was speaking about the general market for pollution, particularly gradual pollution coverages, and his remarks were not intended to characterize the market in Minnesota after the enactment of the Minnesota Environmental Response & Liability Act.*

The Minnesota Environmental Response & Liability Act represents a significant expansion of tort law liabilities. The liabilities under that Act are extensive as well as ill-defined. As you are aware, insurers need to know the boundaries of their liabilities in order to properly price the risk, to reserve sufficient loss amounts, and naturally to attempt to provide some profit for the benefit of their shareholders. Two provisions of the Minnesota law present such formidable obstacles to our underwriters that we urge you not to consider proposing similar provisions in any federal proposal.

First, the cause of action created by that legislation will allow the award of almost unlimited damages using the standard of strict, joint and several liability on a retroactive basis. While the law states [illegible] personal injury cause of action cannot be sought if the release or threatened release occurred "wholly before" the effective date of July 1, 1983, the law as we read it is almost indefinitely retroactive. It will be difficult for any defendant to prove, particularly with regard to groundwater contamination, when a release or threat of release began or had wholly occurred. Also, since the law contains a provision under which the judge appears able to assign accrual of the cause of action at almost any point, this adds to the concern about the retroactive application.

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* In a telephone conversation on November 21, 1983 with Mr. Mahoney, he stated that he "never addressed the victim's compensation act at all," and that his remarks were taken out of context by Ms. Long. He said he was only addressing the issue of availability of environmental impairment liability coverage for those companies seeking to purchase insurance.

The second problem for insurers is the provision relating to causation. The November 8 and November 9, 1983 hearings before your Subcommittee contained many references to the issue of causation. This issue is of paramount concern to insurers as it triggers the involvement of the insurance industry. We noted that some speakers on November 8 urged enactment of legislation to weaken the burden of proof of causation. Insurers are gravely concerned about the weakened causation provisions in the Minnesota law. Section 7 of that Minnesota law would prohibit a judge from directing a verdict against a plaintiff if the plaintiff could show that he was exposed to "the" hazardous substance, that the release "could" have resulted in the plaintiff's exposure, and that the injury was caused or significantly contributed to by exposure to "the" substance. This section amounts to a presumption of causation in favor of the plaintiff.

As Leslie Cheek, Vice President of Crum & Forster, pointed out to the Minnesota legislators, there is no requirement that plaintiff's exposure was to the particular hazardous substance of the particular defendant. Further, that provision states that "evidence to a reasonable medical certainty that exposure to the hazardous substance caused or significantly contributed to the death, injury or disease is not required for the question of causation to be submitted to the trier of fact." Mr. Cheek, in his testimony to the Minnesota legislature, pointed out that this opens the question of what, if anything, is required as the plaintiff's burden of proof on the issue of causation. As the damages for which a plaintiff could sue include not only medical expenses and loss of income but any loss of past or future profits, any damages for pain and suffering, as well as damages to the national resources, insurers are justifiably concerned about their exposures if they try to provide pollution coverage.

We submit that intelligent efforts to define environmental liabilities in a reasonable manner will result in an insurance market which can assist in the compensation of individuals harmed through hazardous substances. Our industry has an interest in marketing pollution coverages and is in fact trying to grapple with some of the complexities in this area through task forces and intra and inter-industry research.

Our industry is complex and many faceted. Statements are easily taken out of context. As an example, Ms. Long called your attention to the paper prepared by R. J. Taylor of the brokerage company Fred S. James and seemed to cite it as presenting a favorable view of the Minnesota Act. That study merely compared the then proposed Minnesota bill to the Federal Superfund. While that study stated that it is important to keep in mind that not all liability created under the Federal Superfund is currently insurable, it concluded that the enactment of state legislation such as the Minnesota bill will not "in itself make insurance coverage unavailable to all risks within the state" (report at pages 4 and 17). This does not constitute a representation that insurance under that law would be available and affordable.

The American Insurance Association believes that in fact, the coverage is not.

The American Insurance Association appeared before the North Carolina Governor's Waste Management Board to discuss liability issues relating to the hazardous waste industry. Their 1982 Annual Report with recommendations showed that members of the Board had studied the issues in depth. Notably, with respect to the issue of concern to your Subcommittee, the Board concluded that it was premature at this time "to assume that the volume or complexity of hazardous waste liability cases will be so great that the general court system cannot handle them adequately (report at page 56). Further, the Board noted that implementation of a stringent regulatory program should reduce the potential for injuries. AIA supports this regulatory conclusion.

The Subcommittee is to be commended for its attempts to have present scientists involved with hazardous substances from the Centers for Disease Control, the National Institute for Occupational Safety & Health, and the National Science Foundation, as well as for its open-mindedness toward the opinions of the chemical industries such as those represented on November 9 by Union Carbide and the Atlantic Richfield Companies.

Insurance is indeed only a part of the solution to the problem involving hazardous substances and compensation for losses. Yet, as Mr. Kusch of Atlantic Richfield pointed out, it is not an insignificant factor in considering how to compensate persons, property and the environment. Accordingly, we would urge you to consider the limitations of our industry as well as the desire of our industry to provide a market for compensation. The American Insurance Association appreciates the opportunity to be involved in the legislative process. We previously forwarded to you our position paper in response to the 301(e) study developed with the assistance of Sheila Birnbaum of New York University and we would be pleased to answer any questions that you might have at any time.

Respectfully submitted,

M. Patricia Casey

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MPC/dt
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VIA POST OFFICE EXPRESS MAIL

May 2, 1983

The Honorable Rudy Perpich
Governor
State of Minnesota
State Capitol
St. Paul, Minnesota 55155

Dear Sir:

I am President of the American Insurance Association, a trade association of 165 stock property and casualty insurers. Many of our members do business in Minnesota. They greatly value their Minnesota clients.

Our member companies, as well as the insurance industry as a whole, have responded to the need for coverage for environmental harms through the environmental impairment liability coverage. Our Association has been heavily involved with the consideration of environmental issues at state and federal levels. We are strongly supportive of enforcement of laws to protect our environment and our public's health. We believe absolutely that prevention of harm to the environment is essential. During the past two years, our member companies have responded to the new challenge; they have entered the environmental liability market, and developed new insurance policies, including a policy covering gradual contamination. They have aggressively marketed this coverage.

However, underwriters currently have little data available because this coverage is brand new. They must base the premium charged on their best analysis of the risk, and the existing parameters of common law liability.

Reliable reports of the proposed provisions from the Joint Conference Committee pertaining to H.F. 76 and S.F. 220 indicate that the Minnesota Legislature is seriously considering enacting a novel liability structure for those engaged in the hazardous waste industry. If enacted, it will severely curtail the young insurance market which is now forming to cover environmental harms.

Among other provisions, the Conference Committee proposals contain a presumption of causation so vague and sweeping that it would eliminate the predictability of claims against specific insureds needed by underwriters. The Conference Committee proposal would also retroactively apply a standard of strict, joint and several liability for unprecedented personal injury and property damage without limits. Even the federal Superfund, which contains extensive liability provisions for property damages, provides for maximum limits of liability.

The issue of compensation for bodily injury and property damage arising from hazardous waste related incidents is of national importance. It is being considered and debated by academicians, state and local councils, national task forces, and industry. The scientific analysis of the problem and its extensiveness is only just beginning. Nevertheless, insurers have been willing to enter the market despite not knowing the full dimensions of the issue because they so far found market conditions sufficiently reasonable to underwrite.

Our member companies have informed me that should the Conference Committee proposals become law, many of them will consider severely restricting their current policies in the State of Minnesota. Because of the effect of unpredictability and unlimited liability which this legislation would have on the marker, companies at all willing to write this coverage may have to make it so expensive as to be prohibitive, especially for small and medium sized customers.

When you receive this bill, we strongly urge that you veto it.

Sincerely,

T. Lawrence Jones
President

TLJ/lc

bc: Robert Johnson
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of Popham, Haik, Schnobrich, Kaufman
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George M. Mulligan
Fred P. McGarvey
Edward A. Stowell

(Scientific Affairs – A)

Proceedings
American Medical Association
House of Delegates December 1981

REPORTS OF COUNCIL ON SCIENTIFIC AFFAIRS

The following reports, A–G, were presented by William D. Dolan, M. D., Chairman:

A. HEALTH EFFECTS OF "AGENT ORANGE" AND DIOXIN CONTAMINANTS

(Reference Committee E, page 282)

HOUSE ACTION: ADOPTED

The American Medical Association's Council on Scientific Affairs, in response to a request from the Medical Student Section, has reviewed the medical evidence regarding the toxicity and long-term health effects of Agent Orange and its associated contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). This Executive Summary, which was prepared by the Council on Scientific Affairs Advisory Panel on Toxic Substances,* summarizes the findings in its Technical Report on the subject, which is available on request.

BACKGROUND

During the latter stages of the United States' involvement in Vietnam, herbicidal mixtures of 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), otherwise identified by the military as Agent Orange, were sprayed over certain areas of Vietnam for the express purpose of defoliating the jungle and destroying one of the enemy's means of concealment. Similar spray programs have been used in the United States as a means of forestry management. For the past thirty years, mixtures of 2,4-D and 2,4,5-T have been used extensively by the home-owner and farmer for ridding lawns and agricultural acreage of unwanted broadleaf vegetation. Large numbers of persons have been exposed to varying amounts of 2,4-D and/or 2,4,5-T, as well as the contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), in the normal course of employment.

Also over these years, there has been a number of industrial incidents wherein workers, as well as civilian populations, have been subjected to accidental exposure to these compounds. There are now pending a number of litigious actions and government regulatory responses that are based upon alleged adverse health effects from such exposures.

The most serious of these allegations by Vietnam veterans and persons who were involved in accidental industrial exposures assert that Agent Orange, or compounds of a like nature, may have caused malignant tumors, sterility, spontaneous abortions, birth defects, disfiguring skin diseases and other illnesses. In spite of the voluminous data on the biological effects of the phenoxy-type pesticides and the associated chlorinated dioxins, there is still very little substantive evidence for the many claims that have been made against these compounds. Data from experimental animals do indicate that TCDD is a toxic material; however, while suggestive, the animal data are not necessarily applicable to man. Still, a number of those exposures to TCDD of industrial and general populations that have occurred have been sufficiently well documented to offer some insight into the effect of TCDD on man.

Agent Orange, or Herbicide Orange, was a label given by the U. S. military forces to a 50:50 mixture of the n-butyl esters of 2,4-D and 2,4,5-T together with a minor amount (1%) of the free acid of 2,4,5-T and varying amounts of the contaminant TCDD. Agents Green, Pink and Purple also were used as defoliants by the military forces from 1962 through 1964; a time when very few American troops were committed to the field. Another formulation, Herbicide Orange II, was similar to Agent Orange except that the isooctyl ester of 2,4,5-T was substituted for the n-butyl ester of 2,4,5-T. In addition to Agent Orange, other spray defoliants used in Vietnam included Herbicide White, whose active ingredient was picloram (or the tri-isopropanolamine salt of 4-amino-3,5,6-trichloropicolinic acid). This substance is very persistent in the environment and highly carcinogenic in rats and mice. Lesser amounts of Herbicide Blue, which contained sodium cacodylate* (26%) and cacodylic acid (5%), were used. Though the long-term effects in humans of either or both of these compounds is uncertain, picloram and cacodylic acid should be considered along with the above agents that were encountered in Vietnam.

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*John R. Beljan, M. D.; Nelson S. Irey, M. D.; Wendell W. Kilgore, Ph. D.; Kazuo Kimura, M. D., Ph. D.; Raymond R. Suskind, M. D.; Jaroslav J. Vostal, M. D., Ph. D.; R. H. Wheater, M. S., Secretary

TCDD may form as a by-product of the synthesis of 2,4,5-trichlorophenol (TCP), a precursor of 2,4,5-T, when 1,2,4,5-tetrachlorobenzene is subjected to alkaline hydrolysis at elevated temperature and pressure. If the reaction temperature is allowed to go above 180° C, the sodium-2-hydroethoxide (formed from the ethylene glycol solvent and caustic soda) decomposes exothermically and promotes the dimerization of sodium trichlorophenate to TCDD. The presence of TCDD as a contaminant of 2,4,5-trichlorophenol was discovered in 1957, when workmen involved in the manufacture of TCP developed chloracne.

About twenty years ago, commercially available 2,4,5-T contained anywhere from 1 to 70 ppm of TCDD. When the industry became aware of the contaminant's existence and toxicity, production operations were monitored and altered to reduce the level. Current manufacturing operations are able to control the amount of TCDD in commercial 2,4,5-T formulations to less than 0.01 ppm (with occasional batches as high as 0.05 ppm), a level believed to be non-hazardous to humans and other organisms. Data relating to an acceptable maximum level are presently under regulatory review by FIFRA (Federal Insecticide, Fungicide and Rodenticide Act); the recommended maximum concentration is now placed at 0.1 ppm.

Commercial formulations of 2,4,5-T and 2,4-D were used safely in agriculture for over thirty years with no recognizable evidence of carcinogenicity or birth defects in humans. Those adverse effects that did occur from massive doses of either pure 2,4-D or pure 2,4,5-T were manifested soon after the exposure, and the victims recovered with no signs of long-term damage. When the first symptoms were to present months or years after the last exposure to these compounds, it was evident another causal agent had to be responsible. That agent was later suspected to be TCDD. Though closely related chemically to 2,4,5-T, 2,4-D is not generally contaminated with TCDD.

BIOLOGICAL EFFECTS

Two of the more pronounced biological effects of some of the clorinated dioxins are their tendency to cause chloracne (especially, in the rabbit, nude mouse, monkey and man) and the accumulation of fluid (ascites) in the pericardium and peritoneal cavity of chicks.

Chloracne in man is typified by comedones in a malar distribution; the pre- and postauricular portions are often accompanied by hirsutism and sometimes by melanosis and a secondary inflammation. The disease was first described in 1899; its cause was discovered in 1918 to be due to contact with certain clorinated hydrocarbons. Chloracne has now become one of the more common forms of occupational dermatitis. Other acute toxic reactions to dioxin include liver and renal damage, porphyria cutanea tarda, hyperpigmentation, hirsutism, polyneuropathies (e.g., sensory impairments and weakness in lower extremities) and neurasthenic or depressive syndromes. Thus far, long-term effects, except for persistent chloracne, have not been seen.

Chloracne is not caused by 2,4,5-T and 2,4-D per se; if the condition occurs upon exposure to either or both of these compounds, it is most likely that the contaminant TCDD is responsible. Chloracne usually appears within 2 to 3 weeks after the first exposure. Mild chloracne clears up within several months after cessation of exposure; severe chloracne, on the other hand, has been known to persist for as long as thirty years following the last exposure. Persons more responsive are those who are prone to develop acne vulgaris. If one's exposure to TCDD is severe enough, cysts form and, on occasion, inflammation and scarring will occur. If there is no medical history of chloracne, then the likelihood of a significant exposure to, or adverse health effects from, TCDD is remote. Hence, chloracne is the clinical marker of TCDD exposure.

Chronic exposures to TCDD lead to degeneration of the liver and thymus in experimental animals; one sensitive index of exposure is atrophy of the thymus. Porphyria, altered levels in serum enzymes and weight loss are also observed. Major organs to be affected are the liver, blood forming organs and the reticulo-endothelial system. Progressive weight loss, the first clinical sign of toxicity in the monkey, may be accompanied by alopecia, facial edema and a dry, scaly dermatitis over the rest of the body.

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*Sodium cacodylate is the sodium salt of cacodylic acid (hydroxydimethylarsine oxide)

The metabolism of TCDD in man is unknown, and for the present there is only limited information available on the metabolic pathways and metabolites that may occur in other mammals. TCDD is distributed equally among the fat and liver of mammals, to a lesser extent in the kidneys, and is eliminated via the feces. Samples of fat from beef cattle and samples of milk from cows that had grazed on 2,4,5-T-treated pasture or rangeland, in addition to human milk from an area where 2,4,5-T herbicides were used repeatedly over a period of twenty years, had small to undetectable (not more than 10 ppt) amounts of TCDD.

No clearly defined mutagenic effect has been observed in vitro with TCDD. TCDD does induce genetic changes by the Ames test with S typhimurium and E coli but not with repair-defective strains; there is no evidence (from dominant lethal and cytogenetic evaluations in rodents) that such changes occur in whole animals.

Of perhaps more relevance to man are the in vitro studies on mammalian cells – i.e., HeLa; Balb-3T3, normal mouse fibroblasts; SV101, virus (SV40) – transformed 3T3 mouse fibroblasts; human foreskin fibroblasts; and normal human lymphocytes. No significant growth inhibition in the cell cultures nor discernible ultrastructural changes have been observed by electron microscopy.

The teratogenicity and fetotoxicity of TCDD were discovered in 1969, in the course of a study on the biological activity of 2,4,5-T. The sample being used was later found to be contaminated with TCDD. The incidence of cleft palate was greater in two particular mouse strains (C57BL/6 and AKR), while the C57BL/6 mouse and the rat developed a higher incidence of cystic kidney. All doses given the rat led to gastrointestinal hemorrhage in the fetus. The increased ratio of fetal liver to body weight in the mouse suggested that TCDD was fetotoxic in this particular species.

A majority of studies using high doses of 2,4,5-T with 0.1 ppm of TCDD or less showed cleft palate in mice, but no other species, and embryotoxicity in the mouse, rat, hamster, sheep, monkey and rabbit. There is no scientific evidence that 2,4-D, 2,4,5-T or TCDD has caused reproductive difficulties or hazards in the human. No conclusive evidence is yet available that phenoxy herbicides or TCDD are mutagenic or teratogenic in man.

TCDD can induce cancer or serve as a cancer promoter in some strains of rats and mice. In contrast to some other chemical carcinogens, the carcinogenicity is always accompanied by considerable systemic toxicity.

From an environmental view, TCDD breaks down rapidly on leaves of plants, in water and on the surface of soil, especially through the action of sunlight. In soil it generally has a half-life of about 230 days; some soil microorganisms can degrade it, especially if other chlorinated hydrocarbons are present.

EXPERIENCE IN MAN

One of the most extensive human experiences with the adverse effects of TCDD in man involves the residents of Seveso, Italy. In July of 1976, TCDD was accidentally released from the ICMESA* trichlorophenol synthesis plant when a safety disk in a steam-heated reactor vessel ruptured. The plume of reactor contents, including TCDD, rose 160 feet above the factory and fell in a cone-shaped pattern about a mile long and a half-mile wide. This is the largest single population to have been exposed to the compound. Over 37,000 persons were potentially exposed to varying doses.

Two years after the incident occurred, the acute and mid-term health effects were assessed; the mild chloracne, which occurred mainly in a small group of children, healed quickly. Subclinical peripheral nerve impairment was reported; there was also some liver involvement, but without apparent functional disorder. Neither immunoresponse nor susceptibility to infectious diseases was altered.

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*Industrie Chimiche Meda Societa Anonima

The most recent progress report on the long-term epidemiologic survey of the residents of the Seveso area emphasizes the preliminary nature of their findings and reiterates the conclusions of prior investigators. Except for the skin, no organs or body functions were impaired. No derangement of gestation, no fetal lethality and loss, no gross malformations, no growth retardation at term and no cytogenetic abnormalities have yet occurred.

The first of several accidental releases of TCDD and other dioxins, attending the manufacture of 2,4,5-trichlorophenol (TCP) or 2,4,5-T occurred in 1949 in the United States. At least 11 other industrial accidents or exposure incidents have occurred since then, both here and abroad. To date, an estimated 579 workers are known to have been exposed, including 156 employees in the ICMESA plant at Seveso.

CURRENT STUDIES

There are now a number of studies underway by agencies of the U. S. government and industry, which may resolve questions on the kinds and extent of human damage from exposure to low levels of TCDD:

> Through its Department of Environmental and Drug-Induced Pathology, the Armed Forces Institute of Pathology (AFIP) is examining biopsy and autopsy tissue of all Vietnam veterans. To date, only 152 cases have been assessed: the dominant diseases are epidermal inclusion cysts and chronic, non-specific dermatitis. If any malignancies were to have been induced by TCDD, they should be appearing by now, yet there have been no unusual morphological features nor clustering of tumors by diagnosis or site as to implicate Agent Orange.
>
> A soft-tissue sarcoma study also has been proposed that will be conducted jointly by the Armed Forces Institute of Pathology and the National Cancer Institute.
>
> The Air Force, through Project Ranch Hand, will administer and examine the 1,200 personnel who were involved in the actual handling and spraying of Agent Orange. They, and the control population of 20,000, are to be followed over the next twenty years.
>
> The University of California, Los Angeles, was awarded a contract by the Veterans Administration for the design of an epidemiologic study of Vietnam veterans.
>
> Approximately 45,000 Vietnam veterans who expressed concern about the hazards of Agent Orange have been examined by the VA; data on 25,000 of these men have been placed in a special Agent Orange Registry, which may serve later to identify them and to provide medical information as well as indications of health trends over the long term.
>
> The Chloracne Task Force was established to sift out those cases of dermatitis that either resemble or are truly chloracne. Cases of the former type will be reexamined by dermatologists who have an expert knowledge of the disease. Thus far, there are only 700 cases of "skin conditions" out of the total of 3,500 filed claims for damage from Agent Orange.

RECOMMENDATIONS:

The Council on Scientific Affairs recommends that:

1. The above studies on exposed, or allegedly exposed, persons continue to be supported and, if feasible, enlarged to include the cooperative engagement of all internationally known exposure data, as recommended by the International Agency for Research on Cancer (IARC).

2. All physicians be alerted through AMA publications to the classical signs of chloracne and the possible signs and adverse effects of TCDD exposure. They should be encouraged to enlist in the present efforts to identify and treat those persons who have had serious exposures to TCDD, and to cooperate in the collection of vital information that is needed for the ongoing human epidemiologic studies.

James

AVAILABILITY OF POLLUTION LIABILITY INSURANCE UNDER PROPOSED STATE SUPERFUND LEGISLATION

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March 25, 1983

AVAILABILITY OF POLLUTION LIABILITY INSURANCE UNDER PROPOSED STATE SUPERFUND LEGISLATION

A. INTRODUCTION

This report considers the impact which enactment of proposed state legislation to establish environmental response, cleanup and liability programs similar to the federal Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (Superfund) may have on the availability and cost of insurance coverage for pollution liability. More specifically, Minnesota Bill H.F. No. 76, known as the "Environmental Response and Liability Act" was reviewed to determine whether its provisions create new sources of potential liability which differ sufficiently from those which exist under current state and federal environmental laws and from those present under common law to present a problem from the standpoint of insurability. Of course, the issue of whether insurance coverage can be obtained for a given risk is only one of the many factors which the legislature considers in deciding whether a particular piece of legislation should or should not be enacted. But state congressmen are sensitive to the impact which proposed legislation will have on business and industry within the state and in light of the current depressed economy, they do not want to enact measures which will cause their state to lose industry to other regions where less restrictive laws apply. If a certain piece of legislation contains provisions which could result in substantial liability to industry, the legislature will be interested in whether the potential liability can be managed by the purchase of insurance coverage.

H.F. No. 76, if enacted, is legislation which could result in substantial liability on the part of those industries affected by its provisions. This group of affected industries is relatively large since it includes any organization or entity that uses, generates, stores, treats or disposes of hazardous substances or is otherwise responsible for the release of such materials into the environment. The potential liability could also be relatively large since H.F. No. 76 provides for the cleanup of spilled materials, the payment of claims for personal injury and property damage, including claims for the loss of use of property and loss of profits, and relocation of persons threatened by a release. The recent cases involving pollution liability indicate that the exposure from such claims can run into hundreds of millions of dollars.

The question of what impact proposed legislation may have upon the availability of pollution liability insurance is not an easy one to address. Regardless of what logical process one applies to make such a determination, there are factors which affect the marketplace which cannot be adequately considered in advance of the actual enactment of the new law. The insurance market is not static, changes in interest rates, underwriting losses and natural disasters may change the operating philosophies of the insurance companies overnight. With the existing "soft" market for most liability coverages, it may be possible to insure certain risks today, but not be able to guarantee the availability of insurance in the future.

In considering whether Minnesota H.F. No. 76 would have a significant impact on the ability of industry within the state to purchase pollution liability insurance, it seemed logical to consider how much "new" liability would be created by enactment of this legislation and to analyze the factors considered by underwriters in offering environmental damage coverages to see which might be affected by the provisions of the proposed legislation. One way to evaluate the "new" liability is to compare Minnesota H.F. No. 76 with the federal Superfund legislation, which has been in effect since 1980. This is done in Section B of this report which also notes the liability which exists at common law where it would apply in addition to or instead of Superfund.

It is important to keep in mind that not all liability created by Superfund is currently insurable under Comprehensive general liability (sudden) or environmental impairment liability (gradual) insurance policies. Noteworthy exclusions include the cost of cleanup of non-owned sites where the insured has disposed of materials and damage to or cleanup of the insured's premises. It is important to recognize that insurance cannot solve all of the problems associated with the disposal of hazardous substances. The current crisis in waste disposal in this country is a complex problem brought about by many factors including decades of improper disposal practices. These problems will only be resolved by decades of attention to correcting past errors and a significant financial commitment to restoring what has already been damaged. Insurance for pollution liability may be a part of the solution to these problems, but it obviously is only a small part.

B. DIFFERENCES BETWEEN NATIONAL "SUPERFUND" AND MINNESOTA ENVIRONMENTAL RESPONSE & LIABILITY ACT (H.F. NO. 76)

1. Of greatest significance is the fact that H.F. No. 76 creates a cause of action by third parties alleging property damage, personal injury and environmental damage as a result of a

release or threatened release of a hazardous substance, pollutant or contaminant by a responsible person. Under "Superfund", the government may cleanup a spill or site from which a hazardous substance is released (or where a release is threatened) and recover the costs of removal and remedial actions but there is no new right on the part of third persons to bring an action against the person responsible for the release.

a. Section 5 of H.F. No. 76 allows recovery of the following:

  1.) Actual economic loss, including:

    a.) Injury to or destruction of real or personal property, including relocation costs;

    b.) Loss of use of real or personal property; and

    c.) Loss of past or future profits resulting from injury to or destruction of real or personal property. (Without regard to ownership).

2.) All damages for death or personal injury or disease, including:

a.) Medical expenses, rehabilitation, costs of burial expenses;

b.) Loss of past or future profits resulting from personal injury or disease; and

c.) Damages for physical impairment, loss of earning capacity or other pain and suffering.

b. These costs may, of course, be sought in a common law action, but the liability would not necessarily be strict, joint and several as is provided for in Subdivision 1 of Section 5.

2. The recovery of removal and remedial costs provided for in Section 4 of H.F. No. 76 is stated to be strict, joint and

several. Under Superfund, the EPA has been operating under the assumption that liability is strict, joint and several, but this is not specifically stated.

a. The "persons responsible" section of H.F. No. 76 (Section 3, Sub. 1) appears to be equivalent to the liability section of the Superfund Legislation (Section 107 (a)) in other respects.

b. Under Superfund, Section 107 (c) (1) limits liability of responsible persons, except for removal costs, to amounts specified - $50,000,000 for most land-based treatment, storage or disposal facilities. The damages to natural resources and economic damages, including loss of use of property and loss of profits are unlimited under H.F. No. 76, as well as remedial and removal costs.

1.) From a practical standpoint, the common law cases seeking recovery for environmental damage are not limited either. For example, if all claims being sought against Hooker Chemical for releases at Niagara Falls, New York were successful, the company would owe $26.4 billion to third parties claiming personal injury, property damage and other economic or non-economic loss. There are, of course, ways to insure risks which can produce unlimited liability through specifying limits and self-insured retentions in the policy which are acceptable to the underwriters.

2.) Defenses available to a responsible party (Act of God, act of war or act of a third party) are the same under H.F. No. 76 (Section 4, Sub. 6) as under Superfund (Section 107 (b)).

3. Section 107 (c) (3) of Superfund allows imposition of punitive damages in an amount up to three times the costs incurred by the Fund as a result of any release or threatened release where the responsible person fails to provide removal or remedial action upon order of the President. Section 17 of H.F. No. 76 provides for a civil penalty of not more than $10,000 per day for each day that the responsible person

fails to take response actions or fails to make reasonable progress in completing response actions requested.

4. H.F. No. 76 provides a retroactive cut-off date, making the liability provisions applicable only to a release or threatened release which occurs on or after July 17 1983, including a release which began before that date and continues afterwards. (Section 13). There is also a limitation on the application of strict liability for releases where the responsible person can establish that the hazardous substance involved was placed in the facility on or before April 1, 1982 and that the imposition of strict liability would be unfair. These limitations are not especially helpful from the standpoint of underwriters considering insuring this risk since most gradual releases are continuous and when they result in a claim are still continuing. Even where strict liability is not applicable, liability based on common law grounds and subject to normal defenses may still be established. This potentially extends the underwriters' liability far back to past operations when the risk was neither clearly understood nor adequately evaluated.

a. This backward extension of liability for past operations under H.F. No. 76 is no different from that

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under Superfund, except that the liability under H.F. No. 76 includes third party actions which are not provided for under Superfund.

b. There is a potential problem for CGL underwriters under H.F. No. 76 for claims brought under an occurrence-basis policy where the causal connection between an exposure and injury was not established at the time of a sudden and accidental release, but is later determined. With the statute of limitations provision of H.F. No. 76 (Section 10) allowing a claim brought within six years from when the plaintiff discovers a causal connection between the injury, disease or loss and the release of a hazardous substance, this could, for example, result in a claim against the underwriter on an occurrence-basis CGL policy where a release occurred in 1965 or before, but the disease does not manifest itself until 1980 or later and a causal connection is not established until 1983. The claim could be brought any time through 1989.

c. Gradual pollution policies (EIL) are being offered only on a claims-made basis, which means that the current insurer accepts responsibility for all claims which manifest themselves after inception of the current policy period, regardless of when the release took place so long as it continues into the policy period. The liability provisions of H.F. No. 76 should not be any more difficult to underwrite than common law gradual pollution liability, at least with respect to the retroactive liability feature.

d. "Remedy" and "remedial action" definitions are the same in H.F. No. 76 (Section 2, Sub. 14) and Superfund (Section 101, Paragraph 24). Both provide for remedial actions including storage, confinement, perimeter protection, clay cover, neutralization, cleanup, recycling, reuse, etc., and the permanent relocation of residents, businesses and community facilities.

e. "Remove" and "removal activities" under both H.F. No. 76 (Section 2, Sub. 15) and Superfund (Section 101, Paragraph 23) include provisions of alternative water supplies in addition to cleanup and removal of released hazardous substances. In both, this liability is unlimited.

5. The "Apportionment" provision of Section 8 allows a person found jointly and severally liable under H.F. No. 76 to seek apportionment from other parties who may also be liable for a release or threatened release. This is not addressed specifically in Superfund since strict liability is not a part of the Superfund legislation.

   There is nothing new about the concept of apportionment. It is a common law principle which is allowed in any case where joint and several liability is found and a recovery is sought by the claimant from one or more, but not all parties found liable. This feature of H.F. No. 76 should not have a negative impact on the interest of underwriters in offering gradual pollution coverage to risks in Minnesota.

6. Section 7 of H.F. No. 76 prohibits a directed verdict on the issue of causation and permits the trier of fact to draw the inference that death, injury or disease was caused or significantly contributed to by the release of a hazardous substance if it is established that:

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a. the substance was released from a facility under circumstances which could reasonably result in exposure of the person to the substance;

b. the person was exposed to a hazardous substance which is the same as that released from the facility;

c. the person suffered death, injury or disease which is known to be caused or significantly contributed to by the hazardous substance to which the person was exposed and by the amount and duration of the exposure.

While this provision has no parallel in Superfund, it should not present a serious problem for liability underwriters considering offering gradual pollution coverage in Minnesota. * The provision only prohibits a directed verdict on the issue of causation where the conditions outlined are met and allows, but does not require an inference that there is a causal link between the release of a hazardous substance and death, injury or disease where the other conditions are present. Since Paragraph (c) requires that the death, injury or disease is known to be caused or significantly contributed

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to by the substance involved in concentrations and for durations actually encountered, the applicability of Section 7 should be quite limited. Most diseases, injuries and deaths are only thought to be caused or contributed to by exposures to toxic substances and the proof of causation in fact is not an easy burdle to overcome.

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7. Section 15, Subdivision 2 requires recording a special notice of potential hazards on the title of any real property where hazardous substances have been deposited. The use of such land is also restricted to activities which do not disturb the integrity of the storage facility. This provision provides for civil penalties of up to $100,000 for violations of the recording requirement and makes the property owner, who fails to record an affadavit, responsible for any release or threatened release from the facility.

   a. This provides potentially new causes of action for property owners with respect to alienated properties. There have, however, already been actions in which real estate contracts have been rescinded as a result of an owner's failure to disclose that the site had been used for disposal of hazardous substances. The common law grounds for such an action include breach of an implied warranty of fitness for a specific purpose and negligent misrepresentation, as well as willfull concealment of material information concerning the site.

C. ANALYSIS OF FACTORS CONSIDERED IN OFFERING INSURANCE FOR ENVIRONMENTAL EXPOSURES INCLUDING THE LIABILITY ARISING UNDER THE PROVISIONS OF MINNESOTA H.F. NO. 76

Underwriters considering the insuring of environmental exposures associated with either the gradual or sudden release of harmful substances, evaluate the following factors to determine whether a risk is insurable and, if it is, what such coverage will cost:

1. Type and quantity of hazardous substances used, generated, treated, stored or disposed of by the prospective insured;
2. How these materials are being used, stored, transported, transferred and disposed of;
3. Whether operations involving hazardous materials are properly segregated from other non-hazardous activities;
4. Whether employees are well trained in how to handle hazardous

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substances and in responding to emergency situations involving a release or threatened release;

5. The attention given to engineering factors designed to prevent a release and to contain materials which may be released;

6. Attention given to preplanning for emergencies involving hazardous substances, including the availability of neutralizing and absorbing materials;

7. Past history of the prospect with respect to environmental claims and violations of environmental regulations;

8. Environmental pathways by which harmful substances can migrate from the insured premises to the property of others; and

9. The nature and proximity of the surrounding population and its sensitivity to damage as a result of exposure to the materials used or generated by the insured.

Whether liability arises under common law theories of negligence, nuisance, trespass and use of ultrahazardous substances or any of] * the dozens of statutes designed to protect the environment is not the major consideration of the underwriter. A well-run facility that scores high in the analysis of the factors outlined above will, in all probability, be insurable now and in the future. On the other hand, a facility which cannot demonstrate a high level of concern for protection of the environment, attention to engineering details and sound management may not currently be insurable and will most probably not be in the future (or will be able to purchase coverage only at a cost which will be prohibitive). The enactment of state legislation such as Minnesota Bill H.F. No. 76 will not, in itself, make insurance coverage unavailable to all risks within the state. It may, however, enter into the underwriter's consideration of a specific facility, and may have an effect on the availability or price of coverage for that site.

Of special concern in this matter of insurability is the commercial waste disposal facility. The limitations of liability for persons involved in transporting household refuse (Section 4, Sub. 4 and Section 5, Sub. 3) and for municipalities (Section 26) will help to make environmental impairment coverage available for private parties and governmental agencies involved in disposal of domestic refuse where landfill or incinerator facilities are well designed and well maintained. There will, however, still be a problem in insuring facilities which have been in operation for many years and where current monitoring cannot assure underwriters that no harmful substances are migrating from the site. This should not be affected in a significant manner by the liability provisions of H.F. No. 76, since pollution insurance for these facilities is difficult to obtain even in the absence of a state superfund law.

Hazardous waste disposal, treatment or storage facilities will have even more stringent engineering requirements placed upon them as prerequisites to obtaining pollution insurance than domestic waste disposal sites. The liability of these facilities will be insurable only where the owners/operators can assure underwriters that the operation is well designed, operated in accordance with acceptable engineering practices and maintained in like-new condition. Insurance for these facilities will be relatively expensive, but it would be even in the absence of H.F. No. 76. Underwriters of gradual pollution coverage for these facilities are probably equipped to properly evaluate the risk of claims arising from a release from the site. There may, in the future, be a much more careful review of the hazardous waste facility by the CGL underwriter who provides coverage for sudden and accidental releases than has been attempted in the past, and it may be necessary to provide this coverage on a claims-made rather than an occurrence basis to assure an adequate market for the risk.

For generators of hazardous substances, the passage of H.F. No. 76 will not have as much of an impact on the availability or cost of gradual or sudden and accidental pollution liability insurance as will be experienced by hazardous waste treatment, storage and disposal facilities. This is primarily because of the exclusion of coverage in the gradual pollution policy for the cost of cleanup of third party sites (waste disposal facilities owned by others) and of the cost of cleanup of the insured's own facilities. With these items excluded, the risks which are being transferred to the underwriter are much easier to deal with and much more like those which already exist at comon law than is the case with a facility which is in the business of treating, storing or disposing of hazardous wastes.

D. CONCLUSION

All of this theoretical analysis of what may occur if a certain piece of legislation is enacted is of interest to public officials who are considering how to encourage sound management of operations which use hazardous substances and to businessmen who must live with the rules which are created to respond to the public's desire for a safe and pleasant environment. No worthwhile objective such as those sought by S.F. No. 76 can be accomplished wihtout a cost - ultimately borne, not by the state or business, but by the consumers of the products of our industrial enterprises. Insurance cannot provide a complete assurance that citizens of a state will be protected against the consequences of releases of harmful substances into the environment or that the companies who are responsible for a release will be able to pay for all of the damages which may result from their operations. The cost of such assurances can only be guarenteed by the public itself, when that public places a higher value on a clean environment than on other amenities which compete for the limited resources available.

Insurance for environmental liability can, at best, only attempt to redistribute the costs associated with releases of hazardous substances among those who are subject to the risk of such release in a way which allows any one or more of these entities to absorb the cost of a loss without suffering ruinous financial consequences. Collectively, all such companies which have a hazardous materials exposure will spend more to transfer this risk to insurers than they would to pay the costs as they are incurred, but the sharing mechanism will allow the substitution of a known current cost for uncertain future liability. This, like all other insurable risks, will be of interest to underwriters only if they can take in more in premiums than they pay out in losses and expenses.

ENVIRONMENTAL RESPONSE FUND

CHAPTER 121

H.F.No. 76

An Act relating to the environment; establishing an environmental response, compensation and compliance fund to pay for removal and remedial action associated with certain hazardous substances

Underscoring and strikeouts are as shown in enrolled act

released into the environment and for other purposes; providing for liability for cleanup costs, personal injury, economic loss, and damage to natural resources resulting from releases of hazardous substances; providing for studies; imposing taxes, fees, and penalties; providing for injunctive relief; appropriating money; amending Minnesota Statutes 1982, sections 115A.24, subdivision 1; 466.01, by adding a subdivision; and 466.04, subdivision 1; proposing new law coded in Minnesota Statutes, chapter 116; proposing new law coded as Minnesota Statutes, chapter 115B; repealing Minnesota Statutes 1982, section 115A.24, subdivision 2.

Be it enacted by the Legislature of the State of Minnesota:

Section 1.

115B.01. Citation

Sections 115B.01 to 115B.24 may be cited as the Environmental Response and Liability Act.

Sec. 2.

115B.02. Definitions

Subdivision 1. **Application.** For the purposes of sections 115B.01 to 115B.20, the following terms have the meanings given them.

Subd. 2. **Act of God.** "Act of God" means an unanticipated grave natural disaster or other natural phenomenon of an exceptional, inevitable, and irresistible character, the effects of which could not have been prevented or avoided by the exercise of due care or foresight.

Subd. 3. **Agency.** "Agency" means the pollution control agency.

Subd. 4. **Director.** "Director" means the director of the pollution control agency.

Subd. 5. **Facility.** "Facility" means:

(a) Any building, structure, installation, equipment, pipe or pipeline (including any pipe into a sewer or publicly owned treatment works), well, pit, pond, lagoon, impoundment, ditch, landfill, storage container, motor vehicle, rolling stock, or aircraft;

(b) Any watercraft of any description, or other artificial contrivance used or capable of being used as a means of transportation on water; or

(c) Any site or area where a hazardous substance, or a pollutant or contaminant, has been deposited, stored, disposed of, or placed, or otherwise come to be located.

"Facility" does not include any consumer product in consumer use.

Subd. 6. **Federal superfund act.** "Federal Superfund Act" means the Comprehensive Environmental Response, Compensation and Liability Act of 1980, 42 U.S.C. Section 9601 et seq.

Subd. 7. **Fund.** "Fund" means the environmental response, compensation and compliance fund established under section 20.

Subd. 8. Hazardous substance. "Hazardous substance" means:

(a) Any commercial chemical designated pursuant to the Federal Water Pollution Control Act, under 33 U.S.C. Section 1321(b)(2)(A);

(b) Any hazardous air pollutant listed pursuant to the Clean Air Act, under 42 U.S.C. Section 7412; and

(c) Any hazardous waste.

"Hazardous substance" does not include natural gas, natural gas liquids, liquefied natural gas, synthetic gas usable for fuel, or mixtures of such synthetic gas and natural gas, nor does it include petroleum, including crude oil or any fraction thereof which is not otherwise a hazardous waste.

Subd. 9. Hazardous waste. "Hazardous waste" means:

(a) Any hazardous waste as defined in section 116.06, subdivision 13, and any substance identified as a hazardous waste pursuant to rules adopted by the agency under section 116.07; and

(b) Any hazardous waste as defined in the Resource Conservation and Recovery Act, under 42 U.S.C. Section 6903, which is listed or has the characteristics identified under 42 U.S.C. Section 6921, not including any hazardous waste the regulation of which has been suspended by act of Congress.

Subd. 10. Natural resources. "Natural resources" has the meaning given it in section 116B.02, subdivision 4.

Subd. 11. Owner of real property. "Owner of real property" means a person who is in possession of, has the right of control, or controls the use of real property, including without limitation a person who may be a fee owner, lessee, renter, tenant, lessor, contract for deed vendee, licensor, licensee, or occupant; provided that:

(1) A lessor of real property under a lease which in substance is a financing device and is treated as such under the United States Internal Revenue Code, common law, or statute, is not an owner of the real property;

(2) A public utility holding a public utility easement is an owner of the real property described in the easement only for the purpose of carrying out the specific use for which the easement was granted; and

(3) Any person holding a remainder or other nonpossessory interest or estate in real property is an owner of the real property beginning when that person's interest or estate in the real property vests in possession or that person obtains the unconditioned right to possession, or to control the use of, the real property.

Subd. 12. Person. "Person" means any individual, partnership, association, public or private corporation or other entity including the United States government, any interstate body, the state and any agency, department or political subdivision of the state.

Subd. 13. **Pollutant or contaminant.** "Pollutant or contaminant" means any element, substance, compound, mixture, or agent, other than a hazardous substance, which after release from a facility and upon exposure of, ingestion, inhalation, or assimilation into any organism, either directly from the environment or indirectly by ingestion through food chains, will or may reasonably be anticipated to cause death, disease, behavioral abnormalities, cancer, genetic mutation, physiological malfunctions (including malfunctions in reproduction) or physical deformations, in the organisms or their offspring.

"Pollutant or contaminant" does not include natural gas, natural gas liquids, liquefied natural gas, synthetic gas usable for fuel, or mixtures of such synthetic gas and natural gas.

Subd. 14. **Public utility easement.** "Public utility easement" means an easement used for the purposes of transmission, distribution, or furnishing, at wholesale or retail, natural or manufactured gas, or electric or telephone service, by a public utility as defined in section 216B.02, subdivision 4, a cooperative electric association organized under the provisions of chapter 308, a telephone company as defined in section 237.01, subdivisions 2 and 8, or a municipality producing or furnishing gas, electric, or telephone service.

Subd. 15. **Release.** "Release" means any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment which occurred at a point in time or which continues to occur.

"Release" does not include:

(a) Emissions from the engine exhaust of a motor vehicle, rolling stock, aircraft, watercraft, or pipeline pumping station engine;

(b) Release of source, byproduct, or special nuclear material from a nuclear incident, as those terms are defined in the Atomic Energy Act of 1954, under 42 U.S.C. Section 2014, if the release is subject to requirements with respect to financial protection established by the federal nuclear regulatory commission under 42 U.S.C. Section 2210;

(c) Release of source, byproduct or special nuclear material from any processing site designated pursuant to the Uranium Mill Tailings Radiation Control Act of 1978, under 42 U.S.C. Section 7912(a)(1) or 7942(a); or

(d) Any release resulting from the application of fertilizer or agricultural or silvicultural chemicals, or disposal of emptied pesticide containers or residues from a pesticide as defined in section 18A.21, subdivision 25.

Subd. 16. **Remedy or remedial action.** "Remedy" or "remedial action" means those actions consistent with permanent remedy taken instead of or in addition to removal actions in the event of a release or threatened release of a hazardous substance, or a pollutant or contaminant, into the environment, to prevent, minimize or eliminate the release in order to protect the public health or welfare or the environment.

"Remedy" or "remedial action" includes, but is not limited to:

(a) Actions at the location of the release such as storage, confinement, perimeter protection using dikes, trenches, or ditches, clay cover, neutralization, cleanup of released hazardous substances, pollutants or contaminants, or contaminated materials, recycling or reuse, diversion, destruction, segregation of reactive wastes, dredging or excavations, repair or replacement of leaking containers, collection of leachate and runoff, onsite treatment or incineration, provision of alternative water supplies, and any monitoring and maintenance reasonably required to assure that these actions protect the public health and welfare and the environment; and

(b) The costs of permanent relocation of residents and businesses and community facilities when the agency determines that, alone or in combination with other measures, relocation is more cost effective than and environmentally preferable to the transportation, storage, treatment, destruction, or secure disposition offsite of hazardous substances, or pollutants or contaminants, or may otherwise be necessary to protect the public health or welfare.

"Remedy" or "remedial action" does not include offsite transport of hazardous substances, pollutants or contaminants, or contaminated materials or their storage, treatment, destruction, or secure disposition offsite unless the agency determines that these actions:

(1) Are more cost effective than other remedial actions;

(2) Will create new capacity to manage hazardous substances in addition to those located at the affected facility, in compliance with section 116.07 and subtitle C of the Solid Waste Disposal Act, 42 U.S.C. Section 6921 et seq.; or

(3) Are necessary to protect the public health or welfare or the environment from a present or potential risk which may be created by further exposure to the continued presence of the hazardous substances, pollutants or contaminants, or contaminated materials.

Subd. 17. Remove or removal. "Remove" or "removal" means:

(a) The cleanup or removal of a released hazardous substance, or a pollutant or contaminant, from the environment;

(b) Necessary actions taken in the event of a threatened release of a hazardous substance, or a pollutant or contaminant, into the environment;

(c) Actions necessary to monitor, test, analyze, and evaluate a release or threatened release of a hazardous substance, or a pollutant or contaminant;

(d) Disposal or processing of removed material; or

(e) Other actions necessary to prevent, minimize, or mitigate damage to the public health or welfare or the environment, which may otherwise result from a release or threatened release.

"Remove" or "removal" includes, but is not limited to, security fencing or other measures to limit access, provision of alternative water supplies, temporary evacuation and housing of threatened individuals not otherwise provided for, action taken pursuant to the Federal Superfund Act, under 42

U.S.C. Section 9604(b), and any emergency assistance which may be provided under the Disaster Relief Act of 1974, 42 U.S.C. Section 5121 et seq.

Subd. 18. Respond or response. "Respond" or "response" means remove, removal, remedy, and remedial action.

Subd. 19. Water. "Water" has the meaning given to the term "waters of the state" in section 115.01, subdivision 9.

Sec. 3.

115B.03. Responsible person

Subdivision 1. General rule. For the purposes of sections 115B.01 to 115B.20, and except as provided in subdivisions 2 and 3, a person is responsible for a release or threatened release of a hazardous substance, or a pollutant or contaminant, from a facility if the person:

(a) Owned or operated the facility: (1) when the hazardous substance, or pollutant or contaminant, was placed or came to be located in or on the facility; (2) when the hazardous substance, or pollutant or contaminant, was located in or on the facility but before the release; or (3) during the time of the release or threatened release;

(b) Owned or possessed the hazardous substance, or pollutant or contaminant, and arranged, by contract, agreement or otherwise, for the disposal, treatment or transport for disposal or treatment of the hazardous substance, or pollutant or contaminant; or

(c) Knew or reasonably should have known that waste be accepted for transport to a disposal or treatment facility contained a hazardous substance, or pollutant or contaminant, and either selected the facility to which it was transported or disposed of it in a manner contrary to law.

Subd. 2. Employees and employers. When a person who is responsible for a release or threatened release as provided in subdivision 1 is an employee who is acting in the scope of his employment:

(a) The employee is subject to liability under section 115B.04 or 115B.05 only if his conduct with respect to the hazardous substance was negligent under circumstances in which he knew that the substance was hazardous and that his conduct, if negligent, could result in serious harm.

(b) His employer shall be considered a person responsible for the release or threatened release and is subject to liability under section 115B.04 or 115B.05 regardless of the degree of care exercised by the employee.

Subd. 3. Owner of real property. An owner of real property is not a person responsible for the release or threatened release of a hazardous substance from a facility in or on the property unless that person:

(a) was engaged in the business of generating, transporting, storing, treating, or disposing of a hazardous substance at the facility or disposing of waste at the facility, or knowingly permitted others to engage in such a business at the facility;

(b) knowingly permitted any person to make regular use of the facility for disposal of waste;

(c) knowingly permitted any person to use the facility for disposal of a hazardous substance;

(d) knew or reasonably should have known that a hazardous substance was located in or on the facility at the time right, title, or interest in the property was first acquired by the person and engaged in conduct by which he associated himself with the release; or

(e) took action which significantly contributed to the release after he knew or reasonably should have known that a hazardous substance was located in or on the facility.

For the purpose of clause (d), a written warranty, representation, or undertaking, which is set forth in an instrument conveying any right, title or interest in the real property and which is executed by the person conveying the right, title or interest, or which is set forth in any memorandum of any such instrument executed for the purpose of recording, is admissible as evidence of whether the person acquiring any right, title, or interest in the real property knew or reasonably should have known that a hazardous substance was located in or on the facility.

Any liability which accrues to an owner of real property under sections 115B.01 to 115B.15 does not accrue to any other person who is not an owner of the real property merely because the other person holds some right, title, or interest in the real property.

An owner of real property on which a public utility easement is located is not a responsible person with respect to any release caused by any act or omission of the public utility which holds the easement in carrying out the specific use for which the easement was granted.

Sec. 4.

115B.04. Liability for response costs and natural resources; limitations and defenses

Subdivision 1. **Liability.** Except as otherwise provided in subdivisions 2 to 12, and notwithstanding any other provision or rule of law, any person who is responsible for a release or threatened release of a hazardous substance from a facility is strictly liable, jointly and severally, for the following response costs and damages which result from the release or threatened release or to which the release or threatened release significantly contributes:

(a) All reasonable and necessary response costs incurred by the state, a political subdivision of the state or the United States;

(b) All reasonable and necessary removal costs incurred by any person; and

(c) All damages for any injury to, destruction of, or loss of natural resources, including the reasonable costs of assessing such injury, destruction, or loss.

Subd. 2. **Liability for pollutant or contaminant excluded.** There is no liability under this section for response costs or damages which result from the release of a pollutant or contaminant.

Subd. 3. **Liability for a threatened release.** Liability under this section for a threatened release of a hazardous substance is limited to the recovery by the agency of reasonable and necessary response costs as provided in section 115B.17, subdivision 6.

Subd. 4. **Liability of political subdivisions.** The liability of a political subdivision under this section is subject to the limits imposed under section 466.04, subdivision 1.

Subd. 5. **Transportation of household refuse.** A person who accepts only household refuse for transport to a treatment or disposal facility is not liable under this section for the release or threatened release of any hazardous substance unless he knew or reasonably should have known that the hazardous substance was present in the refuse. For the purpose of this subdivision, household refuse means garbage, trash, or septic tank sanitary wastes generated by single or multiple residences, hotels, motels, restaurants and other similar facilities.

Subd. 6. **Defense to certain claims by political subdivisions and private persons.** It is a defense to a claim by a political subdivision or private person for recovery of the costs of its response actions under this section that the hazardous substance released from the facility was placed or came to be located in or on the facility before April 1, 1982, and that the response actions of the political subdivision or private person were not authorized by the agency as provided in section 115B.17, subdivision 12. This defense applies only to response costs incurred on or after July 1, 1983.

Subd. 7. **Defense for intervening acts.** It is a defense to liability under this section that the release or threatened release was caused solely by:

(a) An act of God;

(b) An act of war;

(c) An act of vandalism or sabotage; or

(d) An act or omission of a third party or the plaintiff.

"Third party" for the purposes of clause (d) does not include an employee or agent of the defendant, or a person in the chain of responsibility for the generation, transportation, storage, treatment, or disposal of the hazardous substance.

The defenses provided in clauses (c) and (d) apply only if the defendant establishes that he exercised due care with respect to the hazardous substance concerned, taking into consideration the characteristics of the hazardous substance in light of all relevant facts and circumstances which he knew or should have known, and that he took precautions against foreseeable acts or omissions and the consequences that could foreseeably result from those acts or omissions.

Subd. 8. **Intervening acts of public agencies.** When the agency or the federal environmental protection agency assumes control over any release or threatened release of a hazardous substance by taking removal actions at the site of the release, the persons responsible for the release are not liable under sections 115B.01 to 115B.15 for any subsequent release of the hazardous substance from another facility to which it has been removed.

Subd. 9. **Releases subject to certain permits or standards; federal post-closure fund.** It is a defense to liability under this section that:

(a) The release or threatened release was from a hazardous waste facility as defined under section 115A.03, for which a permit had been issued pursuant to section 116.07 or pursuant to subtitle C of the Solid Waste Disposal Act, 42 U.S.C. Section 6921 et seq., the hazardous substance was specifically identified in the permit, and the release was within the limits allowed in the permit for release of that substance;

(b) The hazardous substance released was specifically identified in a federal or state permit and the release is within the limits allowed in the permit;

(c) The release resulted from circumstances identified and reviewed and made a part of the public record of a federal or state agency with respect to a permit issued or modified under federal or state law, and the release conformed with the permit;

(d) The release was any part of an emission or discharge into the air or water and the emission or discharge was subject to a federal or state permit and was in compliance with control rules or regulations adopted pursuant to state or federal law;

(e) The release was the introduction of any hazardous substance into a publicly owned treatment works and the substance was specified in, and is in compliance with, applicable pretreatment standards specified for that substance under state and federal law; or

(f) Liability has been assumed by the federal post-closure liability fund under 42 U.S.C. Section 9607(k).

Subd. 10. **Natural resources.** It is a defense to liability under this section, for any injury to, destruction of, or loss of natural resources that:

(a) The natural resources were specifically identified as an irreversible and irretrievable commitment of natural resources in an approved final state or federal environmental impact statement, or other comparable approved final environmental analysis for a project or facility which was the subject of a governmental permit or license; and

(b) The project or facility was being operated within the terms of its permit or license.

Subd. 11. **Rendering assistance in response actions.** It is a defense to liability under this section that the response costs or damages resulted from acts taken or omitted in preparation for, or in the course of rendering care, assistance, or advice to the director or agency pursuant to section 115B.17 or in accordance with the national hazardous substance response plan pursuant to the Federal Superfund Act, under 42 U.S.C. Section 9605, or at the direction of an on-scene coordinator appointed under that plan, with respect to any release or threatened release of a hazardous substance.

Subd. 12. **Burden of proof for defenses.** Any person claiming a defense provided in subdivisions 6 to 11 has the burden to prove all elements of the defense by a preponderance of the evidence.

Sec. 5.

115B.05. Liability for economic loss, death, personal injury and disease; limitations and defenses

Subdivision 1. **Liability.** Except as otherwise provided in subdivisions 2 to 10, and notwithstanding any other provision or rule of law, any person who is responsible for the release of a hazardous substance from a facility is strictly liable, jointly and severally, for the following damages which result from the release or to which the release significantly contributes:

(a) All damages for actual economic loss including:

(1) Any injury to, destruction of, or loss of any real or personal property, including relocation costs;

(2) Any loss of use of real or personal property;

(3) Any loss of past or future income or profits resulting from injury to, destruction of, or loss of real or personal property without regard to the ownership of the property; and

(b) All damages for death, personal injury, or disease including:

(1) Any medical expenses, rehabilitation costs or burial expenses;

(2) Any loss of past or future income, or loss of earning capacity; and

(3) Damages for pain and suffering, including physical impairment.

Subd. 2. **Liability for pollutant or contaminant excluded.** There is no liability under this section for damages which result from the release of a pollutant or contaminant.

Subd. 3. **Certain employee claims not covered.** Except for a third party who is subject to liability under section 176.061, subdivision 5, there is no liability under this section for the death, personal injury or disease of an employee which is compensable under chapter 176 as an injury or disease arising out of and in the course of employment.

Subd. 4. **Liability limitations.** The liability of a political subdivision under this section is subject to the limits imposed under section 466.04, subdivision 1.

Subd. 5. **Transportation of household refuse.** A person who accepts only household refuse for transport to a treatment or disposal facility is not liable under this section for the release or threatened release of any hazardous substance unless he knew or reasonably should have known that the hazardous substance was present in the refuse. For the purpose of this subdivision, household refuse means garbage, trash, or septic tank sanitary wastes generated by single or multiple residences, hotels, motels, restaurants and other similar facilities.

Subd. 6. **Defense for intervening acts.** It is a defense to liability under this section that the release or threatened release was caused solely by:

(a) An act of God;

(b) An act of war;

(c) An act of vandalism or sabotage; or

(d) An act or omission of a third party or the plaintiff.

"Third party" for the purposes of clause (d) does not include an employee or agent of the defendant, or a person in the chain of responsibility for the generation, transportation, storage, treatment, or disposal of the hazardous substance.

The defenses provided in clauses (c) and (d) apply only if the defendant establishes that he exercised due care with respect to the hazardous substance concerned, taking into consideration the characteristics of the hazardous substance in light of all relevant facts and circumstances which he knew or should have known, and that he took precautions against foreseeable acts or omissions and the consequences that could foreseeably result from those acts or omissions.

Subd. 7. **Intervening acts of public agencies.** When the agency or the federal environmental protection agency assumes control over any release or threatened release of a hazardous substance by taking removal actions at the site of the release, the persons responsible for the release are not liable under sections 115B.01 to 115B.15 for any subsequent release of the hazardous substance from another facility to which it has been removed.

Subd. 8. **Releases subject to certain permits or standards; federal post-closure fund.** It is a defense to liability under this section that:

(a) The release or threatened release was from a hazardous waste facility as defined under section 115A.03, for which a permit had been issued pursuant to section 116.07 or pursuant to subtitle C of the Solid Waste Disposal Act, 42 U.S.C. Section 6921 et seq., the hazardous substance was specifically identified in the permit, and the release was within the limits allowed in the permit for release of that substance;

(b) The hazardous substance released was specifically identified in a federal or state permit and the release is within the limits allowed in the permit;

(c) The release resulted from circumstances identified and reviewed and made a part of the public record of a federal or state agency with respect to a permit issued or modified under federal or state law, and the release conformed with the permit;

(d) The release was any part of an emission or discharge into the air or water and the emission or discharge was subject to a federal or state permit and was in compliance with control rules or regulations adopted pursuant to state or federal law;

(e) The release was the introduction of any hazardous substance into a publicly owned treatment works and the substance was specified in, and is in compliance with, applicable pretreatment standards specified for that substance under state and federal law; or

(f) Liability has been assumed by the federal post-closure liability fund under 42 U.S.C. Section 9607(k).

Subd. 9. Rendering assistance in response actions. It is a defense to liability under this section that the damages resulted from acts taken or omitted in preparation for, or in the course of rendering care, assistance, or advice to the director or agency pursuant to section 115B.17 or in accordance with the national hazardous substance response plan pursuant to the Federal Superfund Act, under 42 U.S.C. Section 9605, or at the direction of an on-scene coordinator appointed under that plan, with respect to any release or threatened release of a hazardous substance.

Subd. 10. Burden of proof for defenses. Any person claiming a defense provided in subdivisions 6 to 9 has the burden to prove all elements of the defense by a preponderance of the evidence.

Sec. 6.

115B.06. Application to past actions

Subdivision 1. Application of section 115B.05 and additional defense. (a) A defendant in an action under section 115B.05 has the additional defense provided in subdivision 2 for damages caused or significantly contributed to by the release of a hazardous substance from a facility if the defendant shows that the substance was placed or came to be located in or on the facility wholly before January 1, 1973.

(b) Section 115B.05 does not apply to any claim for damages arising out of the release of a hazardous substance which was placed or came to be located in or on the facility wholly before January 1, 1960.

Subd. 2. Additional defense. For a defendant who has made the showing required in subdivision 1, clause (a), it is a defense to liability under section 115B.05 that the activity by which the substance was kept, placed, or came to be located in or on the facility was not an abnormally dangerous activity. The determination of whether the activity was an abnormally dangerous activity shall be made by the court.

Sec. 7.

115B.07. Causation

In any action brought under section 115B.05 or any other law to recover damages for death, personal injury, or disease arising out of the release of a hazardous substance, the court may not direct a verdict against the plaintiff on the issue of causation if the plaintiff produces evidence sufficient to enable a reasonable person to find that:

(a) the defendant is a person who is responsible for the release;

(b) the plaintiff was exposed to the hazardous substance;

(c) the release could reasonably have resulted in plaintiff's exposure to the substance in the amount and duration experienced by the plaintiff; and

(d) the death, injury, or disease suffered by the plaintiff is caused or significantly contributed to by exposure to the hazardous substance in an amount and duration experienced by the plaintiff.

Evidence to a reasonable medical certainty that exposure to the hazardous substance caused or significantly contributed to the death, injury, or disease is not required for the question of causation to be submitted to the trier of fact.

Nothing in this section shall be construed to relieve the plaintiff of the burden of proving that the defendant is a person who is responsible for the release and of proving the causal connection between the release of the hazardous substance for which the defendant is a responsible person and the plaintiff's death, injury, or disease.

Sec. 8.

115B.08. Liability under section 115B.04; apportionment and contribution

Subdivision 1. Right of apportionment; factors. Any person held jointly and severally liable under section 115B.04 has the right at trial to have the trier of fact apportion liability among the parties as provided in this section. The burden is on each defendant to show how his liability should be apportioned. The court shall reduce the amount of damages in proportion to any amount of liability apportioned to the party recovering.

In apportioning the liability of any party under this section, the trier of fact shall consider the following:

(a) The extent to which that party's contribution to the release of a hazardous substance can be distinguished;

(b) The amount of hazardous substance involved;

(c) The degree of toxicity of the hazardous substance involved;

(d) The degree of involvement of and care exercised by the party in manufacturing, treating, transporting, and disposing of the hazardous substance;

(e) The degree of cooperation by the party with federal, state, or local officials to prevent any harm to the public health or the environment; and

(f) Knowledge by the party of the hazardous nature of the substance.

Subd. 2. Contribution. If a person is held jointly and severally liable under section 115B.04 and establishes his proportionate share of the aggregate liability, the provisions of section 604.02, subdivisions 1 and 2, shall apply with respect to contribution and reallocation of any uncollectible amounts.

Sec. 9.

115B.09. Liability under section 115B.05; comparative fault and contribution

The provisions of sections 604.01, 604.02, subdivisions 1 and 2, apply to any action for damages under section 115B.05, except that, if the percentage of fault attributable to a defendant is determined under section 604.01, the liability of the defendant shall be limited to two times that percentage of the damages recoverable in the action.

Sec. 10.

115B.10. No avoidance of liability; insurance and subrogation

An owner or operator of a facility or any other person who may be liable under sections 115B.01 to 115B.15 may not avoid that liability by means of any conveyance of any right, title, or interest in real property, or by any indemnification, hold harmless agreement, or similar agreement. Nothing in this section shall be construed:

(a) To prohibit any party who may be liable under sections 115B.01 to 115B.15 from entering an agreement by which that party is insured, held harmless or indemnified for part or all of that liability;

(b) To prohibit the enforcement of any insurance, hold harmless or indemnification agreement; or

(c) To bar any cause of action brought by a party who may be liable under sections 115B.01 to 115B.15 or by an insurer or guarantor, whether by right of subrogation or otherwise.

Sec. 11.

115B.11. Statute of limitations

No person may recover pursuant to sections 115B.01 to 115B.15 unless the action is commenced within six years from the date when the cause of action accrues. In determining when the cause of action accrues for an action to recover damages for death, personal injury or disease, the court shall consider factors including the following:

(a) When the plaintiff discovered the injury or loss;

(b) Whether a personal injury or disease had sufficiently manifested itself; and

(c) When the plaintiff discovered, or using due diligence should have discovered, a causal connection between the injury, disease, or loss and the release of a hazardous substance.

Sec. 12.

115B.12. Other remedies preserved

Nothing in sections 115B.01 to 115B.15 shall be construed to abolish or diminish any remedy or affect the right of any person to bring a legal action or use any remedy available under any other provision of state or federal law, including common law, to recover for personal injury, disease, economic loss or response costs arising out of a release of any hazardous substance, or for removal or the costs of removal of that hazardous substance. Nothing in sections 115B.01 to 115B.15 shall be construed to limit or restrict in any way the liability of any person under any other state or federal law, including common law, for loss due to personal injury or disease, for economic loss, or for response costs arising out of any release or threatened release of a hazardous substance from a facility regardless of the time at which a hazardous substance was placed or came to be located in or on the facility. The provisions of sections 115B.01 to 115B.15 shall not be considered, interpreted, or construed in any way as reflecting a determination, in whole or in part, of policy regarding the inapplicability of strict liability, or strict liability doctrines under any other state or federal law, including common law, to activities past, present or future, relating to hazardous substances, or pollutants or contaminants, or other similar activities.

Sec. 13.

115B.13. Double recovery prohibited

A person who recovers response costs or damages pursuant to sections 115B.01 to 115B.15 may not recover the same costs or damages pursuant to any other law. A person who recovers response costs or damages pursuant to any other state or federal law may not recover for the same costs or damages pursuant to sections 115B.01 to 115B.15.

Sec. 14.

115B.14. Award of costs

Upon motion of a party prevailing in an action under sections 115B.01 to 115B.15 the court may award costs, disbursements and reasonable attorney fees and witness fees to that party.

Sec. 15.

115B.15. Application of sections 115B.01 to 115B.14

Sections 115B.01 to 115B.14 apply to any release or threatened release of a hazardous substance occurring on or after July 1, 1983, including any release which began before July 1, 1983, and continued after that date. Sections 115B.01 to 115B.14 do not apply to a release or threatened release which occurred wholly before July 1, 1983, regardless of the date of discovery of any injury or loss caused by the release or threatened release.

Sec. 16.

115B.16. Disposition of facilities

Subdivision 1. **Closed disposal facilities; use of property.** No person shall use any property on or in which hazardous waste remains after closure of a disposal facility as defined in section 115A.03, subdivision 10, in any way that disturbs the integrity of the final cover, liners, or any other components of any containment system, or the function of the disposal facility's monitoring systems, unless the agency finds that the disturbance:

(a) Is necessary to the proposed use of the property, and will not increase the potential hazard to human health or the environment; or

(b) Is necessary to reduce a threat to human health or the environment.

Subd. 2. **Recording of affidavit.** Before any transfer of ownership of any property which the owner knew or should have known was used as the site of a hazardous waste disposal facility as defined in section 115A.03, subdivision 10, or which the owner knew or should have known is subject to extensive contamination by release of a hazardous substance, the owner shall record with the county recorder of the county in which the property is located an affidavit containing a legal description of the property that discloses to any potential transferee:

(a) That the land has been used to dispose of hazardous waste or that the land is contaminated by a release of a hazardous substance;

(b) The identity, quantity, location, condition and circumstances of the disposal or contamination to the full extent known or reasonably ascertainable; and

(c) That the use of the property or some portion of it may be restricted as provided in subdivision 1.

An owner must also file an affidavit within 60 days after any material change in any matter required to be disclosed under clauses (a) to (c) with respect to property for which an affidavit has already been recorded.

If the owner or any subsequent owner of the property removes the hazardous substance, together with any residues, liner, and contaminated underlying and surrounding soil, that owner may record an affidavit indicating the removal of the hazardous substance.

Failure to record an affidavit as provided in this subdivision does not affect or prevent any transfer of ownership of the property.

Subd. 3. Duty of county recorder. The county recorder shall record all affidavits presented to him in accordance with subdivision 2. The affidavits shall be recorded in a manner which will assure their disclosure in the ordinary course of a title search of the subject property.

Subd. 4. Penalties. (a) Any person who knowingly violates the provisions of subdivision 1 is subject to a civil penalty in an amount determined by the court of not more than $100,000, and shall be liable under sections 115B.04 and 115B.05 for any release or threatened release of any hazardous substance resulting from the violation.

(b) Any person who knowingly fails to record an affidavit as required by subdivision 2 shall be liable under sections 115B.04 and 115B.05 for any release or threatened release of any hazardous substance from a facility located on that property.

(c) A civil penalty may be imposed and recovered by an action brought by a county attorney or by the attorney general in the district court of the county in which the property is located.

(d) Any civil fines recovered under this subdivision shall be deposited in the fund.

Sec. 17.

115B.17. State response to releases

Subdivision 1. Removal and remedial action. Whenever there is a release or substantial threat of release from a facility of any pollutant or contaminant which presents an imminent and substantial danger to the public health or welfare or the environment or whenever a hazardous substance is released or there is a threatened release of a hazardous substance from a facility:

(a) The agency may take any removal or remedial action relating to the hazardous substance, or pollutant or contaminant, which the agency deems necessary to protect the public health or welfare or the environment. Before taking any action the agency shall:

(1) Request any responsible party known to the agency to take actions which the agency deems reasonable and necessary to protect the public health or welfare or the environment, stating the reasons for the actions, a reasonable time for beginning and completing the actions taking into account the urgency of the actions for protecting the public health or welfare or the environment, and the intention of the agency to take action if the requested actions are not taken as requested;

(2) Notify the owner of real property where the facility is located or where response actions are proposed to be taken, if the owner is not a responsible party, that responsible parties have been requested to take response actions and that the owner's cooperation will be required in order for responsible parties or the agency to take those actions; and

(3) Determine that the actions requested by the agency will not be taken by any known responsible party in the manner and within the time requested.

(b) The director may take removal action which he deems necessary to protect the public health or welfare or the environment if the director determines that the release or threatened release constitutes an emergency requiring immediate action to prevent, minimize or mitigate damage to the public health or welfare or the environment. Before taking any action the director shall make reasonable efforts in light of the urgency of the action to follow the procedure provided in clause (a).

No removal action taken by any person shall be construed as an admission of liability for a release or threatened release.

Subd. 2. Other actions. Whenever the agency or director is authorized to act pursuant to subdivision 1 or whenever the agency or director has reason to believe that a release of a hazardous substance, or a pollutant or contaminant, has occurred or is about to occur, or that illness, disease, or complaints thereof may be attributable to exposure to a hazardous substance, or a pollutant or contaminant, the agency or director may undertake investigations, monitoring, surveys, testing, and other similar activities necessary or appropriate to identify the existence and extent of the release or threat thereof, the source and nature of the hazardous substances, or pollutants or contaminants, and the extent of danger to the public health or welfare or the environment. In addition, the agency may undertake planning, legal, fiscal, economic, engineering, architectural, and other studies or investigations necessary or appropriate to plan and direct a response action, to recover the costs of the response action, and to enforce the provisions of sections 115B.01 to 115B.18.

Subd. 3. Duty to provide information. Any person who the agency has reason to believe is responsible for a release or threatened release as provided in section 115B.03, or who is the owner of real property where the release or threatened release is located or where response actions are proposed to be taken, when requested by the agency, or any member, employee or agent thereof who is authorized by the agency, shall furnish to the agency any information which he may have or may reasonably obtain which is relevant to the release or threatened release.

Subd. 4. Access to information and property. The agency or any member, employee or agent thereof authorized by the agency, upon presentation of credentials, may:

(a) Examine and copy any books, papers, records, memoranda or data of any person who has a duty to provide information to the agency under subdivision 3; and

(b) Enter upon any property, public or private, for the purpose of taking any action authorized by this section including obtaining information from any person who has a duty to provide the information under subdivision 3, conducting surveys or investigations, and taking removal or remedial action.

Subd. 5. Classification of data. Except as otherwise provided in this subdivision, data obtained from any person pursuant to subdivision 3 or 4 is public data as defined in section 13.02. Upon certification by the subject of the data that the data relates to sales figures, processes or methods of production unique to that person, or information which would tend to affect adversely the competitive position of that person, the director shall classify the data as private or nonpublic data as defined in section 13.02. Notwithstanding any other law to the contrary, data classified as private or nonpublic under this subdivision may be disclosed when relevant in any proceeding under sections 115B.01 to 115B.18, or to other public agencies concerned with the implementation of sections 115B.01 to 115B.18.

Subd. 6. Recovery of expenses. Any reasonable and necessary expenses incurred by the agency or director pursuant to this section, including all response costs, and administrative and legal expenses, may be recovered in a civil action brought by the attorney general against any person who may be liable under section 115B.04 or any other law. The agency's certification of expenses shall be prima facie evidence that the expenses are reasonable and necessary. Any expenses incurred pursuant to this section which are recovered by the attorney general pursuant to section 115B.04 or any other law, including any award of attorneys fees, shall be deposited in the fund and credited to a special account for additional response actions as provided in section 115B.20, subdivision 2, clause (b) or (d).

Subd. 7. Actions relating to natural resources. For the purpose of this subdivision, the state is the trustee of the air, water and wildlife of the state. An action pursuant to section 115B.04 for damages with respect to air, water or wildlife may be brought by the attorney general in the name of the state as trustee for those natural resources. Any damages recovered by the attorney general pursuant to section 115B.04 or any other law for injury to, destruction of, or loss of natural resources resulting from the release of a hazardous substance, or a pollutant or contaminant, shall be deposited in the fund and credited to a special account for the purposes provided in section 115B.20, subdivision 2, clause (f).

Subd. 8. Actions relating to pesticides or fertilizer or soil or plant amendments. When the commissioner of agriculture has reported an incident involving the release of pesticides under the provisions of section 18A.37 or the release of fertilizers or soil or plant amendments, and the agency determines that the incident constitutes a release of a hazardous substance, or a pollutant or contaminant, the agency shall authorize the commissioner, subject to the provisions of subdivision 13, to take any action which the agency would be authorized to take under subdivisions 1 to 4. Subject to the provisions of section 115B.20, subdivision 3, the agency shall reimburse the commissioner from the fund for the reasonable and necessary expenses incurred in taking those actions and may recover any amount spent from the fund under subdivision 6.

Subd. 9. Actions relating to occupational safety and health. The agency, director and the commissioner of labor and industry shall make reasonable efforts to coordinate any actions taken under this section and under sections 182.65 to 182.674 to avoid duplication or conflict of actions or requirements with respect to a release or threatened release affecting the safety of any conditions or place of employment.

Subd. 10. Actions relating to health. The agency and director shall make reasonable efforts to coordinate and consult with the commissioner of health in planning and directing response actions with respect to a release or threatened release affecting the public health. If the commissioner of health, upon the request of the agency, takes any actions authorized under this section, the agency shall reimburse the commissioner from the fund for the reasonable and necessary expenses incurred in taking those actions and may recover any amount spent from the fund under subdivision 6.

Subd. 11. Limit on actions by political subdivisions. When the agency or director has requested a person who is responsible for a release or threatened release to take any response action under subdivision 1, no political subdivision shall request or order that person to take any action which conflicts with the action requested by the agency or director.

Subd. 12. Authorization of certain response actions. For the purpose of permitting a political subdivision or private person to recover response costs as provided in section 115B.04, subdivision 6, the agency may authorize the political subdivision to take removal or remedial actions or may authorize the private person to take removal actions with respect to any release of a hazardous substance which was placed or came to be located in the facility before April 1, 1982. The authorization shall be based on application of the criteria in the rules of the agency adopted under subdivision 13 or, if the rules have not been adopted, under the criteria set forth in subdivision 13 on which the rules are required to be based. The authorization shall not be inconsistent with the criteria. This subdivision shall not be construed to prohibit a political subdivision or private person from taking removal or remedial actions without the authorization of the agency.

Subd. 13. Priorities; rules. By November 1, 1983, the agency shall establish a temporary list of priorities among releases or threatened releases for the purpose of taking remedial action and, to the extent practicable consistent with the urgency of the action, for taking removal action under this section. The temporary list, with any necessary modifications, shall remain in effect until the agency adopts rules establishing state criteria for determining priorities among releases and threatened releases. The agency shall adopt the rules by July 1, 1984. After rules are adopted, a permanent priority list shall be established, and may be modified from time to time, according to the criteria set forth in the rules. Before any list is established under this subdivision the agency shall publish the list in the state register and allow 30 days for comments on the list by the public.

The temporary list and the rules required by this subdivision shall be based upon the relative risk or danger to public health or welfare or the environment, taking into account to the extent possible the population at risk, the hazardous potential of the hazardous substances at the facilities, the potential for contamination of drinking water supplies, the potential for direct human contact, the potential for destruction of sensitive ecosystems, the administrative and financial capabilities of the agency, and other appropriate factors.

Sec. 18.

115B.18. Failure to take requested actions; civil penalties; action to compel performance; injunctive relief

Subdivision 1. Civil penalties. Any person responsible for a release or threatened release from a facility of a pollutant or contaminant which presents an imminent and substantial danger to the public health or welfare or the environment or for a release or threatened release of a hazardous substance from a facility shall forfeit and pay to the state a civil penalty in an amount to be determined by the court of not more than $20,000 per day for each day that the person fails to take reasonable and necessary response actions or to make reasonable progress in completing response actions requested as provided in subdivision 3.

The penalty provided under this subdivision may be recovered by an action brought by the attorney general in the name of the state in connection with an action to recover expenses of the agency under section 115B.17, subdivision 6, or by a separate action in the district court of Ramsey County. All penalties recovered under this subdivision shall be deposited in the fund.

Subd. 2. Action to compel performance. When any person who is responsible for a release or threatened release from a facility of a pollutant or contaminant which presents an imminent and substantial danger to the public health or welfare or the environment or for a release or threatened release of a hazardous substance from a facility, fails to take response actions or to make reasonable progress in completing response actions requested as provided in subdivision 3, the attorney general may bring an action in the name of the state to compel performance of the requested response actions. If any person having any right, title, or interest in and to the real property where the facility is located or where response actions are proposed to be taken is not a person responsible for the release or threatened release, the person may be joined as an indispensable party in an action to compel performance in order to assure that the requested response actions can be taken on that property by the responsible parties.

Subd. 3. Requests for response actions. A request for emergency removal action shall be made by the director. Other requests for response actions shall be made by the agency. A request shall be in writing, shall state the action requested, the reasons for the action, and a reasonable time by which the action must be begun and completed taking into account the urgency of the action for protection of the public health or welfare or the environment.

Subd. 4. Injunctive relief. The release or threatened release of a hazardous substance, or a pollutant or contaminant, shall constitute a public nuisance and may be enjoined in an action, in the name of the state, brought by the attorney general.

Sec. 19.

115B.19. Purposes of fund and taxes

In establishing the environmental response, compensation and compliance fund in section 115B.20 and imposing taxes in section 115B.22 it is the purpose of the legislature to:

(a) Encourage treatment and disposal of hazardous waste in a manner that adequately protects the public health or welfare or the environment;

(b) Encourage responsible parties to provide the response actions necessary to protect the public and the environment from the effects of the release of hazardous substances;

(c) Encourage the use of alternatives to land disposal of hazardous waste including resource recovery, recycling, neutralization, and reduction;

(d) Provide state agencies with the financial resources needed to prepare and implement an effective and timely state response to the release of hazardous substances, including investigation, planning, removal and remedial action;

(e) Compensate for increased governmental expenses and loss of revenue and to provide other appropriate assistance to mitigate any adverse impact on communities in which commercial hazardous waste processing or disposal facilities are located under the siting process provided in chapter 115A;

(f) Recognize the environmental and public health costs of land disposal of solid waste and of the use and disposal of hazardous substances and to place the burden of financing state hazardous waste management activities on those whose products and services contribute to hazardous waste management problems and increase the risks of harm to the public and the environment.

Sec. 20.

115B.20. Environmental response, compensation and compliance fund

Subdivision 1. Establishment. The environmental response, compensation and compliance fund is created as an account in the state treasury and may be spent only for the purposes provided in subdivision 2.

Subd. 2. Purposes for which money may be spent. Subject to appropriation by the legislature the money in the fund may be spent for any of the following purposes:

(a) Preparation by the agency for taking removal or remedial action under section 115B.17, including investigation, monitoring and testing activities, enforcement and compliance efforts relating to the release of hazardous substances, pollutants or contaminants under section 115B.17 or 115B.18;

(b) Removal and remedial actions taken or authorized by the agency or director under section 115B.17, including related enforcement and compliance efforts under section 115B.17 or 115B.18, and payment of the state share of the cost of remedial action which may be carried out under a cooperative agreement with the federal government pursuant to the Federal Superfund Act, under 42 U.S.C. Section 9604(c)(3) for actions related to facilities other than commercial hazardous waste facilities located under the siting authority of chapter 115A;

(c) Reimbursement to any private person for expenditures made before July 1, 1983 to provide alternative water supplies deemed necessary by the agency and the department of health to protect the public health from contamination resulting from the release of a hazardous substance;

(d) Removal and remedial actions taken or authorized by the agency or director under section 115B.17 including related enforcement and compliance efforts under section 115B.17 or 115B.18, and payment of the state share of the cost of remedial action which may be carried out under a cooperative agreement with the federal government pursuant to the Federal Superfund Act, under 42 U.S.C. Section 9604(c)(3) for actions related to commercial hazardous waste facilities located under the siting authority of chapter 115A;

(e) Compensation as provided by law, after submission by the waste management board of the report required under section 115A.08, subdivision 5, to mitigate any adverse impact of the location of commercial hazardous waste processing or disposal facilities located pursuant to the siting authority of chapter 115A;

(f) Planning and implementation by the commissioner of natural resources of the rehabilitation, restoration or acquisition of natural resources to remedy injuries or losses to natural resources resulting from the release of a hazardous substance;

(g) Inspection, monitoring and compliance efforts by the agency, or by political subdivisions with agency approval, of commercial hazardous waste facilities located under the siting authority of chapter 115A;

(h) Grants by the agency or the waste management board to demonstrate alternatives to land disposal of hazardous waste including reduction, separation, pretreatment, processing and resource recovery, for education of persons involved in regulating and handling hazardous waste;

(i) Intervention and environmental mediation by the legislative commission on waste management under chapter 115A; and

(j) Grants by the agency to study the extent of contamination and feasibility of cleanup of hazardous substances and pollutants or contaminants in major waterways of the state.

Subd. 3. **Limit on certain expenditures.** The director or agency may not spend any money under subdivision 2, clause (b) or (d) for removal or remedial actions to the extent that the costs of those actions may be compensated from any fund established under the Federal Superfund Act, 42 U.S.C. Section 9600 et seq. The director or agency shall determine the extent to which any of the costs of those actions may be compensated under the federal act based on the likelihood that the compensation will be available in a timely fashion. In making this determination the director or agency shall take into account:

(a) The urgency of the removal or remedial actions and the priority assigned under the Federal Superfund Act to the release which necessitates those actions;

(b) The availability of money in the funds established under the Federal Superfund Act; and

(c) The consistency of any compensation for the cost of the proposed actions under the Federal Superfund Act with the national contingency plan, if such a plan has been adopted under that act.

Subd. 4. **Revenue sources.** Revenue from the following sources shall be deposited in the environmental response, compensation and compliance fund:

(a) The proceeds of the taxes imposed pursuant to section 115B.22, including interest and penalties;

(b) All money recovered by the state under sections 115B.01 to 115B.18 or under any other statute or rule related to the regulation of hazardous waste or hazardous substances, including civil penalties and money paid under any agreement, stipulation or settlement but excluding fees imposed under section 116.12;

(c) All interest attributable to investment of money deposited in the fund; and

(d) All money received in the form of gifts, grants, reimbursement or appropriation from any source for any of the purposes provided in subdivision 2, except federal grants.

Subd. 5. **Recommendation by LCWM.** The legislative commission on waste management shall make recommendations to the standing legislative committees on finance and appropriations regarding appropriations from the fund.

Subd. 6. **Report to legislature.** By November 1, 1984, and each year thereafter, the agency shall submit to the senate finance committee, the house appropriations committee and the legislative commission on waste management a report detailing the activities for which money from the environmental response, compensation and compliance fund has been spent during the previous fiscal year.

Sec. 21.

115B.21. Taxes; definitions

Subdivision 1. **Application.** The definitions provided in this section and section 115B.02 apply to sections 115B.21 to 115B.24.

Subd. 2. **Commissioner.** "Commissioner" means the commissioner of revenue.

Subd. 3. **Generator.** "Generator" means a person who generates hazardous waste and who is required to disclose the generation of hazardous waste under the hazardous waste rules of the agency adopted under section 116.07.

Subd. 4. **Long term containment.** "Long term containment" means land disposal or storage for a period of more than one year.

Subd. 5. **Treatment.** "Treatment" means any material, technique or process designed to change the physical, chemical or biological character or composition of a hazardous waste in order to: (a) neutralize it; (b) render it nonhazardous or less hazardous; (c) render it safer to transport, store or dispose of; (d) make it amenable to storage; or (e) reduce its volume.

Subd. 6. **Wastewater treatment unit.** "Wastewater treatment unit" means a device which is part of a wastewater treatment facility subject to regulation pursuant to the federal Clean Water Act under 33 U.S.C. Section 1317 (b) or 1342.

Sec. 22.

115B.22. Hazardous waste generator tax

Subdivision 1. **Taxes imposed; exclusions.** Each generator of hazardous waste shall pay the taxes imposed by this section based upon the volume and destination of the hazardous wastes generated. The taxes imposed by this section do not apply to hazardous wastes destined for recycling or reuse including waste accumulated, stored, or physically, chemically, or biologically treated before recycling or reuse, to used crankcase oil, to hazardous waste which is generated as a result of any response action, or to hazardous waste which meets applicable pretreatment standards or compliance schedules and is discharged to a public sewage treatment works.

Subd. 2. **Long term containment without treatment.** Hazardous waste destined for long term containment without treatment shall be taxed at the rate of 32 cents per gallon of liquid or $32 per cubic yard of solid.

Subd. 3. **Long term containment after treatment.** Hazardous waste destined for long term containment after treatment shall be taxed at the rate of 16 cents per gallon of liquid or $16 per cubic yard of solid.

Subd. 4. Land treatment. Hazardous waste destined for treatment in or on the land shall be taxed at the rate of $32 per cubic yard.

Subd. 5. Other treatment. Hazardous waste destined for treatment, other than as provided in subdivision 6, to produce a material which is not hazardous, including treatment permitted by the agency in a sewage treatment works, or hazardous waste which is destined for destructive treatment by incineration shall be taxed at the rate of eight cents per gallon of liquid or $8 per cubic yard of solid.

Subd. 6. On-site wastewater treatment. The tax imposed under this section does not apply to hazardous waste which is destined for treatment in an on-site wastewater treatment unit to produce a material which is not hazardous before entering a public sewer system or waters of the state but the tax does apply to any residue of treatment which is a hazardous waste.

Subd. 7. Disposition of proceeds. The proceeds of the taxes imposed under this section including any interest and penalties shall be deposited in the fund.

Subd. 8. Review of tax by LCWM. After the waste management board submits the plan required under section 115A.11 to the legislative commission on waste management, the commission shall review the taxes and tax rates imposed under this section in light of the objectives and recommendations of the plan, and shall recommend to the standing tax committees of both houses of the legislature any changes in the taxes or tax rates which are needed to assist or encourage implementation of the strategies adopted by the state for management of hazardous waste.

Sec. 23.

115B.23 Severability

If any tax imposed under section 115B.22 is found to be invalid because of the purpose for which the proceeds were appropriated or made available under section 115B.20, subdivision 2, the proceeds of that tax shall not be appropriated or available for the objectionable purposes, but the tax shall continue to be imposed and the proceeds shall be appropriated and made available for other purposes provided in section 115B.20, subdivision 2.

Sec. 24.

115B.24. Tax administration and enforcement

Subdivision 1. Annual returns. Every generator of hazardous waste subject to taxation pursuant to section 115B.22 shall file a return relating to the tax due for the preceding calendar year with the commissioner of revenue by April 15 each year, in the form prescribed by the commissioner. Payment of the tax, to the extent not paid in full pursuant to subdivisions 2 and 3, shall be submitted with the return.

Subd. 2. Declarations of estimated tax. For 1983, every generator of hazardous waste required to pay a tax pursuant to section 115B.22 shall make a declaration of estimated hazardous waste generated for the last six months of calendar 1983 if the tax can reasonably be estimated to exceed

$500. The declaration of the estimated tax shall be filed by October 15, 1983. The amount of estimated tax with respect to which a declaration is required shall be paid in two equal installments by October 15, 1983 and January 15, 1984. For 1984 and subsequent years, every generator of hazardous waste required to pay a tax pursuant to section 115B.22 shall make a declaration of estimated hazardous waste generated for the calendar year if the tax can reasonably be expected to be in excess of $1,000. The declaration of estimated tax shall be filed by March 15. The amount of estimated tax with respect to which a declaration is required shall be paid in four equal installments on or before the 15th day of March, June, September, and December.

An amendment of a declaration may be filed in any interval between installment dates prescribed above but only one amendment may be filed in each interval. If an amendment of a declaration is filed, the amount of each remaining installment shall be the amount which would have been payable if the new estimate had been made when the first estimate for the calendar year was made, increased or decreased, as the case may be, by the amount computed by dividing

(1) the difference between (A) the amount of estimated tax required to be paid before the date on which the amendment was made, and (B) the amount of estimated tax which would have been required to be paid before that date if the new estimate had been made when the first estimate was made, by

(2) the number of installments remaining to be paid on or after the date on which the amendment is made.

The commissioner of revenue may grant a reasonable extension of time for filing any declaration but the extension shall not be for more than six months.

Subd. 2. **Failure to pay estimated tax.** (a) In case of any underpayment of estimated tax required by this section, except as provided in clause (b), there shall be added to the tax for the taxable year an amount determined at the rate specified in section 270.75, subdivision 4, upon the amount of the underpayment for the period of the underpayment.

For purposes of this subdivision, the amount of the underpayment shall be the excess of

(1) the amount of the installment, over

(2) the amount, if any, of the installment paid on or before the last date prescribed for payment.

The period of the underpayment shall run from the date the installment was required to be paid to whichever of the following dates is the earlier:

(1) April 15, or

(2) With respect to any portion of the underpayment, the date on which the portion is paid. For purposes of this paragraph, a payment of estimated tax on any installment date shall be considered a payment of any

previous underpayment only to the extent the payment exceeds the amount of the installment determined under this subdivision for the installment date.

(b) Notwithstanding the provisions of clause (a), the addition to the tax with respect to any underpayment of any installment shall not be imposed if the total amount of all payments of estimated tax made on or before the last date prescribed for the payment of the installment equals or exceeds the amount which would have been required to be paid on or before that date if the estimated tax were the lesser of:

(1) For 1985 and thereafter, the tax shown on the return of the taxpayer for the preceding year or, for 1984, twice the amount of the tax shown for 1983; or

(2) Eighty percent of the actual liability for the year.

Subd. 4. **Refunds of overpayments of estimated tax.** Refunds of overpayments of estimated tax shall be made as provided in section 290.936.

Subd. 5. **Exchange of information.** Notwithstanding the provisions of section 116.075, the pollution control agency may provide the commissioner of revenue with the information necessary for the enforcement of section 115B.22 and this section. Information disclosed in a return filed pursuant to this section is public. Information exchanged between the commissioner and the agency is public unless the information is of the type determined to be for the confidential use of the agency pursuant to section 116.075 or is trade secret information classified pursuant to section 13.37. Information obtained in the course of an audit of the taxpayer by the department of revenue shall be nonpublic or private data to the extent that it is not directly divulged in a return of the tax.

Subd. 6. **Payment by out-of-state generators.** A generator of any hazardous waste which is generated outside of this state and is transported into this state for long term containment or treatment as described in section 115B.22, subdivisions 2 to 5 shall pay the tax imposed by section 115B.22 at the first point at which the hazardous wastes are received by a person in this state for storage, treatment or long term containment. The tax shall be paid to the person who first receives the wastes in this state at the time the waste is received and shall be remitted by that person to the commissioner of revenue quarterly in the form and manner provided by the commissioner.

Subd. 7. **Duties of the agency and metropolitan counties.** The agency shall provide to the commissioner the names and addresses of all persons known to the agency who are subject to tax under section 115B.22, together with any information which the agency possesses concerning the amount of hazardous waste generated and disposed of by those persons. Metropolitan counties required to regulate hazardous wastes under section 473.811, subdivision 5b, shall provide to the agency the data and information necessary to allow the agency to carry out its duties under this subdivision. Upon request by the commissioner, the agency shall examine returns and reports filed with the commissioner and notify the commissioner of any suspected inaccurate or fraudulent declaration or return. The agency may assist in auditing any person subject to tax under section 22 when requested by the commissioner.

Subd. 8. **Penalties; enforcement.** The audit, penalty and enforcement provisions applicable to taxes imposed under chapter 290 apply to the taxes imposed under section 115B.22 and those provisions shall be administered by the commissioner.

Subd. 9. **Rules.** The commissioner may adopt temporary and permanent rules necessary to implement the provisions of this section and section 115B.22.

Subd. 10. **Administrative expenses.** Any amount expended by the commissioner from a general fund appropriation to enforce and administer section 115B.22 and this section shall be reimbursed to the general fund and the amount necessary to make the reimbursement is appropriated from the fund to the commissioner of finance for transfer to the general fund.

Sec. 25.

116.12 Hazardous waste administration fees

Subdivision 1. **Fee schedules.** The agency shall establish the fees provided in subdivisions 2 and 3 in the manner provided in section 16A.128 to cover the amount appropriated from the general fund to the agency for that year for permitting, monitoring, inspection and enforcement expenses of the hazardous waste activities of the agency.

The legislature may appropriate additional amounts that need not be covered by fees or may provide that the fees shall cover only a portion of the general fund appropriation for the hazardous waste activities of the agency, in order to assure adequate funding for the regulatory and enforcement functions of the agency related to hazardous waste. All fees collected by the agency under this section shall be deposited in the general fund.

Subd. 2. **Hazardous waste generator fee.** Each generator of hazardous waste shall pay a fee on the hazardous waste which he generates. The agency shall compute the amount of the fee due based on the hazardous waste disclosures submitted by the generators and other information available to the agency. The agency shall annually prepare a statement of the amount of the fee due from each generator. The fee shall be paid annually commencing with the first day of the calendar quarter after the date of the statement.

The agency may exempt generators of small quantities of hazardous wastes otherwise subject to the fee if it finds that the cost of administering a fee on those generators is excessive relative to the proceeds of the fee. The fee shall consist of a minimum fee for each generator not exempted by the agency and an additional fee based on the quantity of wastes generated by the generator.

If any metropolitan counties recover the costs of administering county hazardous waste regulations by charging fees, the fees charged by the agency outside of those counties shall not exceed the fees charged by those counties. The agency shall not charge a fee in any metropolitan county which charges such a fee. The agency shall impose a fee calculated as a surcharge on the fees charged by the metropolitan counties and by the agency to reflect the agency's expenses in carrying out its statewide hazardous waste regulatory responsibilities. The surcharge imposed on the fees charged by the metropolitan counties shall be collected by the metropolitan counties in the manner in which the counties collect their generator fees. Metropolitan counties shall remit the proceeds of the surcharge to the agency by the last day of the month following the month in which they were collected.

Subd. 2. **Facility fees.** The agency shall charge an original permit fee, a reissuance fee and an annual operator's fee for any hazardous waste facility regulated by the agency. The agency may include reasonable and necessary costs of any environmental review required under chapter 116D in the original permit fee for any hazardous waste facility.

Sec. 26. Minnesota Statutes 1982, section 115A.24, subdivision 1, is amended to read:

Subdivision 1. **Certificate.** ~~Except as provided in subdivision 2,~~ By December 15, 1982, on the basis of and consistent with its hazardous waste management plan adopted under section 115A.11, the board shall issue a certificate or certificates of need for disposal facilities for hazardous wastes in the state. The certificate or certificates shall indicate the types and volumes of waste for which disposal facilities are and will be needed through the year 2000 and the number, types, sizes, general design and operating specifications, and function or use of the disposal facilities needed in the state. The board shall certify need only to the extent that the board has determined that there are no feasible and prudent alternatives including waste reduction, separation, pretreatment, processing, and resource recovery which would minimize adverse impact upon natural resources, provided that the board shall require the establishment of at least one commercial disposal facility in the state. Economic considerations alone shall not justify certification nor the rejection of alternatives. Alternatives that are speculative and conjectural shall not be deemed to be feasible and prudent. The board shall consider all technologies being developed in other countries as well as in the United States when it considers the alternatives to hazardous waste disposal. The certificate or certificates shall not be subject to the provisions of chapter 14 but shall be the final determination required on the matters decided by the certificate or certificates and shall have the force and effect of law. The certificate or certificates shall not be amended for five years. The board and the permitting agencies, in reviewing and selecting sites, completing environmental impact statements, and issuing approvals and permits for waste disposal facilities described in the certificate or certificates of need, shall not reconsider matters determined in the certification. The board and the permitting agencies shall be required to make a final decision approving the establishment of facilities consistent with the certification. The board and the permitting agencies shall be required to make a final decision approving the establishment of at least one commercial disposal facility for hazardous waste in the state.

Sec. 27. Minnesota Statutes 1982, section 466.01, is amended by adding a subdivision to read:

Subd. 8. For the purposes of sections 466.01 to 466.15, "release" and "hazardous substance" have the meanings given in section 115B.02.

Sec. 28. Minnesota Statutes 1982, section 466.04, subdivision 1, is amended to read:

Subdivision 1. Limits; punitive damages. Liability of any municipality on any claim within the scope of sections 466.01 to 466.15 shall not exceed

(a) $100,000 when the claim is one for death by wrongful act or omission and $100,000 to any claimant in any other case;

(b) $300,000 for any number of claims arising out of a single occurrence~~.~~;

(c) Twice the limits provided in clauses (a) and (b), but not less than $300,000 per claim, when the claim arises out of the release or threatened release of a hazardous substance, whether the claim is brought under sections 115B.01 to 115B.15 or under any other law.

No award for damages on any such claim shall include punitive damages.

Sec. 29. **Recommendations concerning allocation of liability.**

The waste management board shall make recommendations to the legislature by November 1, 1983 regarding the allocation of liability among the owners, operators, and users of a hazardous waste disposal facility established pursuant to sections 115A.18 to 115A.30, including any recommended legislative changes, taking into consideration the need for the facility, the state's involvement in the facility, the need to protect the health, property and environment of the local community from injury and loss, and the need for incentives to encourage the development and use of alternatives to land disposal. The recommendations shall be made after consultation with affected industries, including insurers, generators, transporters, disposers, and treaters of hazardous waste, individuals, including academic, scientific and legal professionals, and groups, including community and environmental groups.

Sec. 30. **Victim compensation study.**

By July 1, 1984, the legislative commission on waste management shall conduct a study and make recommendations to the legislature on the creation of a compensation fund to compensate persons who are injured as the result of a release of a hazardous substance and who would not otherwise be adequately compensated for their injuries. The study shall consider matters including the following:

(a) The appropriate scope of compensation which should be provided by the fund including the extent of any compensation which should be available for medical expenses, disability, loss of income, physical impairment, and death;

(b) Creation of a simple, speedy, and cost efficient claims procedure which provides an effective remedy for injured claimants;

(c) Methods by which compensation can be financed by those who create or contribute to the risk of injury from hazardous substance releases, including the manner by which the state may seek to recover amounts paid from the fund; and

(d) Whether the fund should be established or administered at the federal or state level and the appropriate degree of state and federal cooperation in providing compensation.

Sec. 31. Insurance study.

The commissioner of insurance shall conduct a study of insurance providing coverage for liability under section 5. The commissioner shall submit the results of the study, together with his recommendations, to the legislature by July 1, 1985. The director of the pollution control agency shall cooperate with and provide assistance to the commissioner during the course of the study.

Sec. 32. Appropriation; complement.

Subdivision 1. Appropriation to fund. $5,000,000 is appropriated from the general fund and transferred to the environmental response, compensation, and compliance fund established in section 20. This appropriation is available until expended.

Subd. 2. Tax administration; complement. $50,000 in fiscal year 1984 and $40,000 in fiscal year 1985 is appropriated from the general fund to the commissioner of revenue for the purposes of administering and enforcing sections 21 to 24. This appropriation shall be reimbursed to the general fund under the provisions of section 24, subdivision 10.

The complement of the department of revenue is increased by two positions.

Subd. 3. Appropriation for response actions; complement. $483,700 in fiscal year 1984 and $400,700 in fiscal year 1985 is appropriated from the environmental response, compensation, and compliance fund to the pollution control agency for administrative costs.

The complement of the pollution control agency is increased by ten positions.

All money in the environmental response, compensation, and compliance fund not otherwise appropriated is appropriated to the pollution control agency for the purposes described in section 20, subdivision 2, clauses (a), (b), and (c). This appropriation is available until June 30, 1985.

Subd. 4. Appropriation for compliance actions; complement. $45,600 in fiscal year 1984 and $56,400 in fiscal year 1985 is appropriated from the general fund to the attorney general for the purposes of enforcing this act.

This appropriation shall be reimbursed to the general fund from the environmental response, compensation, and compliance fund, and the amount necessary to make the reimbursement is appropriated to the commissioner of finance for transfer to the general fund.

The complement of the office of the attorney general is increased by two positions.

Subd. 5. **Appropriation for victim compensation study.** $20,000 is appropriated from the general fund to the legislative commission on waste management to carry out the study required by section 30.

Subd. 6. **Appropriation for insurance study.** There is appropriated from the general fund to the commissioner of insurance $5,000 for fiscal year 1984, to conduct the study described in section 31.

Sec. 33. **Repealer.**

Minnesota Statutes 1982, section 115A.24, subdivision 2, is repealed.

Sec. 34. **Effective date.**

Sections 17 to 24 are effective the day following final enactment. The taxes imposed by section 22 are effective July 1, 1983. The remaining sections of this act are effective July 1, 1983.

Approved May 10, 1983.

Summary of the 1983 Superfund Bill:

Environmental Response and Liability Act*

The Environmental Response and Liability Act (Superfund) has three main purposes:

(1) To establish a statutory standard of strict liability for the release of hazardous substances;

(2) To establish authority for the state to respond to releases of hazardous substances;

(3) To create a fund to pay for the cleanup of sites not covered by the Federal Superfund and to provide that money from the Environmental Response Fund could be used for the 10% state match required by the Superfund Act as a condition for receipt of federal cleanup money.

Citizens have become increasingly aware of the serious threats to public health and the environment posed by the discovery of hazardous wastes not only at sites such as Love Canal in New York, but at sites within our own state, such as St. Louis Park, Fridley, New Brighton, and Oakdale. At the time the bill was being considered by the 1982 Legislature, the Minnesota Pollution Control Agency had identified 49 contaminated hazardous waste sites within the state in need of cleanup; that number has now grown to 61. The question then arises: what can be done to clean up those existing sites which are the result of past improper handling of hazardous wastes and what can be done to help prevent future improper handling of hazardous waste?

One way to guard against future hazardous waste incidents would be to make sure that all those associated with the generation, transport and disposal of hazardous waste use the greatest degree of care possible. In order to help accomplish this, the bill establishes a statutory standard of strict, joint and several liability.

Strict, Joint and Several Liability

Strict liability is now a part of the common law and many people are familiar with it in the context of the use of dynamite. When a person is engaged in an ultrahazardous or inherently dangerous activity, the law now provides that that person is liable for the damages which result from that activity without regard to fault. Even though he takes all possible precautions and the damages are not caused by his negligence, the person engaged in the activity will still be liable.

While some other states have enacted laws establishing strict liability and while courts in some other states have applied strict liability for injuries caused by hazardous substances, no case has been decided yet which clearly establishes the standards of liability that will apply in Minnesota. The Superfund establishes the standard of liability in statute.

*The bill was originally introduced in 1981, when is was heard in only one committee. In 1982 it was heard in 12 committees and subcommittees, passed both the House and Senate by wide margins, but was vetoed by the Governor. The bill again passed the Legislature in 1983 and was signed into law by the Governor on May 10.

Risks from hazardous substances can never be completely eliminated. A person engaged in an activity involving hazardous substances may take all precautions that are humanly and technologically possible to limit risk and yet an innocent person may still be injured as a result of exposure to a release. Those supporting the Superfund believe the question this raises is one of fundamental fairness -- should the loss fall on the innocent victim who is injured or on the person who created the risk? The policy of the act is that the loss should fall on persons who create the risk and who are in the best position to control the causes of injury. Thus, those who profit from the use of the substances will bear the cost of any injuries, not the innocent victims of those injuries.

The act provides that persons who are found liable for injuries or cleanup costs are liable jointly and severally. Under existing law, when several parties contribute by their actions to the same injury they are jointly liable for the consequences. If there is joint liability, then under existing Minnesota law the liability is also joint and several (Minnesota Statutes § 604.02, Subdivision 1). In its usual form, joint and several liability means that the injured party may recover his damages against each defendant or any of them, up to 100 percent of the total liability.

It should be pointed out that, in enforcing the federal Superfund law, the Environmental Protection Agency and Department of Justice are applying strict, joint and several liability for the cleanup of contaminated sites.

Limitations and Exceptions to Liability

A number of important exceptions and defenses to liability were added to the act as a result of discussions with interested parties to assure greater fairness in applying the general rule of strict, joint and several liability. Some of these exceptions are summarized here.

Limitation on liability for past acts: Strict, joint and several liability for damages resulting from past acts would apply where wastes were deposited after 1973; between 1960 and 1973, it would apply only if the responsible person's activity would have been abnormally dangerous at that time. It would not apply for wastes deposited prior to 1960.

There is a limitation on joint and several liability in the act. The act provides that liability can be divided up among the defendants according to their degree of responsibility, and no defendant can be required to pay more than two times his apportioned share for personal injury or economic loss. Full joint and several liability would apply for cleanup. Any defendant who has to pay more than his apportioned share can sue other defendants to recover the excess.

There is no liability for either damages or cleanup for the innocent property owner who discovers hazardous substances which were buried on his property without his knowledge or consent and if he has not associated himself with the release.

There is no liability for application of farm or forestry chemicals or for disposal of pesticide containers and residues according to instructions on product labels.

There is no liability if the release is due to an unforeseeable event like a tornado, theft, or vandalism or due to the act of an unrelated third party.

There is no liability for garbage haulers unless they knew or reasonably should have known they were carrying the particular hazardous substance that later causes injury.

There is no liability for a transporter of hazardous waste unless he selected the facility to which the waste was taken or disposed of the waste in a manner contrary to law.

And there is no liability if the state or federal government has issued a permit which allows discharge of the hazardous substances and if the terms of the permit are being fulfilled.

With these limitations the act sets a standard of liability which would give injured parties a fair chance to recover for their injury or loss and it assists the state in recovering its cleanup costs from the party responsible for the contamination.

But what if the person responsible for the release of a hazardous substance cannot be determined or is found to be insolvent? Who then will pay for the cleanup of hazardous waste sites? The act establishes the Environmental Response, Compensation and Compliance Fund to do this. It will be funded from two sources: a general fund appropriation of $5 million for the biennium and a tax on generators of hazardous waste which is expected to raise about $900,000 annually.

State Authority to Clean Up Contaminated Sites

The Superfund authorizes the state Pollution Control Agency (PCA) to take removal or remedial action when there is a release or threatened release of a hazardous substance or any pollutant or contaminant which may present an imminent or substantial danger.

Prior to passage of the act, the PCA did not have the necessary authority and funds to adequately prevent or minimize environmental contamination from hazardous waste. In some cases the responsible parties were unknown or bankrupt. When a responsible party had been identified the PCA could request, demand or attempt to negotiate the desired cleanup action. However, if this effort was unsuccessful, the only option left to the PCA was to file a lawsuit. The result was litigation which could take years to reach a conclusion. In the meantime, toxic chemical contamination progressively spread deeper into the groundwater systems increasing the environmental damage and cost of cleanup. The authority given to PCA under Superfund would permit it to directly address these problems by allowing to "clean up first, litigate later."

Tax on Generators of Hazardous Wastes

The tax in the act is based on the amount of waste generated and o the ultimate disposition of the waste. The highest tax is on waste whi is to be landfilled without treatment -- $32.00 per cubic yard or 32¢ p gallon.

Hazardous wastes destined for long-term containment after treatmen will be taxed at $16.00 per cubic yard or 16¢ per gallon.

Hazardous wastes which are incinerated or chemically treated will taxed at $8.00 per cubic yard or 8¢ per gallon.

The purpose of the tax is threefold and is consistent with the purposes of the 1980 Waste Management Act: (1) to encourage a reducti the amount of hazardous waste generated; (2) to encourage the treatmen and processing of hazardous waste rather than land disposal; and (3) t raise revenue to clean up areas which have been contaminated by hazard wastes.

The question then arises: How much money will be needed to clean hazardous waste sites in this state?

After passage of the federal "Superfund" act in late 1980, it was hoped that the federal government would clean up a major portion of th contaminated sites around the country. However, it has since been rec nized by all interested parties that the Superfund will be inadequate respond to all hazardous waste sites. In fact, EPA has indicated that there may be as many as 9,000 hazardous waste sites across the countr At the same time, EPA officials have indicated to Congress that Super may only provide for the actual cleanup of 170 sites. Thus, states w be responsible for sites not covered by Superfund, as well as for pro matching money for sites that may receive Superfund monies. These st responsibilities will result in the need for enormous amounts of mone

It is difficult to say precisely just how enormous the need will in Minnesota. We will probably get Superfund dollars to clean up onl few of our contaminated sites. There are now 61 sites contaminated b hazardous waste in the state which are in need of cleanup. The cost cleanup at each of these sites varies widely, ranging from several hu thousand dollars to many millions. The Pollution Control Agency esti that it will cost about $50 million to clean up all of the sites dis thus far. Additional money will be needed if more contaminated site found.

Support for the Superfund Act

The act had broad support from the League of Minnesota Cities, Association of Metropolitan Municipalities, the Association of Minne Counties, the Metropolitan Inter-County Association, the League of W Voters, the Minnesota AFL-CIO, the Farmers Union, Minnesota COACT, t Sierra Club, the Izaak Walton League, the Minnesota Audubon Council, Minnesota Trial Lawyers Association, the Joint Religious Legislative Conference and the Minnesota Catholic Conference. In addition, maj newspapers around the state urged passage of the bill.

HOUSE RESEARCH

Bill Summary

H.F. 76 S.F.

SUBJECT: Hazardous Waste Superfund (passed by House 4/18/83)

AUTHORS: Long, Munger, Simoneau, Sieben, Evans

COMMITTEE:

DATE: 4/25/83

OVERVIEW

This bill has three main purposes:

(1) To establish a statutory standard of strict liability for the release of hazardous substances;

(2) To establish authority for the state to respond to releases of hazardous substances;

(3) To create a fund to pay for the cleanup of sites not covered by the federal Superfund and to provide that money from the state Superfund could be used for the 10% state match required by the federal Superfund law as a condition for receiving federal cleanup money.

SECTION BY SECTION SUMMARY

Section 1. CITATION. Sections 1 to 19 are the "Environmental Response and Liability Act."

Section 2. DEFINITIONS.

Subd. 3. "Agency" means the state pollution control agency (PCA).

Subd. 5. "Facility" includes buildings, structures, pits, ponds, landfills, vehicles and other sites where a hazardous substance, pollutant or contaminant has been deposited.

Subd. 8. "Hazardous substance" includes certain chemicals and hazardous air pollutants as designated by federal law and hazardous wastes. The term does not include certain natural and synthetic gases, nor petroleum.

Subd. 9. "Hazardous Waste" means those substances identified as hazardous waste according to state or federal law.

Legislative Analyst: Stan Jacobson 296-1885

Phone:

Subd. 12. "Person" includes individuals, partnerships, corporations, the federal government, the state and political subdivisions of the state.

Subd. 13. "Pollutant or contaminant" includes substances which may cause death or disease in organisms. Certain natural and synthetic gases are excluded.

Subd. 14. "Release" includes spilling, dumping or otherwise discharging into the environment either on a single occasion or continuously. The term does not include engine exhausts, nuclear releases, and applications of fertilizer.

Subd. 15. "Remedy" includes actions taken instead of or in addition to removal actions to prevent, minimize or eliminate the release of a hazardous substance, pollutant or contaminant in order to protect the public health, welfare or environment.

Subd. 16. "Removal" includes actions taken to clean up or remove a released hazardous substance, pollutant or contaminant from the environment.

Subd. 17. "Response" includes both remedy and removal actions.

Section 3. RESPONSIBLE PERSON

Subd. 1. GENERAL RULE. Defines who is responsible for the release of a hazardous substance, pollutant or contaminant. Responsible persons include those who operate a facility, and those who possess or transport, and arrange for disposal, of the hazardous substance, pollutant or contaminant.

Subd. 2. EMPLOYEES AND EMPLOYERS. If an employee has acted within the scope of his employment, he is personally liable for the release of a hazardous substance only if he was negligent; his employer is liable whether or not the employee was negligent.

Subd. 3. OWNER OF REAL PROPERTY. A landowner is not responsible for the release of a hazardous substance unless he knew about or cooperated with the disposal activities.

Section 4. LIABILITY FOR RESPONSE COSTS AND NATURAL RESOURCES.

Subd. 1. LIABILITY. A person responsible for the release of a hazardous substance is strictly liable for the costs of removal by anyone and of remedy by the government, as well as for harm to natural resources. All responsible persons are jointly and severally liable under this section.

Subd. 2. EXCLUDES POLLUTANT OR CONTAMINANT. There is no liability under this section for the release of a pollutant or contaminant.

Subd. 3. THREATENED RELEASE. Liability for a threatened release of a hazardous substance is limited to the response costs of the PCA.

Subd. 4. POLITICAL SUBDIVISIONS. Limits on the liability of a political subdivision are provided in section 26.

Subd. 5. HOUSEHOLD REFUSE. A transporter of household refuse is not liable under this section for the release of a hazardous substance unless he knew, or should have known, that the hazardous substance was in the refuse.

Subd. 6. DEFENSE TO CERTAIN RESPONSE CLAIMS. If a "response action" (clean up, etc.) was not authorized by the PCA, neither a political subdivision nor a private person can recover under this section for the costs of the response action for any hazardous substance placed in a facility before April 1, 1982.

Subd. 7. DEFENSE FOR INTERVENING ACTS. Someone who would ordinarily be responsible for the release of a hazardous substance is not liable under this section if the release was caused solely by an act of God, war or a "third party" (vandals, etc.).

Subd. 8. PERMITTED RELEASES. There is no liability under this section if the release of a hazardous substance was permitted by the state or federal government, or if liability has been assumed under federal law.

Subd. 9. NATURAL RESOURCES. There is no liability under this section for harm to natural resources if the harm was identified as an irretrievable commitment of natural resources according to state or federal law.

Subd. 10. RENDERING ASSISTANCE TO THE GOVERNMENT. There is no liability under this section for harm which resulted while providing aid to the PCA or to federal authorities.

Subd. 11. BURDEN OF PROOF. Puts the burden of proof for the defenses in subdivisions 6 to 10 on the defendant.

Section 5. LIABILITY FOR ECONOMIC LOSS, DEATH, PERSONAL INJURY AND DISEASE.

Subd. 1. LIABILITY. A person responsible for the release of a hazardous substance is strictly liable for all resulting actual economic losses, death, personal injury and disease. All responsible persons are jointly and severally liable under this section.

Subd. 2. EXCLUDES POLLUTANT OR CONTAMINANT. There is no liability under this section for the release of a pollutant or contaminant.

Subd. 3. CERTAIN EMPLOYEE CLAIMS. There is no liability under this section if a claim for personal injury or disease is compensable under the workers compensation law.

Subd. 4. LIABILITY LIMITS. The liability of a political subdivision or a small business under this section is limited to $300,000 per person and $600,000 per occurrence. The liability of anyone else under this section is limited to $1.5 million.

Subd. 5. HOUSEHOLD REFUSE. A transporter of household refuse is not liable under this section for the release of a hazardous substance unless he knew, or should have known, that the hazardous substance was in the refuse.

Subd. 6. DEFENSE FOR INTERVENING ACTS. Someone who would ordinarily be responsible for the release of a hazardous substance is not liable under this section if the release was caused solely by an act of God, war or a third party.

Subd. 7. PERMITTED RELEASES. There is no liability under this section if the release of a hazardous substance was permitted by the state or federal government, or if liability has been assumed under federal law.

Subd. 8. RENDERING ASSISTANCE TO THE GOVERNMENT. There is no liability under this section for harm which resulted while providing aid to the PCA or to federal authorities.

Subd. 9. DEFENSE FOR CERTAIN PAST ACTIONS. There is no liability under this section if the hazardous substance was placed in the facility before April 1, 1982 and the defendant's activities were not abnormally dangerous. The standards provided in existing Minnesota common law are used to decide whether the defendant's activities were abnormally dangerous.

Subd. 10. BURDEN OF PROOF. Puts the burden of proof for the defenses in subdivisions 6 to 9 on the defendant.

Section 6. CAUSATION. Provides a method that is easier than under current law for the claimant to prove that his injury was caused by the release of a hazardous substance.

Section 7. APPORTIONMENT. (Rules for determining who pays how much among several defendants.)

Subd. 1. APPORTIONMENT FACTORS. The jury may apportion liability among the defendants based on factors listed in this subdivision.

Subd. 2. LIMITATION. If a person is held "jointly and severally liable" and if he can prove what his proportionate share is of the aggregate liability, his total liability is limited to three times his proportionate share. (Ordinarily, if a person is held "jointly and severally liable", there is no limit on his total liability.)

Subd. 3. CONTRIBUTION. Anyone who is jointly and severally liable and who pays more than his proportionate share may seek contribution from anyone else who is also liable.

Section 8. TRANSFERRING LIABILITY. No one may avoid liability under this Act by attempting to transfer his own liability to someone else.

Section 9. STATUTE OF LIMITATIONS. A lawsuit claiming damages must be started within six years of the time when the claim could have been made based on the time of discovering the harm and its causal connection to the release of a hazardous substance.

Section 10. OTHER REMEDIES PRESERVED. Any right or remedy available under other state, federal or common law is unaffected by this Act.

Section 11. DOUBLE RECOVERY PROHIBITED. No one may recover twice for the same costs or damages.

Section 12. APPLICATION OF SECTIONS 1 TO 12. Sections 1 to 12 do not apply to the release of a hazardous substance which occurred entirely before July 1, 1983, regardless of the date of discovery of harm caused by the release.

Section 13. DISPOSITION OF FACILITIES.

Subd. 1. USE OF A CLOSED FACILITY. No one may disturb the containment or monitoring systems on any property in which hazardous waste remains after the facility is closed, unless authorized to do so by the PCA.

Subd. 2. RECORDING AFFIDAVIT. If a landowner knows that his property was used as a disposal site for hazardous waste or that there has been extensive contamination by release of a hazardous substance, the owner must file an affidavit with the county recorder to that effect.

Subd. 3. DUTY OF COUNTY RECORDER. The county recorder must record any affidavit or notation made under subdivision 2 so that disclosure is assured during a title search.

Subd. 4. PENALTIES. Anyone who knowingly violates either subdivision 1 or 2 may be liable for all costs and damages recoverable under sections 4 and 5. In addition, anyone knowingly violating subdivision 1 may be liable for a civil fine not exceeding $100,000.

Section 14. STATE RESPONSE TO RELEASES

Subd. 1. REMOVAL AND REMEDIAL ACTIONS. The PCA may take any removal or remedial action necessary to protect public health, welfare or the environment. These actions may be taken whenever there is a release or a threatened release of a hazardous substance and whenever there is a release or a substantial threat of a release of a pollutant or contaminant presenting an imminent and substantial danger to the public. The PCA must first request any person known to be responsible to take the necessary actions. However, the director has special powers to act quickly in an emergency.

Subd. 2. OTHER ACTIONS. The PCA may gather information about the extent of the release or threatened release of a hazardous substance, pollutant or contaminant and undertake studies in order to carry out removal or remedial actions.

Subd. 3. DUTY TO PROVIDE INFORMATION. Anyone responsible for a release or a threatened release of a hazardous substance, pollutant or contaminant must provide the PCA with relevant information.

Subd. 4. ACCESS TO INFORMATION AND PROPERTY. The PCA may examine written records and enter onto property in order to carry out its duties under this section.

Subd. 5. CLASSIFICATION OF DATA. In general, information obtained by the PCA under subdivisions 3 and 4 is public data. However, the subject of the information may apply to the director of the PCA for classification of the data as private or nonpublic.

Subd. 6. RECOVERY OF EXPENSES. The PCA may recover the expenses it incurs in carrying out its duties under this section in a civil action against a responsible person.

Subd. 7. ACTIONS RELATING TO NATURAL RESOURCES. The state is the trustee of the air, water and wildlife of the state. Actions for damages to the air, water or wildlife are brought in the name of the state as trustee.

Subd. 8. ACTIONS RELATING TO PESTICIDES, FERTILIZER, ETC. Transfers the PCA's powers under this section to the commissioner of agriculture if the release of a hazardous substance, pollutant or contaminant involves pesticides, fertilizers, soil or plant amendments.

Subd. 9. ACTIONS RELATING TO OCCUPATIONAL SAFETY. Directs the PCA and the commissioner of labor and industry to coordinate their actions regarding a release or threatened release affecting occupational safety.

Subd. 10. ACTIONS RELATING TO HEALTH. Directs the PCA and the commissioner of health to coordinate their actions regarding a release or threatened release affecting public health.

Subd. 11. LIMIT ON ACTIONS BY POLITICAL SUBDIVISIONS. No political subdivision may order anyone to act in a way which conflicts with a request by the PCA.

Subd. 12. AUTHORIZATION OF CERTAIN RESPONSE ACTIONS. The PCA may authorize a political subdivision or a private person to take certain response actions regarding hazardous substances placed in a facility before April 1, 1982. This authorization will permit the political subdivision or private person to recover costs under section 4.

Subd. 13. PRIORITIES; RULES. The PCA must adopt a temporary list and permanent rules for setting priorities to take remedial action.

Section 15. MISCELLANEOUS CIVIL REMEDIES.

Subd. 1. CIVIL PENALTIES. Any responsible person who fails to make reasonable progress in completing response actions requested by the PCA is liable to the state for a civil penalty up to $25,000 per day.

Subd. 2. ACTION TO COMPEL PERFORMANCE. The attorney general may sue to compel performance by any responsible person who fails to make reasonable progress in completing response actions requested by the PCA.

Subd. 3. REQUESTS FOR RESPONSE ACTIONS. Requests by the PCA for a responsible person to take response actions must be in writing and provide a reasonable time for the action to be taken.

Subd. 4. INJUNCTIVE RELIEF. The release or threatened release of a hazardous substance, pollutant or contaminant is a public nuisance which the state may seek to enjoin.

Section 16. PURPOSES OF FUND AND TAXES. Establishes the purposes of the environmental response, compensation and compliance fund (the "state superfund"), including protection of public health, reduction of hazardous waste through recycling and placing the financial burden of state waste management activities on those who produce these wastes.

Section 17. ENVIRONMENTAL RESPONSE, COMPENSATION AND COMPLIANCE FUND. (THE "STATE SUPERFUND.")

Subd. 1. ESTABLISHMENT. Creates the "state superfund."

Subd. 2. PURPOSES. Lists the purposes for which the money in the fund may be spent, including response actions by the PCA, reimbursement for alternative water supplies, compensating a community in which a hazardous waste site is established under state law, restoring natural resources, and the PCA's expenses in monitoring compliance by commercial facilities.

Subd. 3. LIMITS ON CERTAIN PAYMENTS. Requires the PCA to coordinate payments from the state superfund with payments from the federal superfund.

Subd. 4. REVENUE SOURCES. Lists revenue sources for the state superfund including taxes imposed by section 20 and other fees.

Subd. 5. RECOMMENDATION BY LCWM. Requires the legislative commission on waste management to make recommendations to standing legislative committees concerning appropriations from the state superfund.

Subd. 6. REPORT TO LEGISLATURE. Requires the PCA to submit reports annually to the legislature concerning money spent from the state superfund.

Section 18. RECOMMENDATIONS CONCERNING ALLOCATION OF LIABILITY. Requires the waste management board to make recommendations to the legislature regarding the allocation of liability at the state-sited hazardous waste disposal facility.

Section 19. DEFINITIONS FOR TAXES. Defines terms used in section 20, including "generator" as a person who generates hazardous waste and is required by state law to disclose this fact.

Section 20. HAZARDOUS WASTE GENERATOR TAX.

Subd. 1. TAX IMPOSED. Imposes taxes on hazardous waste generators. Exempts hazardous wastes which are recycled or reused.

Subd. 2. LONG TERM CONTAINMENT WITHOUT TREATMENT. Taxes hazardous waste destined for long term containment at 32¢ per gallon or $32 per cubic yard.

Subd. 3. LONG TERM CONTAINMENT AFTER TREATMENT. Taxes hazardous waste destined for long term containment after treatment at 16¢ per gallon or $16 per cubic yard.

Subd. 4. OTHER TREATMENT. Taxes hazardous waste destined for treatment which will produce nonhazardous material at 8¢ per gallon or $8 per cubic yard.

Subd. 5. LAND TREATMENT. Taxes hazardous waste destined for treatment in or on land at $32 per cubic yard.

Subd. 6. ON SITE WASTEWATER TREATMENT. There is no tax imposed for hazardous waste which is treated at the generator's site to produce nonhazardous material.

Subd. 7. PROCEEDS OF TAXES. The taxes are deposited in the state superfund.

Subd. 8. REVIEW OF TAX BY LCWM. The legislative commission on waste management will make recommendations regarding the hazardous waste generator tax to the legislature.

Section 21. SEVERABILITY. If any tax imposed by section 20 is found invalid, the other taxes shall continue to be imposed.

Section 22. TAX ADMINISTRATION.

Subd. 1. ANNUAL RETURNS. Each person liable for taxes imposed by section 20 must file an annual tax return with the commissioner of revenue.

Subd. 2. DECLARATIONS OF ESTIMATED TAX. If the estimated annual tax exceeds $1,000, quarterly payments must be made.

Subd. 3. FAILURE TO PAY ESTIMATED TAX. If the estimated tax payment is less than 80 percent of the actual tax liability, interest on the unpaid amount accrues at a rate based on the prime rate charged by commercial banks.

Subd. 4. REFUNDS. Provides a refund for overpayments of estimated tax.

Subd. 5 EXCHANGE OF INFORMATION. Authorizes the PCA to provide information to the commissioner of revenue to enforce the tax.

Subd. 6. PAYMENTS BY OUT OF STATE GENERATORS. An out of state generator who transports hazardous waste into Minnesota for long term containment or treatment must pay the tax imposed by section 20.

Subd. 7. CERTAIN METROPOLITAN COUNTIES. Metropolitan counties required by state law to regulate hazardous wastes must provide the PCA with information to carry out its duties.

Subd. 8. PENALTIES. The same penalties provided for other state tax collections apply to the tax imposed by section 20.

Subd. 9. RULES. Authorizes the commissioner of revenue to adopt rules.

Subd. 10. ADMINISTRATIVE EXPENSES. Coordinates payments between the general fund and the state superfund for certain administrative expenses.

Section 23. HAZARDOUS WASTE ADMINISTRATION FEES.

Subd. 1. FEE SCHEDULES. Provides additional fees for the PCA to enforce the hazardous waste laws.

Subd. 2. HAZARDOUS WASTE GENERATOR FEE. Each generator must pay an annual fee for generating hazardous waste. Generators of small quantities of hazardous waste may be exempted.

Subd. 3. FACILITY FEES. Each facility that obtains a permit from the PCA shall pay a permit fee, a reissuance fee and an annual operator's fee.

Section 24. Amends Minn. Stat. Section 115A.24, Subd. 1. A technical amendment required by section 29.

Section 25. Amends Minn. Stat. Section 466.01 to provide definitions of "release" and "hazardous substance" used in section 25.

Section 26. Amends Minn. Stat. Section 466.04, Subd. 1. Provides that a municipality's liability for a release of a hazardous substance is twice its ordinary limits, that is, $200,000 per claimant and $600,000 for all claims from a single occurrence.

Section 27. INSURANCE STUDY. Requires the commissioner of insurance to study and report to the legislature concerning the availability of insurance against liability for personal injury under section 5.

Section 28. APPROPRIATION. Appropriates $5 million to the state superfund.

Section 29. REPEALER. Repeals Minn. Stat. Sec. 115A.24, Subd. 2. which required enactment of this law before any hazardous waste sites could be established.

Section 30. EFFECTIVE DATE. Sections 14 to 22 are effective the day after enactment. All other sections are effective July 1, 1983.

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COMPENSATION FOR TOXIC SUBSTANCES EXPOSURE

Work Group (Torzi group will serve as Tech. staff)
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Uhlmann
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Rose

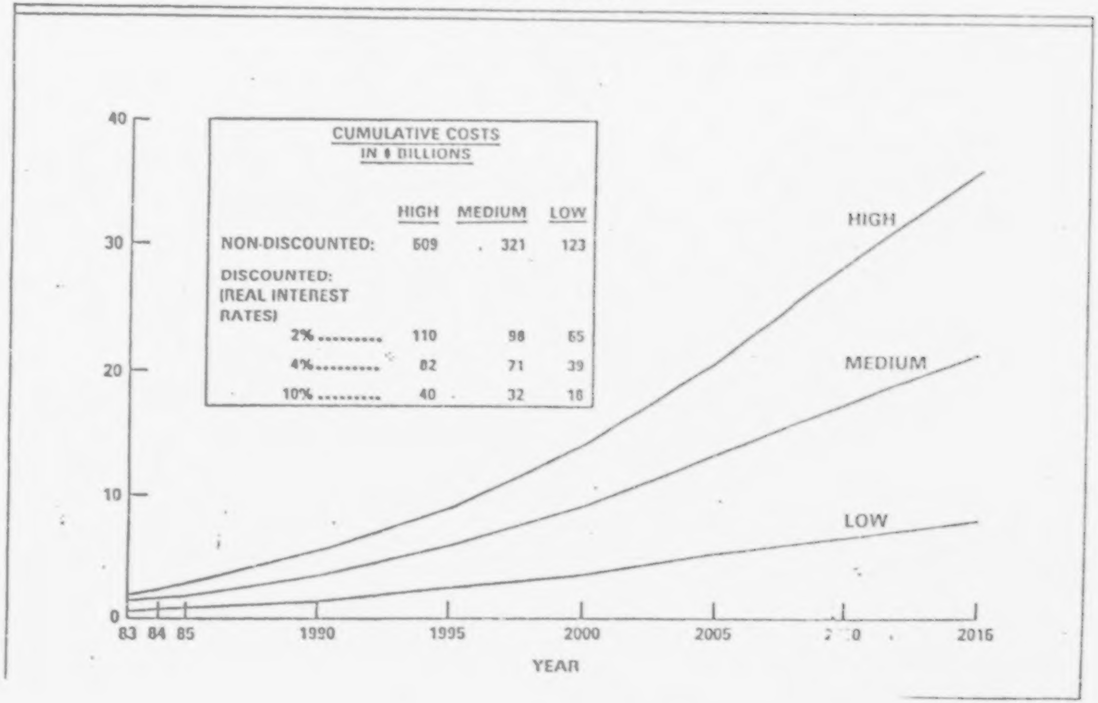
May 19, 1983

I. OVERVIEW

- The Administration is faced with increasing pressure for the creation of administrative compensation systems for persons suffering chronic injuries allegedly caused by exposure to toxic substances, including:
    - asbestos
    - radiation
    - Agent Orange
    - a wide variety of hazardous substances and wastes via Superfund personal injury compensation.
- Considerable legislative action can be expected in this Congress -- with highly publicized hearings and possible action late this year. Immediate concern is scheduled summer hearings on Superfund compensation legislation.
- A prime example is the Miller bill, which would provide compensation for persons exposed to asbestos.
    - A Department of Labor analysis shows potential outlays over next 30 years up to $500 billion, with potential annual expenditures as high as $30 billion/year by 2015. (See Chart 1.)
    - As written, Miller bill funds compensation claims solely through industry contributions, but eventual federal bail-out is inevitable. Bill would necessitate an immediate tax of $1,000/ton of asbestos, increasing to $3,200/ton by 1990 -- compared to current selling price of $340/ton.

ESTIMATED ANNUAL COSTS (HIGH, MEDIUM AND LOW) OF LEADING ASBESTOS OCCUPATIONAL COMPENSATION PROPOSAL 1983 — 2015

$ IN BILLIONS



CUMULATIVE COSTS IN $ BILLIONS

	HIGH	MEDIUM	LOW
NON-DISCOUNTED:	609	321	123
DISCOUNTED: (REAL INTEREST RATES)			
2%	110	98	65
4%	82	71	39
10%	40	32	18

II. HISTORICAL CONTEXT: PARALLELS WITH BREAKDOWN OF POLICY PRINCIPLES DURING EXPLOSIVE 1960'S AND 1970'S GROWTH OF ENTITLEMENTS

- Current toxic substances compensation debate replicates the 1970 era debates over social welfare entitlements. Latter were created with low initial costs, understated long-term cost estimates, and no appreciation for expansionary pressures being set in motion. Today, we are paying for the consequences of "small" steps taken without understanding of long-term policy and fiscal implications.

- Explosive post-1970 growth of Social Security disability and Black Lung compensation -- two existing disability compensation systems directly relevant to the current debate over toxic substances compensation -- underscores long-range threat.

- Just as the 1970's welfare expansion built on the breakdown of conceptual barriers that limited government support to the truly needy, the toxic substances compensation movement is built on the breakdown of principles that link compensation to fault.

- This is a logical consequence of the relentless movement of tort law during the 70's towards absolute liability. This trend is reflected in increasingly stringent product liability doctrines that have converted manufacturers into societal insurers, as well as in the increasing demand for government-sponsored compensation systems.

CHART 2

BLACK LUNG ANNUAL OBLIGATIONS 1970 - 1988 (EST.)

$ IN MILLIONS

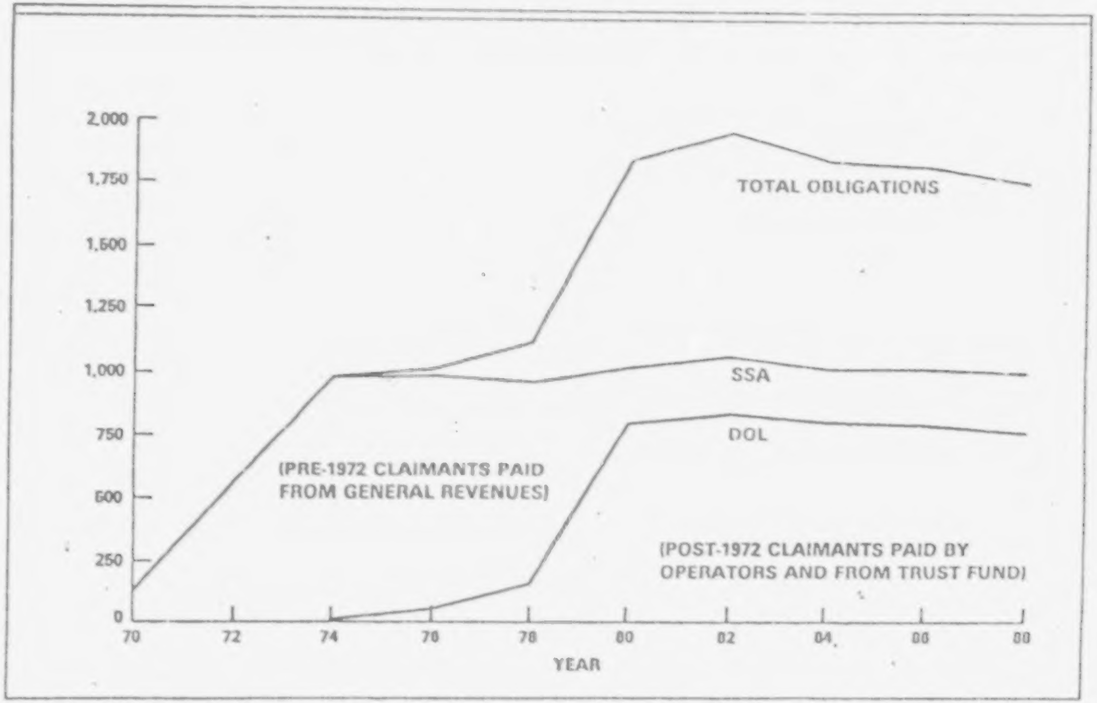


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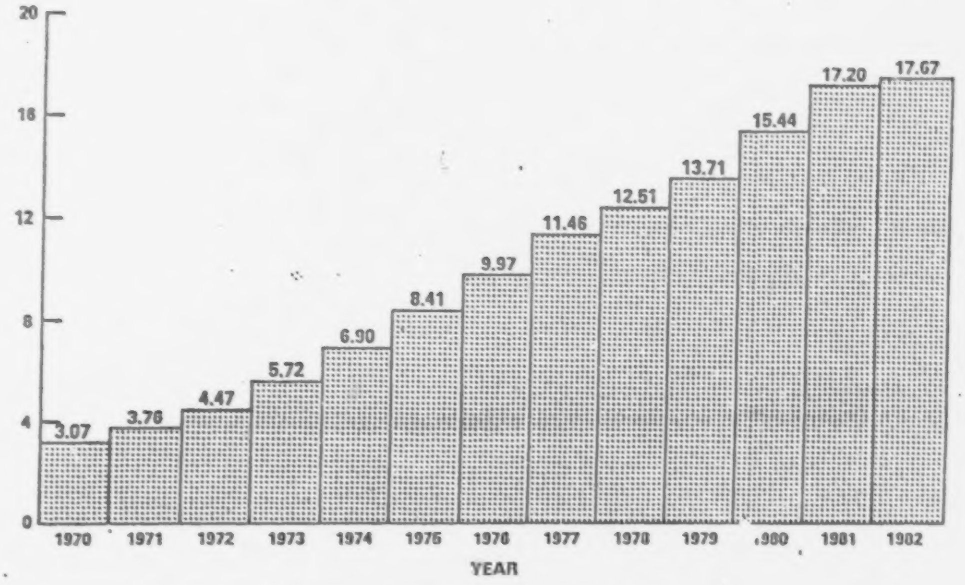
CHART 3

SOCIAL SECURITY DISABILITY INSURANCE CASH BENEFIT PAYMENTS 1970 - 1982

$ IN BILLIONS



HISTORICAL CONTEXT
(continued)

- The incipient movement towards federally financed toxic substances compensation embodies other parallels to 1970's social welfare entitlement expansions, including these:
    - Low initial cost estimates for various compensation schemes -- but once the "right" to government aid or rights of court action are established, the size and cost of programs become politically uncontrollable under constituency pressure for liberalization and expansion.
    - 1970's entitlement expansion preceded by a flood of reports and studies "documenting" the need for the new entitlements, often on the most tenuous of grounds. Dozens of similar studies of toxic torts and compensation are underway. Studies not supporting the case have limited impact, while those that find a need for compensation -- no matter how weak or suspect their conclusions -- serve as the foundation of media attention, hearings, initiatives, etc.
    - Resistence to "modest" first steps in the creation of major entitlement rights poses political difficulties of the highest order -- because it involves the political cost of resisting popular proposals now in order to avoid potentially heavy costs in the more distant future. Failure of earlier administrations to resist 1970's transfer payment expansions is now extremely costly. This Administration will face a similar test with toxic substances compensation -- as full costs will come on-stream in a major sense only during the next decade.
- Unfortunately, the Administration's ability to respond to the growing list of toxic substances compensation proposals is hampered by the lack of centralized policymaking. The Administration's response thus far has been fragmented, and largely determined by individual agencies' unique priorities. As a result, conflicting agency positions have undermined many of the Administration's legislative and litigation efforts.

III. ASBESTOS

- Asbestos Exposure
  - Approximately 13 to 15 million workers were exposed between 1940 and 1980.
  - Estimated total exposed population at 25 to 30 million.
  - Link to certain diseases, including lung cancer, has been definitively established.
- Litigation Status
  - Approximately 20,000 to 25,000 claims already filed.
  - 100,000 to 200,000 are expected in the next two to three decades (see Chart 4).
  - Industry litigation liability may be in excess of $30 billion (see Chart 5) -- probably more than industry assets and available insurance can cover.
- Fiscal Threat
  - Federal government now is perceived as the only available "deep pocket."
  - US already direct or cross defendant in cases involved in 18,000 claims (see Chart 6).
  - Government uniformly successful in court thus far, but lack of any other "deep pocket" makes the government's litigation position vulnerable.
  - Loss of a single case will generate explosion of litigation against US.

IART 4

BEST ESTIMATE OF TOTAL AND ANNUAL INDIVIDUAL ASBESTOS CLAIMS 1980 - 2015

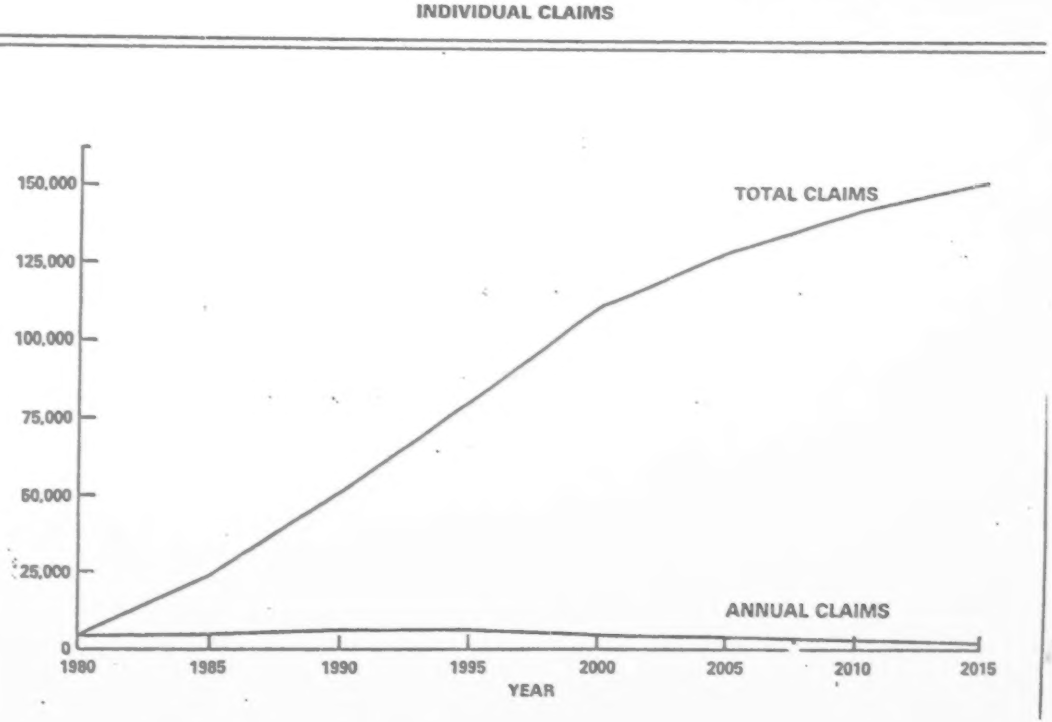


CHART 5

BEST ESTIMATE OF TOTAL ASBESTOS LITIGATION LIABILITY 1980 - 2015*

$ IN BILLIONS

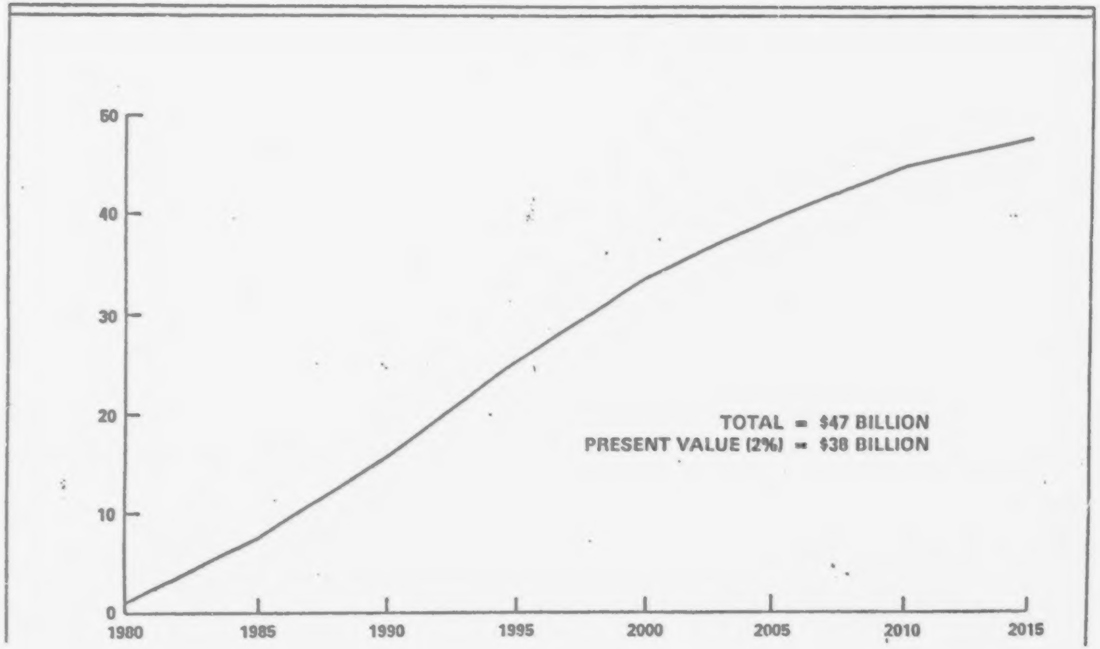
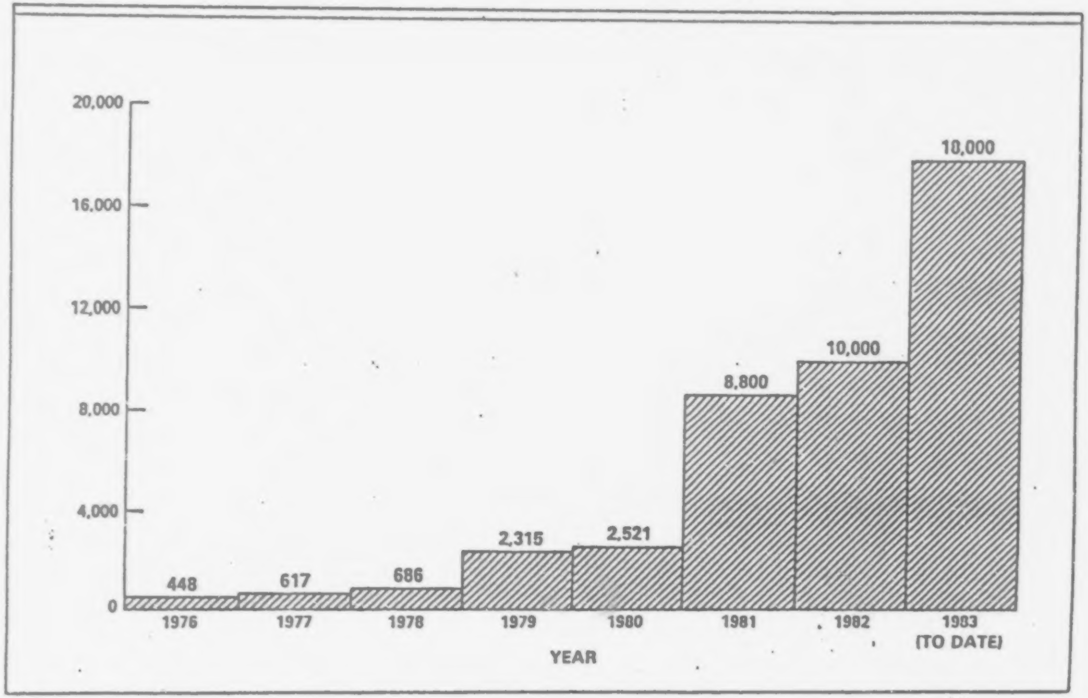


CHART 6

INDIVIDUAL ASBESTOS CLAIMS IN CASES INVOLVING UNITED STATES 1976 – 1983

INDIVIDUAL CLAIMS



IV. AGENT ORANGE

- Pending Agent Orange Compensation Bills -- Typical "foot-in-the-door" approaches advertised as "low-cost" or "procedural".
  - Daschle bill (H.R. 2017) requires compensation for virtually any Vietnam veteran suffering from three specific diseases purportedly related to Agent Orange exposure.
    - Because the medical evidence linking Agent Orange exposure to the three diseases is extremely tenuous, it will become nearly impossible to deny compensation for the myriad of other alleged Agent Orange diseases for which veterans are seeking compensation.
  - Cranston bill (S. 991) is purportedly "procedural," mandating rulemaking procedures to provide compensation for exposure to Agent Orange, low-level radiation and other "hazardous substances".
    - But Cranston bill heavily biases the ultimate result in favor of compensation by requiring formal rulemaking before allowing VA denial of claims, even as to a substance for which there is "insufficient medical or scientific evidence" to determine whether it caused a disease resulting in disability. The VA thus would be required to conduct hearings as to every conceivably toxic substance to which a veteran may have been exposed, and to reinstitute them as new "evidence" appears.
- Rapid Legislative Push Despite Comprehensive CDC Study Several Years Away
  - The two bills are moving forward even though initial results of the major epidemiological study by CDC still are several years off. But once Agent Orange disability compensation is provided, even if only for a handful of diseases, it may be impossible to bar compensation even if the study shows no long-term ill effects from Agent Orange exposure.

- Substantial Fiscal Exposure
  - Over 365,000 veterans are receiving Agent Orange medical care under a statutory standard that mandates such medical care even absent evidence linking the asserted disease to exposure.
  - Over 16,500 Agent Orange disability compensation claims already filed.
  - Since the VA presumes every Vietnam veteran to have been exposed absent evidence to the contrary, 2.5 million Vietnam veterans are potentially eligible for Agent Orange health care and disability compensation.
  - By making veterans' disability compensation available without firm evidence, the integrity of the entire compensation system is jeopordized. This endangers compensation for those war-injured veterans who most clearly deserve and need government support.

V. SUPERFUND

- Legislative Context
  - Currently Superfund is used for clean-up of hazardous sites -- not payment of compensation for personal injury. Compensation was a major issue when Superfund was enacted in 1980, but was eliminated in a Senate compromise.
  - Now facing strong legislative push for expansion of Superfund to serve as a pool to pay for tort law judgments and/or administrative compensation awards for persons exposed to hazardous substances and wastes off the workplace.
  - This will be major legislative issue with influential legislators, including Senate Committee Chairman Stafford, committed to pressing for sweeping legislative initiatives.
- Compensation Commission Report
  - As part of 1980 compromise, a Commission was established to study compensation. The Commission's recommendations adopted the worst of both worlds: an administrative compensation system with liberal evidentiary standards and generous benefits, as well as continued availability of traditional tort law.
- **Open-ended Fiscal Threat**
  - **Although its advocates now price Superfund compensation proposals at "only" a few billion dollars, the amounts at stake are literally open-ended and potentially enormous. Given the ambiguity of Superfund, compensation could be provided for virtually any environmental exposure, and not merely exposures directly related to waste disposal sites. Under the proposed compensation mechanisms substantial amounts will go to lawyers' fees -- creating a strong incentive for increased litigation. For example, one-half to two-thirds of asbestos industry pay-outs go to lawyers' fees and litigation expenses.**

VI. RADIATION

- o Renewed legislative efforts to provide compensation for low-level radiation exposure can be anticipated, fueled in part by anti-nuclear advocates, and in part by pork barrel politics.

  - o S. 921 (Hatch) provides a liberal litigation "remedy" against the United States, undermining many of the government's defenses in pending cases. The bill builds upon Section 7 of the Orphan Drug Act, which requires HHS to promulgate cancer risk assessments for persons exposed to low-level radiation.

  - o S. 11 (Cranston) requires the VA to conduct a major radiation epidemiological study. And, as noted, S. 991 (Cranston) calls for hearings, findings and broad discretion on the part of the VA Administrator to establish compensation awards.

- o The United States presently is involved in the downwind radiation litigations. One case alone, Allen v. United States, involves 1,269 claimants seeking $2 billion in damages. (See Chart 7.)

CHART 7

RADIATION LITIGATIONS INVOLVING UNITED STATES

Cases	Damage Claims
Allen v. United States (under advisement)	$2 billion
47 other pending suits in which the United States is a defendant	$827 million
31 pending suits against DOE contractor which the United States may have to indemnify for litigation losses	$4.78 billion

VII. OTHER TOXIC SUBSTANCES

- Exposure to other toxic substances on and off the workplace will continue to raise compensation issues. Other such toxic substances include dioxin, benzene, vinyl chloride, kepone, formaldehyde, PCB, PBB, as well as more common substances such as cotton dust (brown lung) and silica (white lung). Some of the most serious problem substances and exposure estimates are listed in the following table:

EXPOSURE ESTIMATES

SUBSTANCE	USE	WORKERS	RESIDENTS NEAR INDUSTRIAL OR WASTE SITES	LOW-LEVEL EXPOSURE
VINYL CHLORIDE	PLASTICS FLOOR TILES	MORE THAN 3.5 MILLION	4.6 MILLION	N/A
CARBON TETRACHLORIDE	INDUSTRIAL SOLVENT GRAIN FUMIGANT	3.4 MILLION	8 MILLION	41 MILLION
BENZENE	RAW CHEMICAL GASOLINE ADDITIVE	3 MILLION	N/A	OVER 75% OF POPULATION
FORMALDEHYDE	RESINS TEXTILES COSMETICS INSULATION	UP TO 2.8 MILLION	37.7 MILLION	N/A
CHROMIUM AND COMPOUNDS	ALLOYS PIGMENTS	2.5 MILLION	N/A	ENTIRE POPULATION
1,2 – DICHLOROETHANE	CHEMICAL INTERMEDIATE LEADED FUEL GRAIN FUMIGANT	UP TO 2 MILLION	N/A	ENTIRE POPULATION
POLYCHLORINATED BIPHENYLS (PCBs)	ELECTRICAL COOLING LIQUIDS	N/A	12 MILLION	WIDESPREAD CONTAMINATION
DIOXIN (TCDD)	PESTICIDE CONTAMINANT	N/A	7.7 MILLION	159 MILLION
NICKEL AND COMPOUNDS	ALLOYS ELECTRONICS EDIBLE OILS COINS	709 THOUSAND	720 THOUSAND	ENTIRE POPULATION
ARSENIC AND COMPOUNDS	PESTICIDES PAINTS HIDE PRESERVATION	545 THOUSAND	N/A	ENTIRE POPULATION
CHLOROFORM	SOLVENT DRUGS PESTICIDES	300 THOUSAND	N/A	N/A
CADMIUM AND COMPOUNDS	ELECTROPLATING BATTERIES FUNGICIDES	380 THOUSAND	N/A	SMOKERS
LEAD COMPOUNDS	PAINT HAIR DYES PLASTICS	UP TO 276 THOUSAND	N/A	N/A
ACRYLONITRILE	CHEMICAL INTERMEDIATE	279 THOUSAND	N/A	N/A
1,2 DIBROMOETHANE	LEADED GASOLINE SOLVENT PESTICIDES	UP TO 218 THOUSAND	N/A	650 THOUSAND GAS STATION ATTENDANTS
BERYLLIUM AND COMPOUNDS	ELECTRICAL COMPONENTS	26 THOUSAND	721 THOUSAND	N/A

VIII. POLICY FORMULATION PROBLEMS

- In formulating its policy and strategy on issues relating to toxic torts and compensation, the Administration will have to deal with a host of problems.

  - Piecemeal Legislative Action: Over a hundred bills involving a dozen or more committees can be expected. With the exception of Superfund and Agent Orange, the Administration is less likely to be faced with majors battle than a series of legislative skirmishes.

  - Adverse Judicial Decisions: The federal government's litigation position has been weakened. Of primary concern are legal inroads by manufacturers who are being sued under product liability doctrines, and in turn are suing the United States for indemnification. Equally troublesome are recent judicial decisions holding the federal government liable for "negligent inspections." Under these cases, the United States potentially may be liable for injuries caused by private persons or companies in light of federal responsibility to assure appropriate health safety levels.

  - Political Volatility: This may be one of the most politically sensitive issues of the 1980's. The compensation issue has been described as the next major item on the environmental agenda. This volatility is exacerbated by the fact that most legislative initiatives tend to be dominated by unrepresentative "horror stories."

  - Lack of Coordination Among the Federal Agencies: The Administration's ability to respond to developments has been severely hampered by a lack of coordination among affected federal agencies. In addition, there are dozens of working groups and ongoing studies in the area that are uncoordinated and sometimes directly in conflict. The absence of an internal mechanism for obtaining broad Administration overview of toxic torts and compensation policies has resulted in reactive ad hoc policies in response to individual developments.

IX. TOXIC TORTS/COMPENSATION WORKING GROUP

- There is a need for an inter-agency Working Group to develop and recommend Administration strategy and policy, and to coordinate Administration strategy, policy and data collection among the federal agencies. Its general charter might consist of the following:
  - Identification of issues and problems involved.
  - Identification, analysis and monitoring of relevant legislation and hearings.
  - Identification, analysis and monitoring of relevant litigation.
  - Identification and analysis of relevant agency proceedings.
  - Policy development and recommendation of Administration strategy.
  - Policy coordination among the various Executive Branch agencies.
  - Service as an information clearinghouse within the Administration for issues involving toxic torts and compensation.

  - The activities of such a Working Group will need to be consistent with statutory responsibilities and mandates of affected agencies, and the Executive Order 12291 process. Within that context, the full participation in the Working Group of all affected federal agencies will be necessary if it is to be effective in forestalling inherently uncontrollable long-term fiscal exposure.

Author: Robert Wilmore (OMB

COMPENSATION FOR TOXIC SUBSTANCES EXPOSURE

Work Group (Tozzi group will serve as Tech. staff)
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Uhlmann
Keyworth - Pres. Science Advisor
Rose

May 19, 1983

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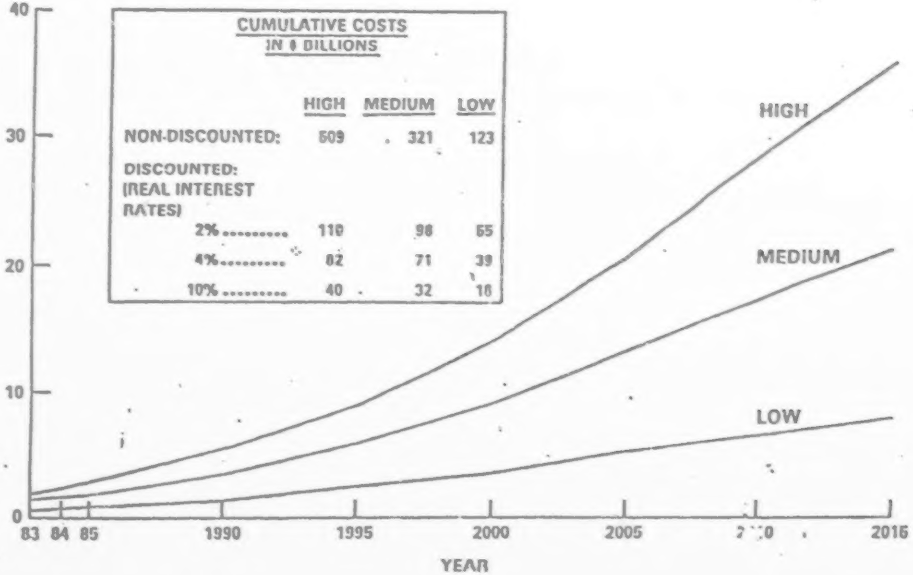
    - Agent Orange
    - a wide variety of hazardous substances and wastes via Superfund personal injury compensation.
- Considerable legislative action can be expected in this Congress -- with highly publicized hearings and possible action late this year. Immediate concern is scheduled summer hearings on Superfund compensation legislation.
- A prime example is the Miller bill, which would provide compensation for persons exposed to asbestos.
    - A Department of Labor analysis shows potential outlays over next 30 years up to $500 billion, with potential annual expenditures as high as $30 billion/year by 2015. (See Chart 1.)
    - As written, Miller bill funds compensation claims solely through industry contributions, but eventual federal bail-out is inevitable. Bill would necessitate an immediate tax of $1,000/ton of asbestos, increasing to $3,200/ton by 1990 -- compared to current selling price of $340/ton.

ESTIMATED ANNUAL COSTS (HIGH, MEDIUM AND LOW) OF LEADING ASBESTOS OCCUPATIONAL COMPENSATION PROPOSAL 1983 — 2015

$ IN BILLIONS



CUMULATIVE COSTS IN $ BILLIONS

	HIGH	MEDIUM	LOW
NON-DISCOUNTED:	509	321	123
DISCOUNTED: (REAL INTEREST RATES)			
2%	110	98	65
4%	82	71	39
10%	40	32	16

II. HISTORICAL CONTEXT: PARALLELS WITH BREAKDOWN OF POLICY PRINCIPLES DURING EXPLOSIVE 1960'S AND 1970'S GROWTH OF ENTITLEMENTS

o Current toxic substances compensation debate replicates the 1970 era debates over social welfare entitlements. Latter were created with low initial costs, understated long-term cost estimates, and no appreciation for expansionary pressures being set in motion. Today, we are paying for the consequences of "small" steps taken without understanding of long-term policy and fiscal implications.

o Explosive post-1970 growth of Social Security disability and Black Lung compensation -- two existing disability compensation systems directly relevant to the current debate over toxic substances compensation -- underscores long-range threat.

o Just as the 1970's welfare expansion built on the breakdown of conceptual barriers that limited government support to the truly needy, the toxic substances compensation movement is built on the breakdown of principles that link compensation to fault.

o This is a logical consequence of the relentless movement of tort law during the 70's towards absolute liability. This trend is reflected in increasingly stringent product liability doctrines that have converted manufacturers into societal insurers, as well as in the increasing demand for government-sponsored compensation systems.

CHART 2

BLACK LUNG ANNUAL OBLIGATIONS
1970 – 1988 (EST.)

$ IN MILLIONS

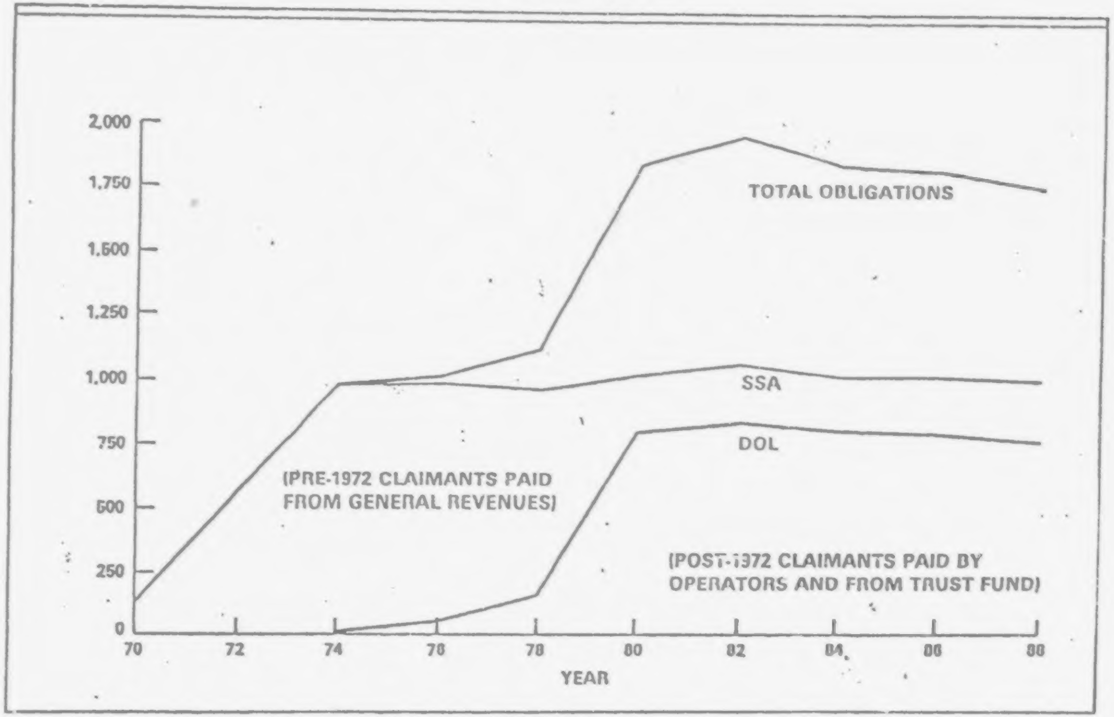
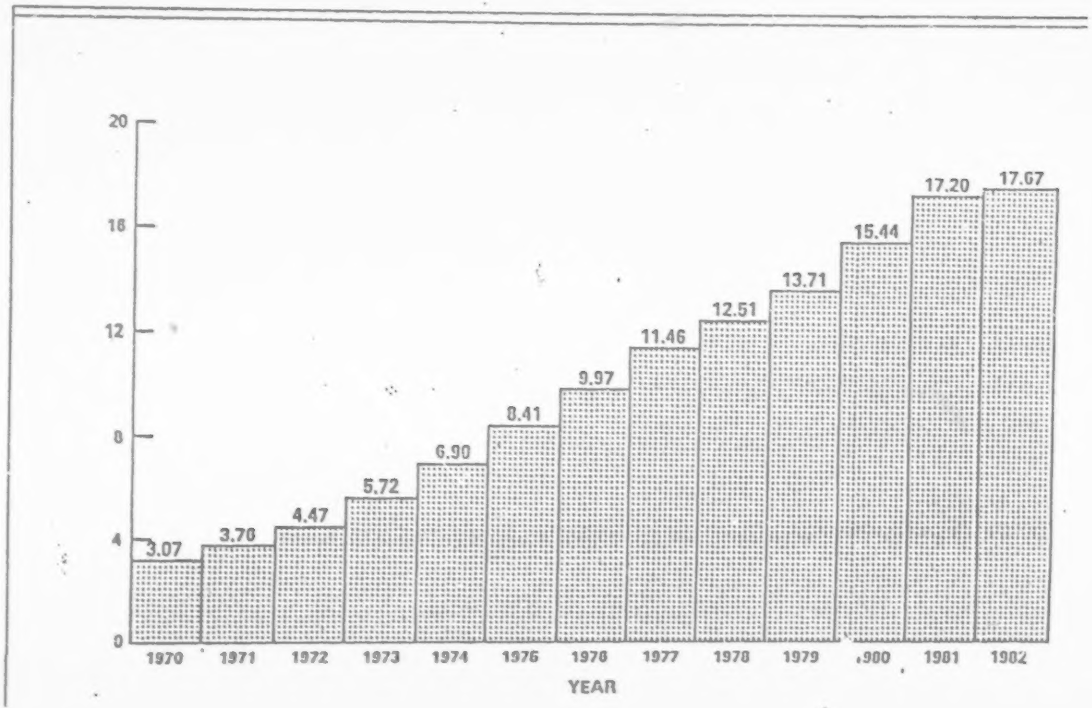


CHART 3

SOCIAL SECURITY DISABILITY INSURANCE CASH BENEFIT PAYMENTS 1970 - 1982

$ IN BILLIONS



HISTORICAL CONTEXT
(continued)

- The incipient movement towards federally financed toxic substances compensation embodies other parallels to 1970's social welfare entitlement expansions, including these:

  - Low initial cost estimates for various compensation schemes -- but once the "right" to government aid or rights of court action are established, the size and cost of programs become politically uncontrollable under constituency pressure for liberalization and expansion.

  - 1970's entitlement expansion preceded by a flood of reports and studies "documenting" the need for the new entitlements, often on the most tenuous of grounds. Dozens of similar studies of toxic torts and compensation are underway. Studies not supporting the case have limited impact, while those that find a need for compensation -- no matter how weak or suspect their conclusions -- serve as the foundation of media attention, hearings, initiatives, etc.

  - Resistence to "modest" first steps in the creation of major entitlement rights poses political difficulties of the highest order -- because it involves the political cost of resisting popular proposals now in order to avoid potentially heavy costs in the more distant future. Failure of earlier administrations to resist 1970's transfer payment expansions is now extremely costly. This Administration will face a similar test with toxic substances compensation -- as full costs will come on-stream in a major sense only during the next decade.

- Unfortunately, the Administration's ability to respond to the growing list of toxic substances compensation proposals is hampered by the lack of centralized policymaking. The Administration's response thus far has been fragmented, and largely determined by individual agencies' unique priorities. As a result, conflicting agency positions have undermined many of the Administration's legislative and litigation efforts.

III. ASBESTOS

- Asbestos Exposure
  - Approximately 13 to 15 million workers were exposed between 1940 and 1980.
  - Estimated total exposed population at 25 to 30 million.
  - Link to certain diseases, including lung cancer, has been definitively established.
- Litigation Status
  - Approximately 20,000 to 25,000 claims already filed.
  - 100,000 to 200,000 are expected in the next two to three decades (see Chart 4).
  - Industry litigation liability may be in excess of $30 billion (see Chart 5) -- probably more than industry assets and available insurance can cover.
- Fiscal Threat
  - Federal government now is perceived as the only available "deep pocket."
  - US already direct or cross defendant in cases involved in 18,000 claims (see Chart 6).
  - Government uniformly successful in court thus far, but lack of any other "deep pocket" makes the government's litigation position vulnerable.
  - Loss of a single case will generate explosion of litigation against US.

IART 4

BEST ESTIMATE OF TOTAL AND ANNUAL INDIVIDUAL ASBESTOS CLAIMS 1980 – 2015

INDIVIDUAL CLAIMS

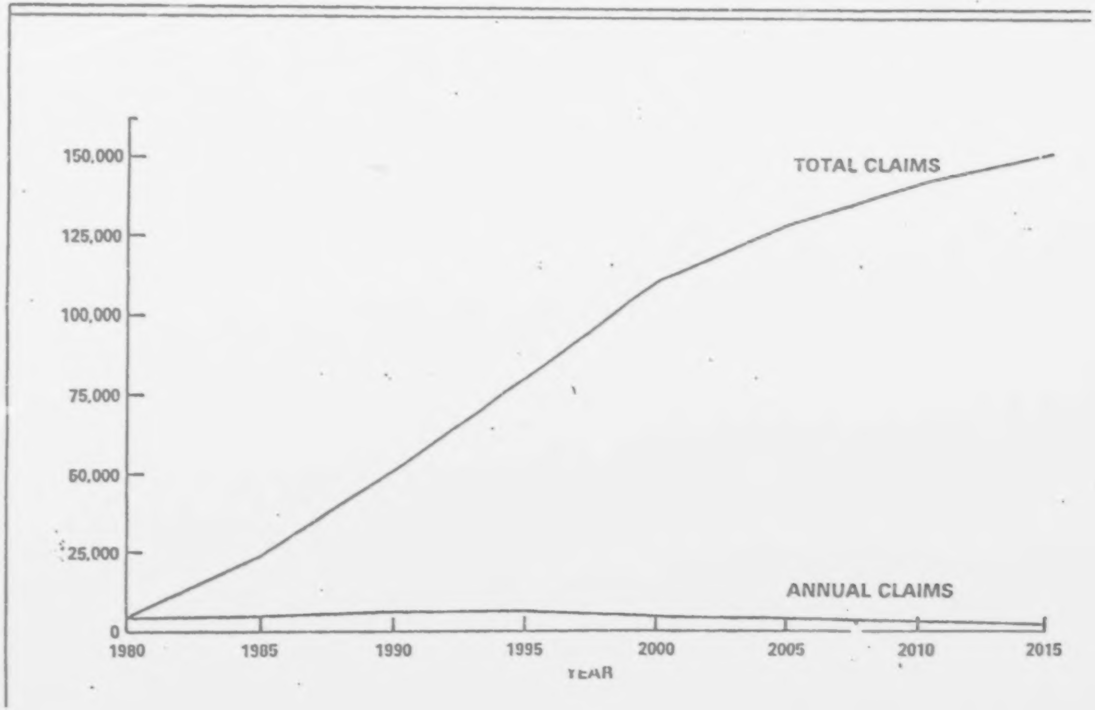
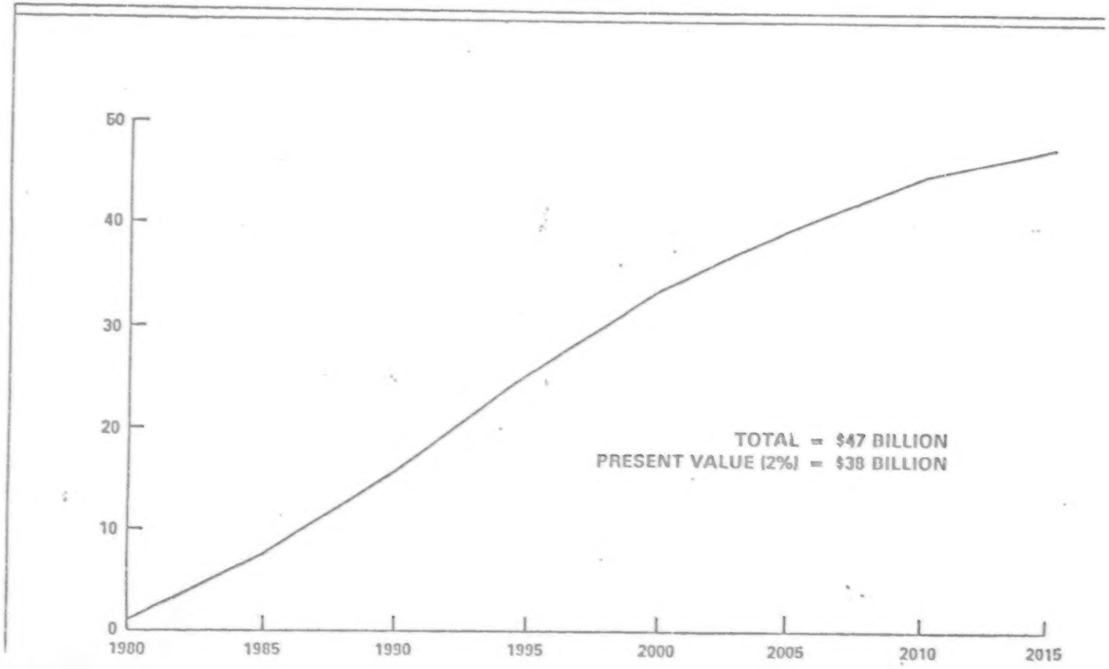


CHART 5

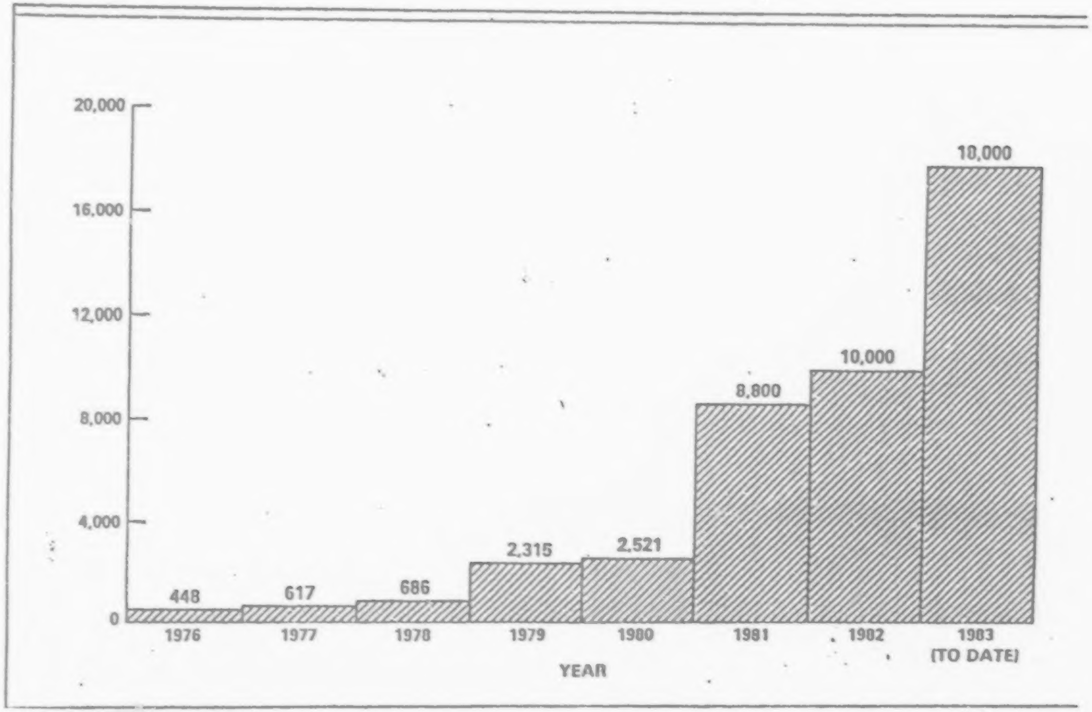
BEST ESTIMATE OF TOTAL ASBESTOS LITIGATION LIABILITY 1980 - 2015*

$ IN BILLIONS



INDIVIDUAL ASBESTOS CLAIMS IN CASES INVOLVING UNITED STATES 1976 – 1983

INDIVIDUAL CLAIMS



IV. AGENT ORANGE

- o Pending Agent Orange Compensation Bills -- Typical "foot-in-the-door" approaches advertised as "low-cost" or "procedural".

  - o Daschle bill (H.R. 2017) requires compensation for virtually any Vietnam veteran suffering from three specific diseases purportedly related to Agent Orange exposure.

    - o Because the medical evidence linking Agent Orange exposure to the three diseases is extremely tenuous, it will become nearly impossible to deny compensation for the myriad of other alleged Agent Orange diseases for which veterans are seeking compensation.

  - o Cranston bill (S. 991) is purportedly "procedural," mandating rulemaking procedures to provide compensation for exposure to Agent Orange, low-level radiation and other "hazardous substances".

    - o But Cranston bill heavily biases the ultimate result in favor of compensation by requiring formal rulemaking before allowing VA denial of claims, even as to a substance for which there is "insufficient medical or scientific evidence" to determine whether it caused a disease resulting in disability. The VA thus would be required to conduct hearings as to every conceivably toxic substance to which a veteran may have been exposed, and to reinstitute them as new "evidence" appears.

- o Rapid Legislative Push Despite Comprehensive CDC Study Several Years Away

  - o The two bills are moving forward even though initial results of the major epidemiological study by CDC still are several years off. But once Agent Orange disability compensation is provided, even if only for a handful of diseases, it may be impossible to bar compensation even if the study shows no long-term ill effects from Agent Orange exposure.

- Substantial Fiscal Exposure
  - Over 365,000 veterans are receiving Agent Orange medical care under a statutory standard that mandates such medical care even absent evidence linking the asserted disease to exposure.
  - Over 16,500 Agent Orange disability compensation claims already filed.
  - Since the VA presumes every Vietnam veteran to have been exposed absent evidence to the contrary, 2.5 million Vietnam veterans are potentially eligible for Agent Orange health care and disability compensation.
  - By making veterans' disability compensation available without firm evidence, the integrity of the entire compensation system is jeopordized. This endangers compensation for those war-injured veterans who most clearly deserve and need government support.

V. SUPERFUND

- Legislative Context
    - Currently Superfund is used for clean-up of hazardous sites -- not payment of compensation for personal injury. Compensation was a major issue when Superfund was enacted in 1980, but was eliminated in a Senate compromise.
    - Now facing strong legislative push for expansion of Superfund to serve as a pool to pay for tort law judgments and/or administrative compensation awards for persons exposed to hazardous substances and wastes off the workplace.
    - This will be major legislative issue with influential legislators, including Senate Committee Chairman Stafford, committed to pressing for sweeping legislative initiatives.
- Compensation Commission Report
    - As part of 1980 compromise, a Commission was established to study compensation. The Commission's recommendations adopted the worst of both worlds: an administrative compensation system with liberal evidentiary standards and generous benefits, as well as continued availability of traditional tort law.
- Open-ended Fiscal Threat
    - Although its advocates now price Superfund compensation proposals at "only" a few billion dollars, the amounts at stake are literally open-ended and potentially enormous. Given the ambiguity of Superfund, compensation could be provided for virtually any environmental exposure, and not merely exposures directly related to waste disposal sites. Under the proposed compensation mechanisms substantial amounts will go to lawyers' fees -- creating a strong incentive for increased litigation. For example, one-half to two-thirds of asbestos industry pay-outs go to lawyers' fees and litigation expenses.

VI. RADIATION

- Renewed legislative efforts to provide compensation for low-level radiation exposure can be anticipated, fueled in part by anti-nuclear advocates, and in part by pork barrel politics.

    - S. 921 (Hatch) provides a liberal litigation "remedy" against the United States, undermining many of the government's defenses in pending cases. The bill builds upon Section 7 of the Orphan Drug Act, which requires HHS to promulgate cancer risk assessments for persons exposed to low-level radiation.

    - S. 11 (Cranston) requires the VA to conduct a major radiation epidemiological study. And, as noted, S. 991 (Cranston) calls for hearings, findings and broad discretion on the part of the VA Administrator to establish compensation awards.

- The United States presently is involved in the downwind radiation litigations. One case alone, Allen v. United States, involves 1,269 claimants seeking $2 billion in damages. (See Chart 7.)

CHART 7

RADIATION LITIGATIONS INVOLVING UNITED STATES

Cases	Damage Claims
Allen v. United States (under advisement)	$2 billion
47 other pending suits in which the United States is a defendant	$827 million
31 pending suits against DOE contractor which the United States may have to indemnify for litigation losses	$4.78 billion

VII. OTHER TOXIC SUBSTANCES

- o Exposure to other toxic substances on and off the workplace will continue to raise compensation issues. Other such toxic substances include dioxin, benzene, vinyl chloride, kepone, formaldehyde, PCB, PBB, as well as more common substances such as cotton dust (brown lung) and silica (white lung). Some of the most serious problem substances and exposure estimates are listed in the following table:

EXPOSURE ESTIMATES

SUBSTANCE	USE	WORKERS	RESIDENTS NEAR INDUSTRIAL OR WASTE SITES	LOW LEVEL EXPOSURE
VINYL CHLORIDE	PLASTICS FLOOR TILES	MORE THAN 3.6 MILLION	4.6 MILLION	N/A
CARBON TETRACHLORIDE	INDUSTRIAL SOLVENT GRAIN FUMIGANT	3.4 MILLION	8 MILLION	41 MILLION
BENZENE	RAW CHEMICAL GASOLINE ADDITIVE	3 MILLION	N/A	OVER 75% OF POPULATION
FORMALDEHYDE	RESINS TEXTILES COSMETICS INSULATION	UP TO 2.8 MILLION	27.7 MILLION	N/A
CHROMIUM AND COMPOUNDS	ALLOYS PIGMENTS	2.5 MILLION	N/A	ENTIRE POPULATION
1,2 – DICHLOROETHANE	CHEMICAL INTERMEDIATE LEADED FUEL GRAIN FUMIGANT	UP TO 2 MILLION	N/A	ENTIRE POPULATION
POLYCHLORINATED BIPHENYLS (PCBs)	ELECTRICAL COOLING LIQUIDS	N/A	12 MILLION	WIDESPREAD CONTAMINATION
DIOXIN (TCDD)	PESTICIDE CONTAMINANT	N/A	7.7 MILLION	169 MILLION
NICKEL AND COMPOUNDS	ALLOYS ELECTRONICS EDIBLE OILS COINS	709 THOUSAND	720 THOUSAND	ENTIRE POPULATION
ARSENIC AND COMPOUNDS	PESTICIDES PAINTS HIDE PRESERVATION	545 THOUSAND	N/A	ENTIRE POPULATION
CHLOROFORM	SOLVENT DRUGS PESTICIDES	360 THOUSAND	N/A	N/A
CADMIUM AND COMPOUNDS	ELECTROPLATING BATTERIES FUNGICIDES	300 THOUSAND	N/A	SMOKERS
LEAD COMPOUNDS	PAINT HAIR DYES PLASTICS	UP TO 278 THOUSAND	N/A	N/A
ACRYLONITRILE	CHEMICAL INTERMEDIATE	278 THOUSAND	N/A	N/A
1,2 DIBROMOETHANE	LEADED GASOLINE SOLVENT PESTICIDES	UP TO 218 THOUSAND	N/A	650 THOUSAND GAS STATION ATTENDANTS
BERYLLIUM AND COMPOUNDS	ELECTRICAL COMPONENTS	26 THOUSAND	721 THOUSAND	N/A

VIII. POLICY FORMULATION PROBLEMS

- In formulating its policy and strategy on issues relating to toxic torts and compensation, the Administration will have to deal with a host of problems.
  - Piecemeal Legislative Action: Over a hundred bills involving a dozen or more committees can be expected. With the exception of Superfund and Agent Orange, the Administration is less likely to be faced with majors battle than a series of legislative skirmishes.
  - Adverse Judicial Decisions: The federal government's litigation position has been weakened. Of primary concern are legal inroads by manufacturers who are being sued under product liability doctrines, and in turn are suing the United States for indemnification. Equally troublesome are recent judicial decisions holding the federal government liable for "negligent inspections." Under these cases, the United States potentially may be liable for injuries caused by private persons or companies in light of federal responsibility to assure appropriate health safety levels.
  - Political Volatility: This may be one of the most politically sensitive issues of the 1980's. The compensation issue has been described as the next major item on the environmental agenda. This volatility is exacerbated by the fact that most legislative initiatives tend to be dominated by unrepresentative "horror stories."
  - Lack of Coordination Among the Federal Agencies: The Administration's ability to respond to developments has been severely hampered by a lack of coordination among affected federal agencies. In addition, there are dozens of working groups and ongoing studies in the area that are uncoordinated and sometimes directly in conflict. The absence of an internal mechanism for obtaining broad Administration overview of toxic torts and compensation policies has resulted in reactive ad hoc policies in response to individual developments.

IX. TOXIC TORTS/COMPENSATION WORKING GROUP

- There is a need for an inter-agency Working Group to develop and recommend Administration strategy and policy, and to coordinate Administration strategy, policy and data collection among the federal agencies. Its general charter might consist of the following:

  - Identification of issues and problems involved.
  - Identification, analysis and monitoring of relevant legislation and hearings.
  - Identification, analysis and monitoring of relevant litigation.
  - Identification and analysis of relevant agency proceedings.
  - Policy development and recommendation of Administration strategy.
  - Policy coordination among the various Executive Branch agencies.
  - Service as an information clearinghouse within the Administration for issues involving toxic torts and compensation.

  - The activities of such a Working Group will need to be consistent with statutory responsibilities and mandates of affected agencies, and the Executive Order 12291 process. Within that context, the full participation in the Working Group of all affected federal agencies will be necessary if it is to be effective in forestalling inherently uncontrollable long-term fiscal exposure.

EXCERPT FROM

INJURIES AND DAMAGES FROM HAZARDOUS WASTES -- ANALYSIS AND IMPROVEMENT OF LEGAL REMEDIES

A Report to Congress in Compliance with §301(e) of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (P.L. 96-510, December 11, 1980) by the "Superfund §301(e) Study Group"

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George C. Freeman, Jr.
Richard F. Gerry
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Jeffrey O'Connell
Warren R. Spannaus

Members of the Study Group

July 1, 1982

Fourth Recommendation: Tier One -- Rebuttable Presumptions

The Study Group recommends that legislation establishing the compensation system provide for two kinds of rebuttable presumptions; the first extending merely to the issue of causation, the second going beyond proof of causation into such issues as the nature and extent of the disease or injury resulting from exposure to hazardous wastes. The first set of presumptions would be based on the proof of several elements which then give rise to rebuttable presumptions of causation. The second set of presumptions should be based on the proof of the same elements as well as on previously prepared and adopted "Toxic Substance Documents" based on scientific data that link the exposure to certain designated hazardous wastes to their known effects in causing injury or disease. The second set of presumptions would also meet additional elements of proof.

In both instances, the same basic showing should be made. If the claimant in an administrative compensation proceeding proves:

1) that the source involved in the claim was engaged at the time of exposure in the production, transportation or disposal of hazardous wastes; and

2) that the claimant was exposed to such hazardous waste by proximity, contact, ingestion, inhalation, or by some other form of exposure; and

3) that the claimant suffered death or the kind of injury or disease which is known to result from such exposure then the following rebuttable presumptions should arise: (a) that the exposure to the hazardous waste generated, transported or disposed of, by or in the source, contributed to the death, injury or disease of the claimant; and (b) that the source by or in which such hazardous wastes were generated, transported or disposed of was responsible for the claimant's exposure and for the resulting death, injury or disease.

In the rebuttable presumption set forth above, to show that death or the kind of injury or disease suffered by the claimant is "known to result" from such exposure, a Toxic Substance Document (as described infra) shall be relied on, if such a document has been prepared for the specific hazardour substance or waste.

In addition to these basic presumptions, additional presumptions may apply if the federal agency authorized to administer the compensation system (which will be operated by the states subject to federal statutory requirements), has issued documents to provide evidentiary data showing that specific hazardous substances or wastes cause specific diseases or illnesses.

Such documents, here referred to as Toxic Substance Documents should be prepared by the agency on the basis of scientific data, including its accumulated information on particular hazardous wastes or substances, collected in connection with the administration of the Clean Air Act (CAA), the Clean Water Act (CWA), Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), Resource Conservation and Recovery Act (RCRA), Toxic Substances Control Act (TOSCA), Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), as well as the Occupational Safety and Health Act (OSHA), and on the basis of evidence collected in connection with past or pending cases. The Agency should produce Toxic Substance Documents for some of the best known 20 or 30 hazardous substances on which data are available, including dioxin, chromium, kepone and PCB, and that the Agency could add Toxic Substance Documents from time to time for additional hazardous substances or wastes as their danger becomes more evident, and as data become available. A Toxic Substance Document should be adopted following the publication of the proposed document, together with all supporting data and materials, set forth in full or by reference to published scientific papers, and should be adopted and promulgated following an opportunity for comment.[20] An appropriate rulemaking procedure should be followed to assure the scientific accuracy of the results.

For each hazardous substance or waste, the applicable Toxic Substance Document should set forth the following:

TSD's

1) Elements of exposure including

a) nature of exposure, such as skin contact, ingestion, inhalation;

b) level of exposure, stated, where appropriate, in terms of a range of exposure levels expressive of the intensity of the exposure measured, when appropriate, by ambient concentration;

c) duration of exposure, expressed, where appropriate, in terms of a range of time periods of exposure.

2) The disease or diseases resulting from exposure, including data on:

a) the etiology of the disease, as related to nature, level and duration of exposure;

b) the symptoms of the disease and the available methods for its diagnosis, including direct observation, clinical and other diagnostic tests;

c) the latency period, if any, of the disease, as related to the nature, level and duration of exposure;

d) the prognosis for the disease, including the anticipated course of the disease, its treatment and its impact or duration in terms of disability, reduction or loss of function, and reduction of life expectancy;

e) other impacts, including mutagenicity, teratogenicity and carcinogenicity in the progeny of the person exposed.

(3) Other parameters relating to exposure, etiology and the course and consequences of the disease, including aspects of susceptibility and their variation depending on such factors as age and sex and such other conditions as pregnancy, or disease from causes other than exposure to the hazardous wastes or substances involved.

When a Presumption Document has been prepared for a particular hazardous waste, upon proof of exposure, source and proof of the occurrence of death or disease -- as in the case of the rebuttable presumption of causation --additional rebuttable presumptions shall apply as to matters of exposure, etiology, prognosis, nature and duration of the disease, insofar as these matters are covered in the Toxic Substance Document and insofar as they may be relevant or material to the claim.

Discussion

1. The Recommendations

A majority of the Study Group was in agreement on the need for reliance on rebuttable presumptions on the First Tier, as part of the compensation remedy. The Study Group was in general agreement that the rebuttable presumptions here recommended for the Tier One compensation remedy are inappropriate for the Tier Two plenary tort action. Several members of the Group expressed strong doubts on the desirability of statutory presumptions, and some members suggested

REPORT ON THE FEASIBILITY OF ORGANIZING A STUDY TO ASSESS THE POTENTIAL HEALTH HAZARDS OF WASTE DISPOSAL SITES WITHIN THE UNITED STATES

Submitted by

UNIVERSITIES ASSOCIATED FOR RESEARCH AND EDUCATION
IN PATHOLOGY, INC.

June 22, 1983

UAREP, with technical assistance from the Life Sciences Research Office (LSRO) of the Federation of American Societies for Experimental Biology (FASEB), has completed the feasibility study requested by CMA. A detailed report, presented in two parts, is submitted herewith. Part I describes the feasibility study, and Part II describes the proposed comprehensive review. We have concluded that within the time constraint of 10 months it is feasible to carry out a review of existing data and prepare a report covering the following general areas:

1. Prepare a list of the chemicals most likely to occur in a majority of waste disposal sites and which are most likely to create health hazards.

2. Identify the acute and, to a limited extent, the long-term toxic effects likely to result from human exposures to such chemicals. This would largely be based on published data in the peer review literature and make use of certain secondary sources of material available from industry as well as state and federal agencies.

3. Estimate the levels and duration of exposure to these chemicals at or above which toxic effects might be expected.

4. Identify those clinical and laboratory investigations which are likely to be most useful in detecting health effects in exposed populations and the epidemiological techniques to be utilized in so doing.

5. Develop scientific criteria for identifying immediate health hazards requiring urgent investigation or attention.

In carrying out such a review UAREP proposes to utilize a blue-ribbon panel of senior scientists and two special task forces assisted by professional and support staff.

We believe the review can be completed in 8 to 10 months, that adequate data is available and that the scientific and professional expertise necessary for organization of the panels, task forces, and staff can be obtained.

Kenneth H. Endicott

Kenneth H. Endicott, M.D.
Executive Officer

I. REPORT OF UAREP FEASIBILITY STUDY GROUP TO CMA ON THE POSSIBILITY OF ORGANIZING A STUDY TO ASSESS THE POTENTIAL HEALTH HAZARDS OF WASTE DISPOSAL SITES WITHIN THE UNITED STATES

A. INTRODUCTION

Public concern over the potential health hazards of waste disposal throughout the United States has reached a high level. The U.S. Congress and several state legislatures have enacted laws aimed at identifying hazardous sites and taking remedial action. Several thousand sites have been identified and the U.S. Environmental Protection Agency has designated that over 400 require "priority attention".

The problem of hazardous wastes has been the subject of considerable media coverage but the scientific documentation is more limited. Among the scientific publications are "Assessment of Health Effects at Chemical Disposal Sites" (Life Science and Public Policy Program of the Rockefeller University, 1981), and "Management of Hazardous Industrial Wastes: Research and Development Needs" (National Research Council, 1983). The former emphasizes the difficulties of making risk assessments in view of the complications involved in evaluating exposures. The latter reviews and makes certain recommendations regarding waste disposal and also recommends that "both public and environmental risks should be covered in a manner that allows comparison with existing risks that are acceptable to society," and that "an educational program be established to provide the public and industry with soundly based information on the options and risks that are acceptable to society" (page 14). The technical and practical problems of health effects on an individual basis are outlined in the study of the Love Canal (EPA, 1980) and, more recently, Heath (1983) has examined the epidemiology difficulties.

Whereas the National Research Council study does provide a certain consensus regarding the generic and practical issues involved in waste management, the nature and extent of health problems associated with waste disposal sites are much more controversial. Accordingly, though a small number of sites have been the subject of intensive study, the relevance and extent of these findings as related to the problem on a national scale is very uncertain and requires more intensive investigation.

In view of these facts and the delay in carrying out adequate in-depth studies of health effects on an individual waste disposal site basis and the need to obtain more factual information of potential health effects, the Chemical Manufacturers Association (CMA) concluded that it would be of value to all concerned if a group of knowledgeable and prestigious scientists could examine available scientific data and reach an interim conclusion regarding the nature and extent of health hazards which may exist at waste disposal sites as a basis for interim strategy and policy. Further, such a group would outline the scientific criteria for identifying potentially hazardous sites and indicate the methodology of investigation.

In March 1983, CMA entered into an agreement with Universities Associated for Research and Education in Pathology, Inc. (UAREP) to carry out a Feasibility Study to learn how and whether such aims might be achieved through the organization of a scientific review based on the expertise of the academic community.

B. SCOPE OF THE FEASIBILITY STUDY

A small group was set up within UAREP and the Life Sciences Research Office (LSRO) of the Federation of American Societies for Experimental Biology (FASEB) to initiate the feasibility study. The goals of the group were (1) to determine what the aims and objectives of such a scientific study would be, i.e., health effects, exposures, etc.; (2) to determine how such a study would be organized and conducted; (3) to identify sources of information relative to (1) and (2) above; and (4) if possible, to identify what additional studies should be considered, if necessary. The group took into consideration the following areas as pertinent to the objectives of such a scientific review:

1. Information on i) the criteria of health effects to be used in evaluating endpoints of hazards, ii) the methodology of determining how such causal associations are to be determined, and iii) the extent and limitations of the universe of substances likely to be associated with waste disposal sites and their potential exposures for humans.

2. Identification of sources of data, as well as their relevance and potential significance in assessing health effects in waste disposal sites.

3. Information as to the availability of appropriate scientific experts in academia who might be available to participate in carrying out the study.

4. Information relevant to the organization of the interim study.

Initially, the Feasibility Study Group identified a number of parameters that might be addressed during the scientific review, including:

1. Determination of what inferences could be drawn from knowledge of hazardous chemical substances present in waste disposal sites and the probability of human exposures in relation to all types of health effects.

2. Identification of what inferences can be drawn regarding the effects of such substances based on other studies on humans or on animals, i.e., in environmental, occupational, or other settings, and included reports from existing waste disposal site studies. This also included the problem of assessing the limitations of epidemiology and other techniques in evaluating the effects of low level exposures to potential agents.

3. The possibility of carrying out preliminary correlation surveys based on i) known waste disposal sites, ii) studies on states known to have a high degree of environmental pollution, and iii) correlation studies based on general investigations in the U.S. in terms of pollution whether urban or rural, and involving air or water.

4. The possibility of the interim group establishing scientific criteria for identifying the existence of imminent health hazards in relation to chemical waste disposal sites and what would be the criteria or endpoints in terms of clinical and pathological data.

5. The possibility of the study group indicating further areas as requiring investigation in depth for the purpose of making the final reports more meaningful.

These various points were examined and an appropriate planning document prepared and circulated to a number of senior scientists as a background for discussion.

A number of experts representative of epidemiology, environmental waste disposal, toxicology, experimental biology, and biostatistics were approached (see Appendix I). In addition, a number of individuals in the field of experimental pathology and two representatives of environmental organizations were contacted. A considerable amount of effort was expended on contacting individuals within the Environmental Protection Agency (EPA) to determine the data and material available in its archives. The CMA advised UAREP/LSRO on contacts within the chemical industry; member companies of CMA prepared and submitted material for consideration by the Feasibility Study Group. Some individuals prepared reports and submitted them to UAREP/LSRO. Others were visited by members of the Feasibility Study Group or visited UAREP in Bethesda.

C. VIEWS OF EXPERTS

As anticipated, there was a wide range of opinions regarding the objectives and organization of any study relative to waste disposal sites. All were agreed on the inherent difficulties involved, but differed as to their importance in relation to an overall review. Some emphasized that if the problem was to be investigated, it should be done well or not at all. The majority agreed that, by far, the best way to investigate the overall problem was to carry out systematic examinations of individual sites. However, some experts also recognized the logistical difficulties associated with doing so in the immediate future, and concluded that some form of interim scientific evaluation of the extent and nature of the health problem was essential to provide some basis for rational public health policies and strategies in the next few years. For example, the Centers for Disease Control (CDC) have investigated only five sites in depth, although there is a considerable amount of material available on an additional twenty. All experts emphasized the fundamental importance of determining human exposures in relation to waste disposal sites, but none could offer exact guidance as to how this might be accomplished except through study of individual sites.

The point was raised by a number of individuals that in the past, few human health effects have been demonstrated to be caused by waste disposal sites and, therefore, it is probable that despite considerable efforts, little additional information may be obtained in future efforts of this type. If true, such a finding is clearly of importance in terms of future public health strategy and control of waste disposal sites since it suggests that more emphasis should be placed on methods of diminishing waste disposal, as done by the Japanese, than on the investigation and clean-up of existing sites. Many consultants indicated their doubt that the public will readily accept such a conclusion in view of preconceptions as to health hazards which have developed over the last several years and because of extensive media coverage of "crises". These aspects undergird the importance of ensuring the credibility of any study which carries out an overall investigation of this problem.

D. IDENTIFICATION OF POTENTIAL DATA SOURCES

The Feasibility Study Group discussed sources of data with the consultants and carried out preliminary surveys of the general literature. UAREP/LSRO staff members reviewed data retrieved from computerized literature services (Medline, Cancerline, Toxline, and Enviroline). They also reviewed such publications as the monograph series of the International Agency for Research on Cancer, and the Environmental Health Criteria documents of the World Health Organization on toxic hazards. In addition, a number of key articles were reviewed relative to the availability of specific material regarding waste disposal sites (*see* Appendix II). In brief, it can be stated that the amount of material available in the literature with respect to specific studies on hazardous waste disposal sites is very small. On the other hand, there are considerable data available regarding individual chemicals and their health effects based on a variety of studies including animal toxicology and epidemiology studies of occupational and other populations which might be of use for drawing inferences as to possible health effects.

There is a large amount of material available in the CDC on the potential health effects of waste disposal sites. Whereas some of it has been published in the general literature, the greater part is available as internal technical reports. However, the CDC staff indicated that this material would be made available to UAREP, and they would be pleased to collaborate directly with the UAREP study group in evaluating and identifying data pertinent to the overall problem. The experts at CDC were of the opinion that only waste disposal sites at Love Canal, New York; Triana, Alabama; Woburn, Massachusetts; and Times Beach, Missouri had been investigated adequately.

Key EPA personnel were contacted but little additional information seemed to be readily available regarding the problem, despite the fact that other experts indicated to the Feasibility Group that EPA has the best data on exposures. Several EPA contacts indicated that the desired information might be obtained from the CDC in Atlanta, but the CDC stated they had exposure data only for sites in which they had carried out some initial health investigations. CDC referred UAREP back to EPA. In conclusion, it proved impossible to identify who in EPA had such material; it was decided to await further approaches until after the organization of a review group.

Industry made material available to UAREP regarding exposures to suspected problem chemicals in the work place. A number of reprints and drafts of studies underway were made available to the Feasibility Study Group which were clearly pertinent and valuable in drawing inferences (see Appendix II). Key reference material is identified in Appendix III. In some cases these documents can be amplified by the literature in peer review journals. These data will be valuable and pertinent relative to information on the potential effects at specific exposure levels, but such data again raise the perennial question of how to deal with the effects of exposure at very low levels which would be fundamental to much of the assessment of the study group.

Preliminary contacts were made in the United Kingdom, and it would appear that within the European Common Market a considerable amount of data have been collected relative to hazardous waste disposal. Apparently, studies have also been carried out on potential health effects in some areas, but relatively little has been published. It is intended to investigate in further depth what material is available later this year, but preliminary investigations indicate that a number of individuals in Europe will be eager to collaborate in this context.

In conclusion, there appears to be less published data regarding the specific effects of waste disposal sites than had been originally anticipated. Although it is clear that much pertinent information is present in archives of various sorts, it will require a concerted effort to obtain and analyze this material. On the other hand, a considerable amount of published literature is available which contains important material on which inferences may be drawn by appropriate experts regarding potential health effects. The weakest area appears to be in relation to potential exposure levels. In this context, preliminary correlation studies may be of value. The Feasibility Study Group further explored what already had been done in this context.

1. Studies of Individual Waste Sites

While all agreed that a comprehensive study of individual waste disposal sites would be the most satisfactory rational method in identifying or excluding potential health effects, to do so would require extensive resources in manpower and expertise and a considerable period of time to implement. Clearly, it is exceedingly difficult to evaluate the potential danger of a waste disposal site unless accurate information, not only on the chemicals present but also on the probable degree of human exposure, is available. This suggests that for the study it will be necessary to draw up some general guidelines as to possible approaches. It will be highly desirable to explore what material is available within CDC in the form of internal reports in this context. CDC had considered but dropped the possibility of using certain chemicals as indicators of pollution. These CDC reports cover the PCB problem in the North Hollywood dump in Tennessee; the PBB problem in Michigan; the asbestos problem in Globe, Arizona; arsenic at Woburn, Massachusetts; and in a mixed deposit at Tar Creek, Kansas and Oklahoma. The reason many reports have not been published is that the data are essentially negative, far from complete and, therefore, "uninteresting". Thus, if relatively little evidence of exposure was found in a population group, no further action relative to health effects was undertaken. It would appear essential to explore further the importance of these negative findings as it may be fundamental to the objectives of the interim study to determine what are the conditions in which exposures are likely to occur and the period that may elapse before a site begins to break down and become a hazard. This will require further contact with appropriate experts in that area.

2. State Correlation Studies

Drs. R. S. Levine and D. Chitwood of the Department of Epidemiology and Public Health at the University of Miami prepared an "Analysis of Existing Data Pertaining to Potential Health Problems from Hazardous Wastes in Florida" in April 1983, acting under Prof. J. Davies. This was a rapid survey of the state of Florida covering areas of waste disposal sites and associated health indices in relation to such deposits. In general, no health effects were found in most areas of the state that could be definitively correlated with the EPA designated sites, although in some sites, there was a trend toward lower indices of health. However, in other areas of the state where pollution was known to be considerable, no health effects were evident. In fact, these often were the most healthful areas relative to the rest of the state and the U.S. as a whole. No definitive conclusions could be drawn on the health effects of such sites. On the other hand, Davies believes that such an approach can demonstrate that, while a health hazard due to a waste disposal site cannot be completely excluded, such studies can provide most useful data as to the probable existence and extent of the health problem. Interestingly enough, this Florida study indicated that an immense amount of material relative to existing sites had already been collected and was present in the State Department of Health. This suggests that in planning and investigating exposures, it may be desirable to use these sources in addition to EPA or CDC. The Miami workers were convinced, therefore, that this approach did provide data of relevance and should be tried out in several other states. They estimated that each such study could be carried out at a cost of approximately $100,000 within a three month frame utilizing the resources of local universities and departments of health. However, these costs may be unrealistically low because for the Florida study personnel, facilities, computers, and related equipment and materials were already in place.

The Feasibility Study Group felt that this approach should be explored further during the review as a potential source of data in carrying out future follow-up investigations to place some limits on the probable extent of the hazard.

E. FEASIBILITY STUDY RESULTS

1. Availability of Data

There is extensive literature available regarding the toxicology of many chemicals, but specific information relating to the amount of each chemical in individual waste disposal sites, the degree of population exposures, and the potential hazards involved are very limited. Thus, unless specific assumptions are made, the problem theoretically can be so huge as to defy analysis. Accordingly, it is estimated that the study will need to confine its review to a limited universe of chemicals, to drawing specific inferences that might be pertinent to the overall problem, and to defining and justifying such assumptions on a scientific basis.

2. Availability of Expert Consultants and Staff

Conversations with a number of experts disclosed a broad interest in toxic waste disposal problems and a willingness to participate in a scientific approach to solving these problems. Recruitment should not present a major problem although some desirable candidates will be unavailable due to other commitments or serious conflicts of interest.

A preliminary list of potential consultants is presented in Appendix IV. This list will be expanded as necessary in the course of the study, especially after the first meeting of the Executive Scientific Panel.

Based on previous experience, it was decided that resolution of problems such as conflict of interest and willingness to testify should not be attempted during the feasibility study but, rather, should await authorization to proceed with the comprehensive review.

F. CONCLUSIONS

1. The Feasibility Study Group concluded that it is both desirable and feasible to carry out a comprehensive review to evaluate the substantial body of available data for the purpose of permitting an interim conclusion as to the nature of the health problems likely to be associated with waste disposal sites. A detailed presentation of the Feasibility Study Group recommendations on the organization and implementation of the comprehensive review study follows (Part II). Such a study would have the following objectives:

i) Preparation of a universe of chemicals most likely to occur in waste disposal sites and which are most likely to create health hazards.

ii) The levels and duration of exposure to these chemicals above which toxic effects might be expected would probably have to depend largely on inferences. Thus, the perennial question of the presence or absence of low exposure level effects would require emphasis and standard methodological approaches. However, a considerable amount of material is available regarding water and air pollution within the U.S. which can be used in determining such possibilities. It will not be possible, however, to utilize all the material available within EPA and other institutions.

iii) Identification of the acute and, to a more limited extent, the long-term toxic effects likely to result from human exposures to such chemicals, and whether they are real or imagined. This review would be based principally on published data in the peer review literature. It would make considerable use of secondary sources of material in view of the great difficulty of evaluating the vast amount of primary and raw material in EPA or state archives, unless the latter had been adequately analyzed.

iv) Identification of those clinical and laboratory investigations which are likely to be most useful in detecting health effects in exposed populations and the epidemiological techniques to be utilized in so doing.

v) Development of scientific criteria for identifying an immediate health hazard requiring urgent investigation or attention.

A comprehensive review is considered desirable and feasible because it would be comparatively inexpensive in terms of money and manpower. While it will add relatively nothing new to the universal literature, it will provide a unique source of evaluated data for scientists and the public regarding the potential health hazards and scientific issues associated with investigating waste disposal sites. It will provide a mechanism whereby the scientific community can be directly involved in investigating such issues and correcting the misconceptions that are widely held by the public. It will identify problems that are likely to occur frequently and thus involve academia in a search for scientific rational solutions. This may be pertinent to the further development of more intensive studies on an individual waste disposal site basis which may be necessary over the next decade.

Such a review might be more credible if it had multiple sponsors and if it were perceived as an interim measure to be followed up by additional studies.

2. The Feasibility Study Group concluded that as the proposed review is being completed, it would be desirable to follow up with studies aimed at compiling and analyzing raw data in the files of EPA, CDC, and various states agencies, carrying out in other states procedures modeled after the Florida study, analyzing the NHANES II data bank and results of the Health Interview Surveys conducted by NCHS to develop baseline health values, and testing the validity of the criteria developed in the review in a limited number of specific toxic waste disposal sites.

Such follow-up studies would require participation of various agencies and organizations and should also be funded from a number of sources probably including government, industry, and private philanthropy. The follow-up studies would also require wider and more extensive involvement of the academic community in developing correlation studies and utilizing data from all sources, as well as more active involvement and cooperation between the academic community and CDC, as well as other government laboratories. In addition, follow-up studies would enhance the probability of producing new data and providing better baselines for developing new scientific and technological criteria for hazardous waste disposal.

The only major disadvantage associated with follow-up studies is the potential cost and the necessity for developing considerable manpower and resources in academia. On the other hand, these costs are likely to be relatively modest in the context of the vast expenses incurred in cleaning up waste disposal sites if this is not done on a rational basis.

II. FEASIBILITY STUDY GROUP RECOMMENDATIONS ON THE ORGANIZATION AND IMPLEMENTATION OF THE COMPREHENSIVE REVIEW STUDY

A. OBJECTIVES:

1. To prepare a list of the universe of chemicals associated with waste disposal sites which could prove hazardous to man as the result of excessive exposure. Identify, if possible, the levels of human exposures likely to occur in association with waste disposal sites.

2. To define the probable health effects likely to occur in association with waste disposal sites and classify these according to their nature, real or imagined, acute, subacute, chronic, or carcinogenic. This is to be based on inferences from the available studies within the literature and inferences drawn from occupational, animal toxicology, and other sources.

3. To identify the clinical and laboratory end results that should be utilized in investigating such problems, and the epidemiological criteria necessary to establish causal relationships. Special emphasis should be given to the evaluation of negative data in relation to low level exposures.

4. To identify the scientific criteria for describing a specific or individual waste disposal site as representing an imminent health hazard.

5. To decide to what extent these conclusions (#1-4) provide a basis for identifying which follow-up studies may be carried out.

B. IDENTIFICATION OF PROBLEM CHEMICALS AND THEIR EVALUATION

Crucial to the development of the study will be the establishment of guideline criteria for identifying the universe of substances in waste sites and for establishing causal associations between exposures and potential human health effects.

1. Universe of Substances and Waste Sites

For logistical reasons, the list of hazardous chemicals must be defined and limited but, at the same time, hazards of perceived potential major significance must not be excluded. Identification of such chemicals and their exposure levels will be fundamental to the success of the study because, above all, it will be necessary to review the effects of probable human exposures likely to occur, or to have occurred, in relation to hazardous waste disposal sites as a basis for any epidemiological approach.

In attempting to analyze the universe of hazardous substances, the following lists of chemicals have been examined during the Feasibility Study:

i) Chemicals identified in the Superfund Priority Sites list of 418 hazardous waste disposal sites. The list of chemicals was prepared by Dr. Blaine McKusick of du Pont.

ii) A list of chemicals provided by CMA and designated by six major chemical-producing companies as those considered to be the 20 most likely to cause risks or public perception of risk. Each company had prepared its own list of 20 problem chemicals. A few chemicals received five to six votes, but most received two or three, indicating that the interests of the six companies are not necessarily similar. However, the final list of 20 represents a consensus of the six chemical producers.

iii) A list of chemicals prepared for the State of California and published in Chemical and Engineering News 1982.

iv) EPA list of relative carcinogenic potencies among 53 chemicals evaluated by the Carcinogen Assessment Group (dated April 5, 1983.)

v) Other sources of material relating to general or local environmental pollution, such as the Love Canal list and lists provided by several consultants.

It is interesting to note that there is a certain degree of overlap among the chemicals on each of the lists mentioned above.

2. Evaluation of Exposures

Estimation of exposures are recognized to vary widely and, until now, no satisfactory methods of calculating these have apparently been developed. Thus, each waste disposal site must be investigated according to a highly systematic plan. The possibility, however, that an appropriate group will be able to provide guidelines for the development of appropriate models as to those chemicals most likely to pose a danger according to the construction, location and nature of a deposit will receive careful attention. Some attempt must be made to distinguish between waste disposal sites that are well constructed and likely to exist for a prolonged period, and those where there is evidence already of immediate breakdown and release. It is recognized that while air and direct exposures may occur, they are unlikely to present an important hazard and that greatest concern relates to potential water contamination and, possibly indirectly, through diet resulting from contamination of local vegetation.

Most important, the overall significance of small waste disposal sites must be examined. For example, these were believed to be the greatest problem in Florida. It will be necessary, therefore, to explore all these aspects of the problem and to ensure that the Executive Panel and two Task Forces contain experts who have had experience in movement of substances from sites. Thus, evidence in this area will depend on i) material from sites which have already been examined, ii) literature reports in this and other countries, iii) the availability of further material of the type that was obtained in Florida from local resources, and iv) other sources. It may be possible that, after reorganization, EPA may be able to provide further information to the study group than they have been able to provide to the Feasibility Group. An additional important source of information will be studies on wild and domestic animals in the region of the sites.

Considering the importance of this aspect of the total problem, the material available from known sites should be examined and analyzed first. Then, the possibility of developing a background database of the overall exposures in the general population of the U.S. relative to suspected compounds should be explored. What is known regarding excess exposures in different situations will be established and specific areas in which appropriate exposure studies should be conducted will be defined. Conversely, there may be sufficient material already in the literature to permit evaluation of the general effects (or lack thereof) at higher levels of exposure, but this, again, almost certainly will require review by a group of experts.

3. Evaluation of Health Effects

In view of the many potential chemicals involved, no attempt will be made to generate primary data or evaluate raw data unless it is absolutely essential. Thus, data on health effects will be predominantly generated from previous reviews or assessments available within the general literature and other pertinent sources which are available to the public. Some of the various aspects that can be studied include:

i) Health Effects

Health effects will be inferred from what is known regarding potential levels of exposure and inferences drawn from high, intermediate, and low levels of exposure in other situations. Any attempt to relate low exposure level health effects to the multitude of chemicals which may occur, not only in relation to waste disposal, without considering the general background of the United States will be difficult and require careful evaluation but must be done. Thus, some attempt may be necessary to develop a profile on what is already known regarding exposures of the human population within the U.S., in terms of tissue deposits, etc., and their significance in disease patterns. Much of this material may, in fact, be available within the literature and could be the subject of further in-depth evaluation.

ii) Acute Effects

It will be important to define end points that can be examined with relative ease because they are not dependent on prolonged latent periods. Such effects should be closely related temporally to exposures if they are significant. Therefore, acute health effects may be susceptible to review whether as indicated by clinical, or clinico/pathological, or biochemical examination. Investigations of acute health-effect criteria should be standardized to a certain extent; criteria should be established to distinguish between organic and psychosomatic lesions. In this context, inferences drawn from occupational, accidental, and similar exposures at higher levels will provide pertinent data.

A list of end points will have to be examined to determine which are most likely to provide information of importance in evaluating health effects related to waste site exposures. Such end points would include:

a) blood studies;
b) urinary studies;
c) analysis of breast milk and other fluids and tissues;
d) evidence of respiratory, kidney, liver and other dysfunctions;
e) evidence of damage to infants, such as congenital malformations, cytogenic abnormalities, etc.

iii) Chronic Effects

Chronic effects, notably cancer, will require careful evaluation in light of the associated long latent period. In view of the small number of individuals liable to be exposed in relation to a particular waste disposal site and considering the known carcinogenic effects to be anticipated from suspected chemicals, it is quite possible that no conclusions can be reached regarding negative or positive findings in this context.

iv) Teratogenic and Mutagenic Effects

Following exposure to suspected chemicals, teratogenic and mutagenic effects may occur rapidly and are identifiable through congenital malformations, but many limitations apply to interpretation of the significance of several tests for mutagenesis. The assistance of CDC can be very valuable in this respect; toxicologists and epidemiologists with special expertise will be required to evaluate the findings.

v) Baseline Data

To compare the health status of individuals exposed to waste disposal sites deposits with that of the general population of the U.S., baseline data are required. Data from the ongoing Health Interview Survey and the second National Health and Nutrition Examination Survey (NHANES II) 1976-80 carried out by the National Center for Health Statistics would be used to identify a basic health profile of the U.S. population. With this information in hand, it may be possible to determine, on a broad basis, whether any indices can be attributed to proximity to waste disposal sites for an exposed population.

vi) Experience in Japan and Europe

Direct contact will be made with appropriate scientists and experts in Europe and Japan, who will participate as consultants to the task forces.

vii) Studies on Flora and Other Markers of Toxicity

A review of data from wild and domestic animals will be made and evaluated in terms of their implications for human disease.

In conclusion, the Feasibility Group agreed that a vast amount of material was available on a national and international scale from which significant and important inferences could be drawn as to the probable nature and extent of ill-health effects that could be anticipated as a result of previous waste disposal practices. In the past this material has been scattered and considered usually in terms of individual chemicals. However, a number of studies have been carried out on air pollution. In contrast, studies on chemically polluted water -- probably the most feared result of waste disposal -- are very poor and in terms of human disease are essentially negative, although the importance of water as a major source of human exposure has been shown for many of the suspected chemicals.

The possibilities of such a review are probably greater than originally believed and further analysis will be of great value in terms of the involvement and understanding of the academic research community.

4. Epidemiological Studies

In view of the small numbers of individuals likely to be involved in most waste disposal site exposures, epidemiological studies will be difficult to perform and will provide little information in most cases. Thus, the comprehensive review will of necessity have to contain an evaluation of the problems and limitations inherent in assessments of health effects related to individual sites. In addition, other methodologies for collecting material on a wider scale will have to be explored.

This particular aspect of the problem will be the subject of a specific task force. In this context, it will be desirable to further explore the experience of the Miami group in Florida as a base for calculating the overall extent of the problem.

5. Identification of Scientific Criteria for Classifying a Waste Disposal Site as an Imminent Health Hazard

This will arise out of the general discussion in the early portions of the survey and be addressed later by the Executive Panel which may, however, wish to redirect the question to one of the Task Forces.

C. ORGANIZATION OF THE STUDY APPROACH

The comprehensive review study will require an Executive Scientific Panel (ESP), two Task Forces (TF1 and TF2) and a full time scientific and support staff secretariat. The ESP will consist of 12 to 15 eminent academic scientists of international reputation. The ESP will be multidisciplinary in membership, including epidemiology, pathology, toxicology, biostatistics, oncology, occupational carcinogenicity, industrial hygiene, biochemistry, public health, toxic waste engineering, and other closely allied fields.

TF1 will address the universe of chemicals and aspects of exposure. TF2 will concentrate on human health effects. Both Task Forces will focus on additional issues and individual problems identified by the ESP and call upon appropriate experts from all sources to provide information and evaluation of the technical and scientific problems inherent in the investigation. A preliminary list of scientists identified as potential candidates for the ESP and Task Forces is attached (see Appendix IV).

In view of the nature of the subject and its unusual political and scientific ramifications, it is difficult to give an immediate outline of the procedures to be adopted by the ESP. These will depend, to a certain extent, on organization of a compatible panel of experts and their preliminary review of the problems and resources available. Accordingly, this necessitates a flexible approach to the initial scientific analysis and structure; the procedures outlined below should not be taken as definitive. It is a general principle that every effort will be made to take cognizance of and acquire any scientific information from any source pertinent to the overall problem, and these data will be freely available at the conclusion. Further, members of the committee will avoid obvious conflicts of interest. Lastly, adequate disciplinary representation and expertise should be included among the members to ensure wide exploration of all scientific viewpoints and to ensure credence in both the academic and public domain.

1. Management and Organizational Procedures (ESP)

While procedures must remain flexible, there are several aspects common to all evaluation studies. These include the following:

i) Preliminary conferences are held with key individuals to explore extant knowledge concerning hazardous waste disposal sites and to further identify experts with access to additional information.

ii) The purpose of these preliminary conferences is not only to identify additional individuals who may have pertinent knowledge or are engaged in research in related relevant topics, but also to define those scientific activities which may be most closely related to the particular type of evaluation. These discussions are used to generate a list of individuals as well as a list of key words to be used in computerized literature searching. Subsequent identification of key review articles and important information sources provide further expansion of the lists of potential consultants and pertinent key words.

iii) A definitive listing of pertinent parameters to be examined by the Executive Committee and Task Forces is prepared as appropriate based on an expanded review of current literature by the staff, current information on other aspects related to scientific evaluation, and consultation with key individuals concerning the scientists identified initially. In this case, care is taken to select all potentially pertinent information which will be balanced by the inclusion of work of investigators involved in related topic areas, or scientists holding divergent views concerning the subject.

iv) Typically, at this point a working paper will be prepared for the ESP by the professional scientific secretariat. This working paper describes the background of the issue and the extent of information available concerning the study topic. These papers, along with selected references, are sent to each participant prior to the ESP meeting at which the subject will be discussed.

v) Experience suggests that discussions with individual investigators or knowledgeable persons, as well as deliberations of the ESP at one to three-day meetings, coupled with a comprehensive literature review and analysis by the Task Forces, constitutes a reasonable basis for drafting an evaluation study. This draft report is prepared by the secretariat based on information obtained from the Task Forces, scientific literature, and from the discussions of the ESP. The draft report reflects documentation from the scientific literature and other valid information sources, as well as pertinent analysis and opinions of the Task Force members and/or ESP members. Where a controversial area exists, all aspects are discussed in considerable detail, and scientific references are given wherever possible. In instances where a consensus opinion of the ESP cannot be derived, an effort is made to include all divergent views.

vi) Drafts on individual topics are submitted to all members of the ESP as appropriate and, on occasion, to additional experts for their critical review and comment. Revision of the draft report is made by the staff based upon comments and emendations submitted by all participating consultants. The final draft may be resubmitted to the ESP for subsequent review and concurrence.

vii) The report of the ESP will be submitted to the UAREP Board for review and approval. This secondary review process ensures that the report will meet acceptable standards for scientific objectivity and fulfillment of study objectives.

viii) The Final Report will be published in toto and a synopsis will be published in the scientific literature.

2. Management and Organizational Procedures (TF)

The two Task Forces will be organized and will operate in an analogous manner, with certain exceptions. First, the ESP will play a major role in identifying qualified experts for membership on the two Task Forces. Second, membership on the Task Forces will be based primarily on scientific and disciplinary expertise with less emphasis on international reputation and possible conflict of interest.

Task Force reports, or series of reports, will be considered internal documents and will be exchanged between Task Forces, but delivered to the ESP. Task Forces are also considered *ad hoc* and may change in membership as determined by the ESP and secretariat. At least two ESP members will be on each Task Force.

3. Procedures to Assure the Adequate Identification as well as the Quality, Relevance, and Completeness of Data

Once specific problem areas have been identified relative to the universe of substances, exposure routes, health effects, etc., the ESP or a steering group of the ESP will explore i) information concerning a particular chemical, ii) toxic effects, and iii) identification of appropriate experts for consultation by the ESP for appointment to TF1 or TF2. Where appropriate, staff will visit investigators at their laboratories. The process of identifying and evaluating pertinent information will continue throughout the evaluation process, but it will probably not be possible to utilize sources of unanalyzed raw data.

Quality, relevance, and completeness of scientific data are a critical aspect of scientific evaluation. Experience dictates that the quality of data is related to the completeness of data and where these data may be found. In the final analysis, a reasoned judgment of the quality, relevance, and completeness of available data is a matter of a determination by the members of the ESP. The validity of the determination rests with the experience and reputation of the individuals who are selected to serve on TF1 and TF2, as well as with those on the ESP.

4. Elimination of Potential Bias and Conflicts of Interest Among Members of the ESP and TF1 and TF2

Evaluation studies of this type must use guidelines that include *ad hoc* self-terminating approaches, emphasis on the minimization of bias, and avoidance of quasi-permanent committees and appearance of vested interest. The following steps and techniques have proven successful in maximizing the objectivity of the input to evaluation studies:

i) Key individuals assist in the identification of potential ESP members and TF participants. These include members of the UAREP Board and other representatives of scientific societies who are able to identify knowledgeable scientists with expertise in particular study topics. The multiplicity of input provides ample opportunity to identify a wide diversity of potential participants. Every effort is directed towards balancing potential participants by the inclusion of consultants who may hold divergent views on a particular topic.

ii) In the course of developing an intensive literature database, it is possible to identify the specific contributions of potential participants to the study. Quality and objectivity of scholarly publications as well as reputation are evaluated by the project's staff.

iii) The final lists of ESP and TF members are reviewed by the UAREP Board. This review procedure is included to eliminate bias and potentially avoid vested interests following which an effort is made to informally contact each individual a second time to invite him or her to participate in the study.

iv) ESP and TF members are asked to complete a conflict of interest form. This form provides information on the extent of financial obligations and involvement in the public and private sectors which may bear upon the particular topic under evaluation. Procedures have been developed for maintaining the confidentiality of these statements; and an internal review is conducted by members of the UAREP Board. Specifically, the Executive Officer of UAREP, the President of UAREP, and the Business Manager of UAREP reviews each statement of conflict of interest for evidence of potential conflict. Where no conflict exists, forms are signed, sealed, and held until completion of the project. Where potential conflict is noted, the opinion of legal counsel is sought.

If and when conflict of interest is apparent, the scientist is informed, while a decision is made as to whether or not he or she should be given further consideration. If in the affirmative, the individual must agree prior to the evaluation study that, if at any point this conflict of interest is raised by the Executive Officer of UAREP or the project director, he or she would state for the meeting record non-participation in the deliberations or decision-making process of the Task Force or ESP.

5. Procedures to Resolve Conflicting Opinions

During Task Force meetings or during the deliberations of the ESP, the respective chairmen will ensure informal, harmonious discussions in which all participants are allowed to present data and comment on information presented. The chairmen must be selected for their experience in this context. Draft reports, revisions, and preparation of the final reports by the secretariat are written to reflect opinions of participating scientists and other knowledgeable individuals, documentation from the scientific literature, and pertinent analysis. Because the staff rapporteurs are recognized as professional scientists by all, the credibility of the document is further enhanced. Every effort is made to present and document divergent views, but to give a consensus of scientific opinion where appropriate. If a consensus is not appropriate, then divergent views are documented as completely and objectively as possible.

In cases where minority opinions are necessary, they are written by the secretariat based upon discussions with those dissenting members. Members with dissenting opinions are asked to indicate their identity and their concurrence with the material that has been independently prepared by the staff. This aspect is further strengthened by the subsequent secondary review and approval of the report by the UAREP Board.

6. Proposed Study Plan

The overall study comprises specific steps as follows:

Month 1: Identify Executive Scientific Panel and Chairperson

The ESP will be comprised primarily of members of the academic community with expertise in epidemiology, toxicology, biostatistics, experimental pathology, oncology, occupational carcinogenesis, industrial hygiene, biochemistry, public health, and toxic waste engineering. Emphasis will be placed upon obtaining the cooperation of individuals from the academic community who, insofar as possible, have no conflicts of interest or biases. These will be individuals recognized for their expertise in the particular relevant disciplines. There will be approximately 12-15 individuals on the Executive Panel. Budgetary considerations are based on a 15-member Executive Panel and five two-day meetings (or some combination of 10 days of meeting time).

One of the first individuals to be identified would be the Chairperson of the ESP. This individual should be available 100% of the time during the study period and should be recognized for his or her general expertise in review and evaluation of public health and scientific issues, rather than only for expertise in one particular discipline. Because most knowledgeable scientists in industry and government have ipso facto a conflict of interest, an effort will be made to recruit academic members of the scientific and medical community as ESP members.

Nominations will be accepted from all sources, including public announcements and specific requests to various communities concerned with toxic waste sites, such as industry, government, environmentalists, academic scientists, scientific societies, professional organizations, and other sources (e.g., the National Academy of Sciences, Carnegie Mellon Foundation, Ford Foundation, etc.). The primary criteria for membership on the Panel will be scientific expertise and freedom from conflict of interest or bias.

A list of potential invitees as members of the ESP will be prepared by the staff and forwarded to the UAREP Board for concurrence and selection. Individuals selected will be asked to serve on the Panel and to complete the conflict of interest information.

Months 2-3: Initial ESP Meeting

A two-day meeting for organizational purposes will be conducted. Prior to this Panel meeting, all members will have been invited and informed of the conditions of the appointment and the scope of the study. In addition, they will receive a working paper describing the project and an agenda for the meeting. At the meeting, the consultants will be asked to:

i) review and concur with the study plan or make modifications as they deem desirable,

ii) identify the need for Task Force activities and to provide a scope of work for those Task Forces, and

iii) identify individuals to serve on the several Task Forces.

It should be noted that at least two members of the ESP will serve on each Task Force as chairperson and vice chairperson (or recorder or rapporteur). Members of the Task Forces will be selected on the basis of scientific expertise and may include representatives of industry, government, academia, and other communities as required by the scope of Task Force activities.

Months 3-4: Appointment of the Task Forces

At the present time, we believe that only two Task Forces will be necessary.

Universe of Chemicals and Human Exposures Task Force (Task Force #1): The first activity of Task Force #1 (TF1) will be to evaluate the several lists of chemicals that describe the universe of substances found in hazardous waste disposal sites, and to finalize a finite list of substances likely to prove problematic in terms of hazardous waste disposal sites. It should be noted that at this stage, TF1 may require additional outside expertise from both government and industry as consultants.

The second activity of TF1 will be to examine information available on the substances in the finalized universe of chemicals, and to identify levels of probable exposure, methods or route of exposure, and duration of exposures that may prove to have some impact on human or animal health, and on the environment. TF1 will have seven to nine members and may utilize an additional five consultants. TF1 will meet four times for two days each.

Health Effects Task Force (Task Force #2): TF2 will consist of nine to eleven persons and will provide guidance in the evaluation of probable health effects that are to be anticipated in relation to hazardous waste disposal exposures of those chemicals within the universe of substances that have been determined to be potential exposure problems.

TF2 will consider acute and chronic health effects that are likely end points of exposure, information available from previous epidemiological studies and from occupational medicine, scientific evaluation of toxicity in laboratory animal studies, and data from other waste disposal sites. TF2 may wish to organize itself into subgroups. Additionally, it is expected that TF2 will require a greater input from staff in terms of literature and data analysis. TF2 may also require more time and meetings than TF1. TF2 will meet five times for two days each.

Months 3-6: Meetings of Task Forces

It is anticipated that TF1 must meet first and that TF2's progress will be dependent upon the output from TF1. The budget reflects a total of six meetings for the two Task Forces; however, it is anticipated that TF1 would meet twice and TF #2 would meet four times. Both TF1 and TF2 would draft reports, with the reports of TF1 being submitted to TF2.

The final output from TF1 would be a finalized list of problem chemicals, and information concerning environmental, animal, and human exposures that may prove to have demonstrable health effects. Emphasis would be upon human health effects. Clearly, this background information would be essential for TF2 in categorizing the likely end points or criteria for human health hazards and in identifying sources of data where the Health Effects Task Force would look for information relative to the toxic effects and potential health effects of the substances.

Months 4-7: ESP Meeting

Following the completion of the reviews of TF1 and TF2 and the completion of their draft reports, the ESP would utilize this information to create a draft of the review study report.

The conclusions of the review study draft report would point out what should be looked for in any waste disposal situation based upon extant scientific knowledge concerning the potential hazard to man, rather than upon political expedience, public complaints, or other reasons. A strong case would be developed for a reasoned judgment on scientific evidence of potential risk. The Tentative Final Report of the ESP would be published and made publicly available for comment.

Month 8: Open Public Forum

After publication of the Tentative Final Report of the ESP, an opportunity would be provided for all members of the public and the several communities of interested parties to come and address, either in writing or orally, the ESP at an Open Public Forum. The topic for discussion would be a review of and comments on the Draft Report in terms of garnering opinion as to the approach being presented and identification of any additional needs or deficiencies. This open public forum would be recorded and a transcript of the entire forum would be made available at cost to all participants. The ESP would be seated in front of the room and would listen to and accept all comments. The purpose of the Open Public Forum is information gathering and there would be no emphasis on entering into a dialogue or presenting the information in an adversarial manner. The organization and conduct of the open forum would be disseminated as widely as possible. In making arrangements for this, those individuals who wish to testify orally would be asked to provide written statements. Those not wishing to testify orally would be given deadlines for submission of written material. By negotiation, all oral testimony would be confined to a finite period of time.

Months 9-10: Review of Information Received

The ESP would meet a third time to discuss all information presented at the Open Public Forum and to revise as necessary the Tentative Final Draft of the Report. Based upon the decisions reached by the ESP in a review of the included comments, an attempt would be made to reach a consensus by the panel and a Final Report would be prepared. This Final Report would be provided to the sponsor of the study and would be disseminated widely and made available at cost to the public.

D. ESTIMATION OF COSTS

In analyzing the necessity of input from Task Forces and ESP, and to provide adequate time for review of the information collected by Task Forces, for preparation of Task Force reports, and for subsequent analysis of this information by the ESP and preparation of the Final ESP Report, as well as a desire to have informed public input, it seems logistically necessary to provide for a minimum of a 10-month period for the Comprehensive Review Study. The budget has been developed on the basis of 10 months. The budget also contains additional information concerning the number of meeting days for members of the Task Forces and panel.

COMPREHENSIVE REVIEW STUDY

10-Month Budget

Personnel[a]	Annual Salary Basis	10-Month Budget	
Project Director[b]	$ 62,400		
Staff Scientists[c] (2)	101,400		
Associate Scientists[d] (2)	58,800		
Administrative Assistant	24,953		
Technical Data Analyst	19,394		
Technical Literature Specialist	19,394		
Senior Secretary	17,958		
Clerk	12,226		
Subtotal	$316,525	$263,771	
Fringe Benefits & Payroll Taxes @ 28%		73,856	
Subtotal Personnel			$337,627
Consultants[e]			
a) Executive Scientific Panel 15 members @ $500/day 5 meetings - 2 days each		75,000	
b) Task Force #1 9 members @ $450/day 4 meetings - 2 days each		32,400	
c) Task Force #2 11 members @ $450/day 5 meetings - 2 days each		49,500	
Subtotal			$156,900

Travel and Per diem[f]		
Travel @ $600; per diem @ $100		
a) Executive Scientific Panel		
5x15x600	45,000	
5x2x15x100	15,000	
b) Task Force #1		
4x9x600	21,600	
4x2x9x100	7,200	
c) Task Force #2		
5x11x600	33,000	
5x11x2x100	11,000	
Subtotal		$132,800
Other Direct Costs[g]		
Telephone @ $600/month	5,400	
Postage and mailing (includes overnight mail) @ $975/month	8,775	
Expendible supplies	3,600	
Computerized literature retrieval @ $240/month	2,160	
Word processing @ $320/month	2,880	
Report preparation (2TF + 1 tentative + 1 final) 4 reports @ 150 pp. each @ $27/p., 550 cc.	16,200	
Duplication & photocopying, est. 216,000 @.07/p.	15,120	
Open public forum:[h]		
Hall rental for 2 days	1,100	
Transcript preparation	2,350	
Conference center expenses[i]		
1 meeting - 4 days	2,400	
Miscellaneous	975	
Subtotal		$ 60,960
TOTAL DIRECT COSTS		$688,287
INDIRECT COSTS[j] (39.6% of salaries, wages and consultant fees = $494,527)		$195,833
TOTAL COSTS		$884,120
FIXED FEE @ 10%		88,412
TOTAL CONTRACT		$972,532

BUDGET NOTES

a) All personnel are 100% FTE for 10-month period. It is essential that scientific staff be full time; some salary lapse with support staff may occur during months #1 and #10, but wage figures are not adjusted.

b) The Project Director would be a senior scientist (M.D. and/or Ph.D.) whose international reputation and scientific credentials are impeccable. His or her expertise would be in toxicology, pathology, and epidemiology.

c) Staff Scientists (2) would be mid-level M.D. or Ph.D. investigators with at least 10 years of professional experience and relevant education. Each would be initially assigned to a Task Force as staff coordinator. Staff scientists would be responsible to the Project Director and would play a major role in data collection and analysis, and preparation of reports.

d) Two M.S. or B.S. level associate scientists with at least 3-5 years experience in data analysis and review, as well as skills in scientific writing.

e) Consultant fees are based on the mid-point of daily fees paid by government contracts and private industry. Fees are necessary to attract and hold the most highly qualified persons as well as the necessity to require that they contribute substantially to the ESP and Task Force reports. Fees for ESP members are based on $500/day and those for Task Force members, $450/day. The budget assumes expert consultants from industry or government (as appropriate) would receive no compensation from UAREP.

f) $600 per trip is based on the average travel cost experience of UAREP and LSRO for 1982-83. Per diem is based on the daily rates for the Marriott Hotel, Bethesda, rate plus $24.00 per day.

g) Estimates derived from current projects and previous contractual activities of analogous scope and size.

h) Based on $550 per day (local Bethesda hotel rate).

i) One ESP meeting may be a four-day meeting outside the Bethesda area.

j) UAREP Indirect Cost rate at 39.6% estimated for 1983-84. Rate is based on total direct personnel salaries, wages, and consultant fees.

APPENDIX I

List of Persons and Organizations Consulted in the Feasibility Study

Patricia A. Buffler, Ph.D.
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Associate Professor of Epidemiology
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Joe W. Grisham, M.D., Ph.D.
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Paul Lundy, M.S.
Toxic Substances Program
National Audubon Society
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Vice President for Science
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New York, New York 10022

Russell Peterson, Ph.D.
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New York, New York 10022

Carl Shy, M.D.
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University of North Carolina
School of Public Health
Chapel Hill, North Carolina 27514

Ellen K. Silbergeld, Ph.D.
Environmental Defense Fund
1525 18th Street, N.W.
Washington, D.C. 20036

APPENDIX II

Literature and Reference Material
Reviewed by UAREP/LSRO in the
Feasibility Study
(March - May 1983)

100 - 199

Memoranda (originating outside FASEB)

File No.	From	Subject	Date
100	McKusick, B.	Potentially hazardous chemicals at disposal sites	12/1/82
101	McKusick, B.	Superfund priority sites	1/7/83
102	McKusick, B.	Corn Report (file #500)	1/4/83
103	Foege, W.	Transmits CDC report on long-term followups of populations exposed to toxic hazards, #506	6/21/82
104	Tozzi, J., OMB	Toxic Torts -- evaluating merits of alternative legislative proposals (sent by T. O'Leary, CMA)	4/18/83

CMA Literature File

200 - 299

Letters (Incoming)

File no.	From	To	Subject	Date
200.0	Umland, C., Exxon	Karrh, B., du Pont	CDC's Superfund Responsibilities	12/22/82
200.1	Umland, C.	Endicott, K.	Transmits draft of on-going study (file #550(a),(b))	3/28/83
201	Karrh, B., du Pont	Zoll, D., CMA	Transmits summary of mortality study of chemical workers (file #620)	1/21/83
202	Thomas, L., Sloan-Ket.	Gov. Carey, New York	Transmits Love Canal report (file #505)	10/8/80
203.0	McKusick, B.	Higginson, J.	Transmits study summaries (file #204, 441(b), and 609)	3/16/83
203.1	Mckusick, B.	Higginson, J.	Transmits Contra Costa study (file #509)	3/16/83
203.2	McKusick, B.	Higginson, J.	Transmits summary of Contra Costa study (file #510)	2/28/83
203.3	McKusick, B.	Higginson, J.	Transmits study summaries (file #611-618)	3/16/83
203.4	McKusick, B.	Higginson, J.	Transmits Triana summary (#1017) & article on CA waste rules (#812)	4/13/83
204	Senger, J., Monsanto	Karrh, B., du Pont	Contains study summaries. (Enclosure to letter #203.0)	1/3/83
205	Parker, H. Union Carb.	McKusick, B du Pont	Transmits 2 studies on petrochemical workers (#440 & 441)	3/15/83
206	O'Leary, T., CMA	Higginson, J.	Transmits lists of banned chemicals from CFR (file #1013 & 1014), from EPA (file #1015), and CO_2 study (file #437)	4/19/83
207	Duchie, W., Shell Oil	Higginson, J.	Transmits 7 documents on McColl Site, California, file #519-525	4/13/83

Letters (Cont'd.)

File no.	From	To	Subject	Date
208	Gaffey, W.R., Monsanto Co.	Altman, P.	Transmits reprints (#445-447) and paper (#529) on 2,4,5 T production accident in W. Va.	4/20/83
209	Cole, P., U. of Alabama	Higginson, J.	Response to Dr. Endicott's letter of 4/5/83 on Feasibility Study	4/20/83
210	Gehring, P.J., Dow Chemical	Endicott, K.	Same as for #209	4/21/83
211	O'Leary, T.F., CMA	Endicott, K.	Transmits an OMB memo on toxic torts, file #104	4/22/83
212	Kissell, R.G., du Pont	Altman, P.	Transmits 2 letters from Kissel to CMA Superfund Task Group, file #213 and 214	4/28/83
213	Kissell, R.G., du Pont	CMA Superfund Task Group	EPA Expenditure Status	3/15/83
214	Kissell, R.G., du Pont	CMA Superfund Task Group	Analysis of Proposed National Priority List	2/16/83
215	Dourson, M.L., EPA	Altman, P.	Transmits EPA Document Listing, file #1018	4/29/83
216	Silbergeld, E., Env. Def. Fund	Fisher, K.	Response to UAREP letter of 4/5/83 on Feasibility Study	5/3/83
217	Levine, R.S., U. of Miami	Higginson, J.	Transmits report on hazardous wastes in Florida, and a summary, file #532(a) & (b)	4/28/83
218	Damstra, T., NIEHS	Fisher, K.	Transmits 3 lists of chemicals found in Superfund sites, file #1037 - 1038	5/11/83
219	Buffler, P.A., U. of Texas	Higginson, J.	Comments on Feasibility Study strategy	5/16/83
220	Paulson; Lundy, Audubon Society	Endicott, K.	Response to UAREP letter of 4/5/83 on Feasibility Study	5/17/83
221	Heath, C.W. CDC, Atlanta	Higginson, J.	Inquiried if further input was required of him for waste study	6/5/83

CMA Literature File

300 - 399

Books

File no.	Author	Title
300	Epstein, et al.	Hazardous Waste in America
* --	Brown, M.	Laying Waste: The Poisoning of America by Toxic Chemicals
301	Bridges; et al., editors	Banbury Report 13: Indicators of Genotoxic Exposure

* Not numbered, to prevent damage, because book on loan from Geraldine Cox, CMA.

CMA Literature File

400 - 499

Journal Articles

File no.	Author(s)	Journal	Title
400	Sun, M.	Science	Dioxin's Uncertain Legacy
401	Anonymous	Inside EPA	SAB Criticizes RCRA Degree of Hazard Plan As Not Useful in Reg Decisions
402	Zuesse, E.	Reason	Love Canal--The Truth Seeps Out
403	Pell, S.	Transactions of N.Y. Acad. Sci.	Identification of Risk Factors in Employed Populations
404	Karrh; Pell	Annals of N.Y. Acad. of Sci.	Brain cancer in the du Pont Company
405	Hoar; Pell	J. Occup. Med.	A Retrospective Cohort Study of Mortality and Cancer Incidence Among Chemists
406	O'Berg, M.	J. Occup. Med.	Epidemiologic Study of Workers Exposed to Acrylonitrile
407	Pell, S.	J. Occup. Med.	Mortality of Workers Exposed to Chloroprene
408	Bonnett; Pell	J. Occup. Med.	Du Pont's Health Surveillance Systems
409	Pell; O'Berg; Karrh	J. Occup. Med.	Cancer Epidemiologic Surveillance in the du Pont Company
410	Pell, S.	J. Occup. Med.	Epidemiological Requirements for Medical-Environmental Data Management
411	Dow Chemical	The Point Is...	No Double Standard of Risk
412	Ott; Holder; Langner	J. Occup. Med.	Determinants of Mortality in an Industrial Population (have 2 copies)
413	Townsend; et al.	Am. J. Epidemiol.	Survey of Reproductive Events of Wives of Employees Exposed to Chlorinated Dioxins
414	Kramer; Mutchler	Amer. Indus. Hyg. Assoc. J.	The Correlation of Clinical and Environmental Measurements for Workers Exposed to Vinyl Chloride
415	Ott; Holder; Gordon	Archives of Environ. Health	Respiratory Cancer and Occupational Exposure to Arsenicals
416	Ott; Langner; Holder	Archives of Enriron. Health	Vinyl Chloride Exposure in a Controlled Industrial Environment
417	Ott; et al.	J. Occup. Med.	A Health Study of Employees Exposed to Vinylidene Chloride
418	Roush; Ott	Amer. Indus. Hyg. Assoc. J.	A Study of Benzene Exposure Versus Urinary Phenol Levels

Journal Articles (Cont'd.)

File no.	Author(s)	Journal	Title
419	Ott; Gordon; Schneider	J. Occup. Med.	Mortality Among Employees Chronically Exposed to Caustic Dust
420	Ott; et al.	Archives of Environ. Health	Mortality Among Individuals Occupationally Exposed to Benzene
421	Townsend; Ott; Fishbeck	J. Occup. Med.	Health Exam Findings Among Individuals Occupationally Exposed to Benzene
422	Kramer; et al.	Archives of Environ. Health	Health of Workers Exposed to 1,1,1,-trichloroethane: A Matched-Pair Study
423	Ott; Holder; Olson	J. Occup. Med.	A Mortality Analysis of Employees Engaged in the Manufacture of 2,4,5-trichlorophenoxyacetic acid
424	Ott; Scharnweber; Langner	British Journal Indus. Medicine	Mortality Experience of 161 Employees Exposed to Ethylene Dibromide in Two Production Units
425	Venable; et al.	J. Occup. Med.	A Fertility Study of Male Employees Engaged in the Manufacture of Glycerine
426	Ott; Kolesar; Scharnweber; Schneider; Venable	J. Occup. Med.	A Mortality Survey of Employees Engaged in the Development or Manufacture of Styrene-Based Products
427	Cook; Townsend; Ott; Silverstein	J. Occup. Med.	Mortality Experience of Employees Exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCCD)
428	Bond; et al.	Annals of N.Y. Acad. of Sci.	Case-Control Study of Brain Tumor Deaths Among Employees at a Chemical Manufacturing Plant
429	Egnatz; et al.	J. Occup. Med.	DBCP and Testicular Effects in Chemical Workers An Epidemiological Survey in Midland, Michigan
430	Van Raalte; Grasso	Regulatory Toxicology and Pharmacology	Hematological, Myelotoxic, Clastogenic, Carcinogenic, and Leukemogenic Effects of Benzene
431	Kraybill, H.	Preventive Medicine	Evaluation of Public Health Aspects of Carcinogenic/Mutagenic Biorefractories in Drinking Water
432	Kraybill, H.	Jour. of Amer. Water Works Association	Carcinogenesis of Synthetic Organic Chemicals in Drinking Water
433	Yager; Hines Spear	Science	Exposure to Ethylene Oxide at Work Increases Sister Chromatid Exchanges in Human Peripheral Lymphocytes
434	Janerich; et al.	Science	Cancer Incidence in the Love Canal Area

Journal Articles (Cont'd.)

File no.	Author(s)	Journal	Title
435	Glotfelty; Taylor; Zoller	Science	Atmospheric Dispersion of Vapors: Are Molecular properties unimportant?
436	Hanis, N.; et al.	J. Occup. Med.	Cancer Mortality in Oil Refinery Workers
437	Riley; Bromberger-Barnea	Arch. Environ. Health	Monitoring Exposure of Brewery Workers to CO_2: A Study of Cellar Workers and Controls
438	Hanis, N.; et al.	J. Occup. Med.	Epidemiologic Study of Refinery and Chemical Plant Workers
439	Lynch, J.; et al.	J. Occup. Med.	An Association of Upper Respiratory Cancer With Exposure to Diethyl Sulfate
440 a&b	Austin; Schnatter	J. Occup. Med.	A Cohort Mortality Study of Petrochemical Workers (pre-publ. copy and reprint)
441	Austin; Schnatter	J. Occup. Med.	A Case-Control Study of Chemical Exposures and Brain Tumors in Petrochemical Workers
442	Kraybill, H.	(article in book): Water Chlorination ... Vol 1	Origin, Classification and Distribution of Chemicals in Drinking Water with an Assessment of their Carcinogenic Potential
443	Helmes, C; et al.	J. Environ. Sci. Health	Evaluation and Classification of the Potential Carcinogenicity of Organic Air Pollutants
444	Hohenemser; Kates; Slovic	Science	The Nature of Technological Hazard
445	Marsh, G.M.	Brit. Journal Indus. Medicine	Proportional mortality pattersn among chemical plant workers exposed to formaldehyde.
446	Marsh, G.M.	J. Occup. Med.	Mortality among workers from a plastic-producing plant
447	Zack, J.A.; Suskind, R.R.	J. Occup. Med.	The mortality experience of workers exposed to TCDD in a TCP process accident
448	Hanis, N.; et al.	Annals of N.Y. Acad. of Science	Brain Cancer: Issues and Dilemmas
449	Heath, C.W.	Environ. Health Perspectives	Field Epidemiologic Studies of Populations Exposed to Waste Dumps
450	Kreiss, K.; et al.	J. American Med. Assoc.	Cross-Sectional Study of a Community with Exceptional Exposure to DDT
451	Kreiss, K.; et al.	J. American Med. Assoc.	Association of Blood Pressure and Polychlorinated Biphenyl Levels
452	Clark, C.S., et al.	Arch. Environ. Health	An Environmental Health Survey of Drinking Water . . . in Hardeman County, Tennessee

CMA Literature File

500 - 549

Reports

File no.	Prepared by	Item
500	Corn, M., et al., Johns Hopkins Univ.	Corn Report
501	MITRE Corporation	A Ranking System for Hazardous Substances Releases
502	PEDCo Environmental	Analysis of Eckhardt Committee Survey, for CMA
503	National Research Council	Risk Assessment in the Federal Government
504	Office of Technology Assessment, U.S. Congress	Technologies and Management Strategies for Hazardous Waste Control
505	Thomas, L., Chairman, Governor's Panel, N.Y.	Love Canal Report
506	Heath, C., et al., CDC	Use of Long-term Registries for Health Followup of Populations Exposed to Toxic Hazards
507	Holmes, T.	Ph.D. Thesis Draft: A Mortality Study of Butyl-chlorobutyl rubber workers.
508	EPA	Ambient Water Quality Criteria for DDT.
509	Austin, D., California Dept. of Health Services	Contra Costa County Study on Cancer Incidence
510	Same as #509	Summary of Contra Costa County Study
511	Eckhardt Subcommittee, U.S.H.R.	Waste Disposal Site Survey Report
512	EPA	Hazardous Substances: National Contingency Plan
513	EPA	Proposed Superfund Priorities List of Sites
514(a)&(b)	JRB Associates	Review of Literature on Herbicides, Vol. 1 & 2 (Prepared for the Veterans Administration)
515(a)-(c)	California Department of Health Services	Carcinogen Identification Policy
516(1)-(10)	EPA	Notification of Hazardous Waste Sites, Regions I-1 10 volumes.
517	Scientific Document Service, Seveso, Italy	CEFIC Health Protection Committee Documents. (Studies on accidental release of TCDD in Seveso
518	National Research Council	Long-range Environmental Outlook

Reports (Cont'd.)

File no.	Prepared by	Item
519	Shell Oil ?	A Chronological Summary of the McColl Site, Fullerton, CA (1942-1983).
520	California Dept. of Health Svcs. (DOHS)	Press release summarizing preliminary results of the McColl Site Technical Study, Phase I
521	Radian Corporation for: Calif. DOHS	McColl Phase 2: Environmental Setting of the McColl Site, Fullerton, CA
522	Radian Corporation for: Calif. DOHS	McColl Phase 2: Physical and Chemical Characterization and Distribution of the Waste at McColl
523	Radian Corporation for: Calif. DOHS	McColl Phase 2: Summary of the Nature and Extent of the Problem at the McColl Site
524	Radian Corporation for: Calif. DOHS	McColl Phase 2: Modeling of the Air Emissions from the McColl Site Investigation.
525	Calif. DOHS	Executive Summary of the McColl Site Health Survey
526	NCI; Mitre Corp.; et al. For: NIH	Biorefractories in water: carcinogens, mutagens promoters
527	Kraybill, H.F.	Animal Models and Systems for Risk Evaluation of Low Level Carcinogenic Contaminants in Water
528	Kraybill, H.F.	Outline on Contaminants, Type, Origin and Toxicity of Water Contaminants
529	Zack, J.A.; Gaffey, W. (Monsanto)	A mortality study of workers employed at the Monsanto Company Plant in Nitro, West Virginia
530	CDC	A System for Prevention, Assessment, and Control of Exposures and Health Effects from Hazardous Sites
531	NCI; SRI International	Organic Air Pollutants: Carcinogens, Mutagens, Promoters (52-page report)
532(a)&(b)	Levine, R.; Chitwood, D.; University of Miami; for: State of Florida	Analysis of Existing Data Pertaining to Potential Public Health Problems from Hazardous Wastes in Florida, and a 3-page summary
533	Fred C. Hart Associates for: EPA	Assessment of Hazardous Waste Mismanagement: Damage Case Histories
534	CDC (Heath, et al)	A Study of Cytogenetic Patterns in Persons Living Near the Love Canal
535	Task Force on Environ. Cancer and Heart and Lung Disease	Summary of the Workshop on Exposure to Environment Agents, Their Metabolism, and Mechanisms of Toxicity: Research Needs
536	National Research Council	Management of Hazardous Industrial Wastes: Research and Development Needs

Reports (Cont'd.)

File no.	Prepared by	Item
537	Office of Technology Assessment, U.S. Congress	Role of Genetic Testing in the Prevention of Occupational Disease

CMA Literature File

550 - 599

Studies Underway

File no.	Prepared by	Item
550(a)&(b)	Hanis, N., et al. (Exxon)	A Retrospective Mortality Study of Workers in Three Major U.S. Refineries and chemical plants 1970-1977. Parts 1 & 2

CMA Literature File

600 - 699

Abstracts & Summaries

File no.	Prepared by	Item
600	Ott, M., et al, (Dow)	Health Evaluation of Employees Occupationally Exposed to Methylene Chloride (Abstracts of general study and its subdivisions)
601	Bond, G., et al, (Dow)	A Case-Control Study of Brain Tumor Mortality at a Texas Chemical Plant
602	Ott, M., Langner (Dow)	A Mortality Survey of Men Engaged in the Manufacture of Organic Dyes
603	Cook, R., et al, (Dow)	A Cross-Sectional Study of Ethylene Glycol Monomethyl Ether Process Employees
604	Bond, G., et al, (Dow)	Medical and Morbidity Surveillance Findings Among Dow, Michigan Division Employees Potentially Exposed to TCDD.
605	Reeve, G., et al.	An Investigation of Brain Tumors Among Chemical Plant Employees Using a Sample-Based Cohort Method
606	Carlo, G., et al (Dow)	Assessment of Fertility Potential Among Male Employees
607	Carlo, G.	Clean Air Legislation and Its Scientific Initiatives
608	Currier, M., et al, (Dow)	A Cross-Sectional Study of Employees with potential Workplace Exposure to Ethylene Oxide.
609	Braun, D., Carlysle, S. (Union Carbide)	An Epidemiology Study of Silanes Workers
610	FASEB & NLM	Bibliography and Abstracts on Chemical Waste Disposal
611	Enterline, P., (Shell)	Shell Study: ECH
612	Whorton, D., et al, (Shell)	Shell Study: ECH Fertility Survey
613	Enterline, P. (Shell)	Shell Study: IPA
614	Enterline, P. (Shell)	Shell Study: Lubes Dewax
615	Whorton, D. et al, (Shell)	Shell Study: PTBBA Fertility Survey
616	Rushton, et al, (Am. Petr. Inst.)	Oil Distribution Centers -- Great Britain

Abstracts & Summaries (Cont'd.)

File no.	Prepared by	Item
617	Tabershaw-Cooper (Am. Petr. Inst.)	American Petroleum Institute Study: Refinery Study - U.S.A.
618	Am. Petr. Inst.	API: Epidemiology Studies (in progress)
619	Wen, C, et al.	Paper on Gulf Refinery Study
620	du Pont	General Mortality and Mortality Due to Cancer Among Workers in the Chemical Industry
621	Lipshultz, L.; et al.	Dibromochloropropane and Its Effect on Testicular Function in Man from Journal of Urology 124(4)
622	Theriault; Goulet	A Mortality Study of Oil Refinery Workers from Journal of Occupational Medicine 21(5)
623	Hanis; Stavraky; Fowler	Cancer Mortality in Oil Refinery Workers from Journal of Occupational Medicine 21(3)
624	Austin; Schnatter	A Case-Control Study of Chemical Exposures and Brain Tumors in Petrochemical Workers, from Journal of Occup. Med. In press.
625	Hanis, N.; et al.	Study of Exxon Refinery and Chemical Plant from Journal of Occupational Medicine 24(3)
626	Rushton; Alderson	Study of British Oil Refineries from British Journal of Industrial Medicine 38
627	FASEB & NLM	Bibliography and abstracts on The Effects of Environmental Chemicals on the Immune System
628	FASEB & NLM	Bibliography and abstracts on Health Effects of Environmental Chemicals on the Adult Human Reproductive System
629	NCI	Asbestos and Health: An Annotated Bibliography of Public and Professional Education Materials

CMA Literature File

700 - 749

News Releases & Bulletins

File no.	Date	Item	Subject
700	2/7/83	CMA News Release	CMA's Joining in Suit Against DHHS and EPA
701	3/17/83	CMA News Release	Contract Award to UAREP for Feasibility Study
702	5/17/83	HHS News	CDC study of cytogenetic patterns in residents of Love Canal area (see file #534)

750 - 799

TSCA Bulletins (Toxic Substances Control Act) -- EPA Publication

File no.	Date
750	12/81
751	8/82
752	10/82
753	3/83

CMA Literature File

800 - 899

News Articles

File no.	Date	Source	Title
800	1/23/83	New York Times	Dioxin's Peril to Humans: Proof Is Elusive
801	2/13/83	Washington Post	Superfund "Fast Track" Mired in Procedure
802	3/18/83	Frederick Post	Dioxin Exposure Tests Uncover Only Skin Rashes
803	3/11/83	Science	House Reviews EPA's Record on Pesticides
804	1/22/83	S.F. Examiner	Smog Absolved in East Bay Cancer
805	1/22/83	Oakland Tribune	Higher Cancer Rate in Contra Costa Blamed on Smoking
806	unknown	New York Times	Panel Discounts Two Studies on Love Canal Problem
807	3/29/83	Washington Post	Efforts of Terrified Neighbors Brought Belated End to Dump Site
808	3/29/83	Washington Post	Shuffling EPA Inadequate, Environmentalists Say
809	3/25/83	Science	EPA's Troubles Reach a Crescendo
810	3/25/83	Science	Congress Investigates Malfeasance at EPA
811	unknown	Spotlight	A Framework for Environmental Health Research: the Harvard-EPA Cooperative Agreement
812	11/29/82	Chem. & Eng. News	California Setting New Hazardous Waste Rules
813	11/29/82	Chem. & Eng. News	Former EPA Official Addresses Waste Issues
814	4/23/83	Science News	Second Superfund Buyout Proposed: A Missouri Dioxin Site
815	4/23/83	Science News	Times Beach: The Long Road to Recovery
816	1983	Human Ecology and Health	Perceptions of Environmental Contamination and their Implications for Health
817	6/13/80	Science	Love Canal: False Alarm Caused by Botched Study
818	6/13/80	Science	Love Canal Residents Under Stress
819	May 1983	The Chemist	Surviving a State of Siege: Love Canal Revisited
820	5/28/83	Washington Post	Chemical Health Panel Ordered: To Be New HHS Agency
821	6/3/83	Science	Waste Management

CMA Literature File

900 - 999

Requests for Proposals

File no.	Issuer	Date	Title
900	EPA	3/11/83	Technical Handbook on Decontamination of Buildings, Structures, and Construction Equipment at Superfund Sites
901	NIEHS	3/10/83	Toxicology Research and Testing Program Health and Safety Support

CMA Literature File

1000 - 1099

Miscellaneous

File no.	General Description	Title or Subject	Prepared by
1000	Symposium Proceedings, Rockefeller Univ.	Assessment of Health Effects at Chemical Disposal Sites	Lowrance, W
1001	Agenda & Registration Form	Fourth Annual Symposium on Environmental Epidemiology, Univ. of Pittsburgh, May '83	
1002	2 Tables	Data Development Work Group: Tables on Availability of Data	CMA
1003	Summary	Technical and Epidemiological Studies of the McColl Site (Fullerton, Cal.) 3/14/83	CMA ?
1004	12 Tables	Average Annual Age-Specific Death Rates in the du Pont Company Among Active and Pensioned Employees, 1957-71, and Among United States White Persons, 1964	du Pont
1005	Table	Mortality by Occupation and Industry Among Men 20-64 Years of Age: United States, 1950.	Guralnick DHEW
1006	Draft for CMA Bd. of Dir. Meeting Book	Public Compensation (for harm by hazardous substances)	O'Leary, T CMA
1007	Legal Brief with 3 Attachments	Comments of FMC Corporation on EPA's Proposed National Priorities List. Attachments: Analysis of NPA's Hazard Ranking System -- Potential Impacts ... at FMC/NOD Site -- Annotated National Priorities List	FMC Corp. Arthur D. Little, In
1008	Letter with Attached Application	Cooperative Agreement for Project on Impact of Toxic Substances Regulatory Alternatives On Industry, dated March 21, 1983	EPA
1009	Table	Top 50 Chemicals: Production was lower in 1981 for the Second Year Straight. From: Chemical & Engin. News, June 14, 1982	
1010	Table	Production of Chemicals and Allied Products. From: Survey of Current Business, Jan. 1983	
1011	Tables and List of Chem. Manufacturers	U.S. Production of Selected Synthetic Organic Chemicals	U.S. Int'l Trade Com.
1012	1-page Chemical Profiles	Profiles of About 150 Chemicals From: Chemical Marketing Reporter, issues dated between 1980 and 1982	

Miscellaneous (Cont'd.)

File no.	General Description	Title or Subject	Prepared by
1013	CFR, Title 21 parts 189-193	Food and Drugs - banned substances	Ofc. of Fed. Reg.
1014	CFR, Title 16 parts 1303-1500.17	Commercial Practices - banned substances	Ofc. of Fed. Reg.
1015	EPA Booklet	Suspended and Cancelled Pesticides	EPA
1016	Public Law 96-510	Comprehensive Environmental Response, Compensation, and Liability Act of 1980	US Congress
1017	1-page summary	Triana, Alabama (situation of waste site)	B. McKusick
1018	EPA Document List	Lists documents on various chemicals, prepared by Environ. Criteria and Assessment Office	EPA
1019	Table and Graph	Relative Carcinogenic Potencies Among 53 Chemicals Evaluated by the Carcinogen Assessment Group	EPA
1020	Section from a chapter of draft	Quantitative Estimation. From Chap. 13, Health Assessment Document for Acrylonitrile. By: Carcinogen Assessment Group	EPA
1021	Cover material for Scientists' Forum	Scientists' Forum on an Assessment of the Scientific Knowledge Base Related to Victim Compansation, Jan. 27-29, '82, Wasn. DC	National Science Foundation
1022	Paper presented at above Forum	Identification ... of Health Hazards in Population: Epidemiological Assessment	R. Hoover
1023	"	Tracing the Movement of Pollutants ...	V. Freed
1024	"	Common law tort remedies for pollutant-induced disease	J.W. Wade
1025	"	Clinical Environ. & Occupational Medicine	Carnow; Conibear
1026	"	History of Victim Compensation Science	Silcox
1027	"	Model of compensation for pollutant-induced disease: the case of 2,4,5,T	Jasanoff
1028	"	Model ...: case of nitrilotriacetic acid	S. Albert
1029	"	Model of compensation for DES	L. Ritts
1030	"	Model of compensation for asbestos	S. Soble
1031	Technical Information	Tox Box^{TM}, a hazardous waste container, made by Lab Safety Supply, Wisconsin	Lab Safety Supply
1032	Equipment Catalogue	Safety Equipment for Science & Industry, Lab Safety Supply, Wisconsin	"

Miscellaneous (Cont'd.)

File no.	General Description	Title or Subject	Prepared by
1033	Equipment Catalogue	Air Sampling Catalog: Equipment and Supplies, Gilian Instrument Corp., Wayne, New Jersey	Gilian Corp.
1034 (a) & (b)	Toxline computer printout	Titles of articles on hazardous waste disposal	Toxline
1035 (a) & (b)	Enviroline computer printout	Titles of articles on hazardous waste disposal	Envirolin
1036	Report to Congress	Toxic Substances Control Act (TSCA) Report to Congress for Fiscal Year 1982	EPA
1037	List of chemicals	110 Chemicals found in Superfund waste sites, with specific sites listed for each chemical	CDC
1038	List of chemicals	15 chemicals occurring most frequently in Superfund waste sites	NIEHS
1039	List of chemicals	15 chemicals nominated by EPA for toxicologial testing	NPA
1040	Letter and attachment	Sun Information Services Company's computerized SunHealth Occupational Health Information Management System, dated 4/27/83	Sun Informatic Svcs. Co
1041 a,b,c	Letter and two brochures	Diamond Shamrock Corp.'s Computerized Occupational Health & Environmental Surveillance System (COHESS), dated 4/29/83	Diamond Shamrock
1042	Scientific equipment catalogue	Industrial Hygiene News: 1983/84 Catalog and Buyer's Guide	Ind. Hyg News
1043	News Letter	Occupational Health & Safety Letter, 4/8/83 (article on UAREP's study for CMA on page 4)	Environew Inc.
1044	Ad literature and subscription form	Chemical Regulation Reporter, a weekly publ. of The Bureau of National Affairs, Inc.	Bureau of Nat. Af.
1045	Comments from CAST (Council for Agricul. Science & Technology)	The Missouri Dioxin Controversy: Scientific Overview	CAST

Key Reference Material

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APPENDIX IV

Partial List of Senior Scientific Experts Proposed for Executive Scientific Panel and Task Forces

Susan C. Austin, Sc.D.
Union Carbide Corporation

* Norman E. Breslow, Ph.D.
University of Washington

* Patricia A. Buffler, Ph.D.
University of Texas

* John Cairns, M.D.
Harvard University

Glyn G. Caldwell, M.D.
Centers for Disease Control

* Philip Cole, M.D.
University of Alabama

* Morton Corn, Ph.D.
Johns Hopkins University

Terri Damstra, Ph.D.
National Toxicology Program

* John Davies, M.D., M.P.H.
University of Miami

* W. Richard Doll, M.D., D.Sc.
Imperial Cancer Research Unit
Green College, Oxford University

Philip E. Enterline, Ph.D.
University of Pittsburgh

Joseph F. Fraumeni, Jr., M.D.
National Cancer Institute

William R. Gaffey, Ph.D.
Monsanto Company

Murray B. Gardner, M.D.
University of Southern California

Perry J. Gehring, Ph.D.
Dow Chemical, U.S.A.

James E. Gibson, Ph.D.
Chemical Industry Institute
of Toxicology

Joe W. Grisham, M.D., Ph.D.
University of North Carolina

Clark Heath, M.D.
Centers for Disease Control

Brian E. Henderson, M.D.
University of Southern California

Howard H. Hiatt, M.D.
Harvard University

Barbara Hulka, M.D., M.P.H.
University of North Carolina

* Donald Kennedy, Ph.D.
President, Stanford University

Herman F. Kraybill, Ph.D.
National Cancer Institute

Joshua Lederberg, Ph.D.
President, Rockefeller University

Brian MacMahon, M.D., Ph.D.
Harvard University

Tom S. Miya, Ph.D.
University of North Carolina

John A. Moore, D.V.M.
National Toxicology Program

Robert A. Neal, Ph.D.
President, Chemical Industry
Institute of Toxicology

* Frank J. Rauscher, Jr., Ph.D.
American Cancer Society

* Herbert S. Rosenkranz, Ph.D.
Case Western Research University

Carl Shy, M.D.
University of North Carolina

Ellen K. Silbergeld, Ph.D.
Environmental Defense Fund

* Edward A. Smuckler, M.D., Ph.D.
University of California

Benjamin F. Trump., M.D.
University of Maryland

Arthur C. Upton, M.D.
New York University

* Possible candidate for Executive Panel

1982
ANNUAL REPORT
AND SPECIAL REPORTS

THE GREAT SEAL OF THE STATE OF NORTH CAROLINA
MAY 20 1775
ESSE QUAM VIDERI

GOVERNOR'S WASTE MANAGEMENT BOARD

STATE OF NORTH CAROLINA
OFFICE OF THE GOVERNOR
RALEIGH 27611

JAMES B. HUNT, JR.
GOVERNOR

GOVERNOR'S WASTE MANAGEMENT BOARD
CLAUD "BUCK" O'SHIELDS, JR.
CHAIRMAN

January 17, 1983

Honorable Joe Hackney
Legislative Building
Raleigh, NC 27611

Dear Representative Hackney:

The Governor's Waste Management Board is pleased to present to you a copy of our 1982 Annual Report. The Board makes several timely recommendations within the report which will require legislative action regarding the management of hazardous and low-level radioactive waste in North Carolina.

I urge you to take the necessary time to read and study this report and to give us your comments on the Board's activities and recommendations. If you desire additional information on the recommendations contained in the Annual Report, please notify our office of your needs.

I look forward to working with you on this most important program. Please call our office if we can be of assistance.

Sincerely,

Buck O'Shields

Buck O'Shields, Chairman
Governor's Waste Management Board

kcg

325 North Salisbury St. Albemarle Bldg. Room 513 Raleigh, N. C. 27611 919/733-9020

STATE OF NORTH CAROLINA
OFFICE OF THE GOVERNOR
RALEIGH 27611

JAMES B. HUNT, JR
GOVERNOR

GOVERNOR'S WASTE MANAGEMENT BOARD
CLAUD "BUCK" O'SHIELDS, JR
CHAIRMAN

Governor James B. Hunt, Jr.
State of North Carolina
State Capitol Building
Raleigh, North Carolina 27611

Dear Governor Hunt:

On behalf of the Waste Management Board, I am pleased to submit to you the first Annual Report describing the activities of the Board during 1982 and setting forth recommendations for administrative and regulatory action.

The members of the Board have worked diligently to fulfill the responsibilities of their appointments as set forth in the Waste Management Act of 1981, forever mindful of the intent of the Governor's Task Force on Waste Management in recommending the establishment of the Board. Foremost in the mind of every board member has been the charge made by the Governor's Task Force to "develop a system of waste management which is flexible enough to adjust to emergencies and changing technology, but is firm in its determination to prevent the generation of waste that cannot be disposed of safely" (Report of the Governor's Task Force on Waste Management, February, 1981). Protection of the health and environment of our citizens now and in the indeterminate future has been the Board's overriding concern.

Considering the enormity of our task, I feel that the Board has made great strides during 1982 toward the development of a comprehensive waste management program for North Carolina. Great progress has been made in assessing the volume, distribution, location, and physical and chemical characteristics of hazardous waste and low-level radioactive waste generated or disposed of in the state. The Board has evaluated the possible need to organize State agencies more efficiently to improve overall performance of waste management functions, and a Special Report on this issue is included in this report.

The Waste Management Board has also made great progress in legal issues pertaining to North Carolina's waste management program. Rules of Procedure were developed and filed for the Board to use when reviewing a petition to preempt a local ordinance that prohibits the establishment or operation of a hazardous waste facility or a low-level radioactive waste facility. Rules of Procedure were also developed and filed for the privilege license tax review process. By far the most difficult legal issue which the Board debated was the desirability of establishing by

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statute a standard of strict liability for persons involved in storage, transportation or disposal of hazardous or low-level radioactive waste in North Carolina. The Board's recommendations on this issue are contained in Chapter II of this report.

Last, but certainly not least, the Waste Management Board succeeded in involving many more North Carolinians in the discussion and development of a better waste management program for North Carolina. A large number of guests attended and contributed their input at the Waste Management Board and committee meetings. The Board received and answered many requests for information from both industries and private citizens. The Board developed educational materials, conducted and co-sponsored several educational activities, and has begun planning for several more educational activities. Finally, the Board has instituted and widely publicized the annual program for the Governor's Award of Excellence in Waste Management.

The Waste Management Board is proud of the progress made thus far, and we hope that you will give careful consideration to the following recommendations explained in detail within this report. The Waste Management Board recommends that:

1. a Waste Management Coordinating Committee be established under the Governor's Waste Management Board to enhance and strengthen the present coordination that exists between State agencies and departments;
2. strict liability be applied by statute to any generator, transporter, treater or storer of hazardous waste in control of the waste at the time it causes injury to a claimant;
3. the Solid and Hazardous Waste Management Branch, Division of Health Services, Department of Human Resources, be given the authority by the General Assembly to require annual reports from all generators, treaters, storers and disposers of 1000 kilograms or more of hazardous waste in North Carolina;
4. sufficient funds be appropriated by the General Assembly for a reasonable regulatory program to protect the health, safety and environment of the people of North Carolina;
5. funding be appropriated by the General Assembly to monitor and clean up old waste sites.

The Board believes that implementation of the above recommendations can have a profound positive impact on North Carolina's ability to develop a safe effective waste management program which will better protect the health of the citizens and the environment.

The members of the Waste Management Board wish to thank you for this opportunity to serve you and the citizens of North Carolina. We look forward to continuing our work in 1983.

Sincerely,

Buck O'Shields

Claud "Buck" O'Shields, Chairman
Governor's Waste Management Board

CO/kcg

MEMBERSHIP
GOVERNOR'S WASTE MANAGEMENT BOARD

Board members serve staggered terms and represent a broad range of interests from all geographical areas of the State.

GOVERNOR'S APPOINTMENTS:

General Public With An Interest In The Environment

Claud "Buck" O'Shields (Chairman)
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Fayetteville, NC 28305

Higher Education, Research or Technology

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Captain William H. Briner
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Albert L. Canipe
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LT. GOVERNOR'S APPOINTMENT:

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SPEAKER OF THE HOUSE APPOINTMENT:

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STATE AGENCIES:

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Department of Natural Resources and Community Development

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Natural Resources and Community Development
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Department of Crime Control and Public Safety

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CHAPTER I: ACTIVITIES DURING 1982 CATEGORIZED BY MANDATED FUNCTIONS AND POWERS

PART A: DESCRIPTION OF ACTIVITIES

ONE The Board shall periodically evaluate and assess the volume, distribution, location, and physical and chemical characteristics of hazardous waste and low-level radioactive waste generated or disposed of in the State.

An assessment of the volume, location, distribution, physical and chemical characteristics of hazardous and low-level radioactive waste generated or disposed of in the State is necessary in order to determine the extent of the problem North Carolina has in managing its industrial waste.

1. Hazardous Waste

On January 20, 1982 the Solid and Hazardous Waste Management Branch called for annual reports from the approximately 1,400 industries in North Carolina listed as major generators. The Federal Resource Conservation and Recovery Act had mandated that annual reports be filed with the states by major waste generators (those that produce more than 1,000 kilograms of hazardous waste per month) by March 1, 1982.

On March 15, 1982 the Department of Human Resources' Solid and Hazardous Waste Management Branch was notified that the Environmental Protection Agency had postponed its requirement that major generators of hazardous waste file annual reports.

By the time the State received notification of the postponing of the filing of the annual report, approximately 900 industries had already filed their annual report. Officials of the Solid and Hazardous Waste Management Branch anticipated, as did the Governor's Waste Management Board, that most of the remaining 500 industries would comply with requests for annual reports. As indicated in the annual report, 95% responded to the generator survey and 99% of the treaters, storers and disposers complied with the requests. This is important to note as once the Environmental Protection Agency postponed its requirement for the annual report, North Carolina, by law, could not require the annual report. G.S. 130-166.21D provides that no North Carolina regulation can be more restrictive than the federal regulations. It is felt by the Solid and Hazardous Waste Management Branch and the Governor's Waste Management Board that the State should continue to require an annual report for some period of time in order to track its program of managing hazardous and low-level radioactive waste.

The 1981 Annual Report as compiled by Mr. O. W. Strickland, Head of the Solid and Hazardous Waste Management Branch and his staff, now gives North Carolina its most complete inventory of hazardous waste generated within its borders. Copies of the report are available from the Solid and Hazardous Waste Management Branch and the Governor's Waste Management Board.

The Technical Committee of the Governor's Waste Management Board, in conjunction with the Technical Advisory Committee, is doing a more in-depth analysis of the materials furnished to the State in the annual reports filed by the generators, treaters, storers and disposers (for more detail, see page 16).
This analysis is scheduled for completion by February 18, 1983.

2. Superfund Sites

In February, 1982 the State was notified by the Environmental Protection Agency that 167 sites in 49 counties might contain hazardous waste that had been stored, treated or disposed of in the past (documentation available upon request). The locations of the sites range from the mountains to the coast, and include industrial plants, military facilities, municipal and county landfills and abandoned garbage dumps. Many of the sites have been around for a long time, some since 1933.

These sites were identified in a list compiled under the Comprehensive Environmental Response, Compensation and Liability Act of 1980, commonly known as the "Superfund" Act. All sites were inspected in a cooperative effort with the Solid and Hazardous Waste Management Branch in the Department of Human Resources, the Division of Environmental Management in the Department of Natural Resources and Community Development, and the Environmental Protection Agency. After preliminary inspections of all sites, it was determined that only 9 sites pose a threat to human health thereby requiring monitoring and possibly some remedial action. Two sites have since been cleaned up, one with superfund money and one by the owner. The State continues to monitor the remaining 7 sites.

The Waste Management Board's recommendation concerning funding for monitoring and cleaning up old waste sites is in Chapter II, page 60.

3. Low-Level Radioactive Waste

At the request of the Waste Management Board, the Radiation Protection Section of the Division of Facility Services, Department of Human Resources collected data to determine the quantities and types of low-level radioactive waste disposed of by North Carolina industries, research institutions and medical facilities for the period June 1, 1981 - July 30, 1982.

According to the survey, 23 facilities reported some type of low-level radioactive waste disposal. Almost 57% of the volume of low-level radioactive waste generated in North Carolina during the reporting period was disposed of by transfer to the Chem Nuclear Disposal Site in Barnwell, South Carolina. The remaining 43% is disposed of by transfer to the disposal site in Richland, Washington. In terms of the quantity of radioactive material in Curies, over 97.5% is disposed of by transfer to the South Carolina disposal site; only 2.5% goes to the site located in Washington State.

The report cites possible problems for low-level radioactive waste disposal in North Carolina due to severe volume limitations and restrictions which preclude acceptance of certain types of waste at the South Carolina Disposal Site, as well as mounting efforts to prevent access to the Washington State Site.

The report was presented to the Board in December, 1982. This data will greatly assist the State in determining facility needs for safely managing low-level radioactive waste generated within its borders.

TWO The Board shall periodically review the State's comprehensive waste management system and make recommendations to the Governor, cognizant State agencies, and the General Assembly on ways to improve waste management; reduce the amount of waste generated; maximize resource recovery, reuse, and conservation; and minimize the amount of hazardous waste and low-level radioactive waste which must be disposed of.

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1. Waste Management Technology

The Waste Management Board, especially members of the technical committees (Technical Committee, Technical Advisory Committee on Hazardous Waste and the Technical Advisory Committee on Low-Level Radioactive Waste), has received information, held discussions, and in some cases made recommendations regarding the following areas of waste management technology:

a. Ms. Betsy Dorn, Director of the Piedmont Waste Exchange at the University of North Carolina at Charlotte, presented a report on the role the Waste Exchange can play in educating and promoting the recycling and exchanging of waste materials in North Carolina. As a service of the Waste Information Research and Exchange Program of UNCC, information, research, and education services are utilized to assist Carolina industries with developing waste management strategies that are safe and economical.

b. In February, Mr. Dave Roche, Duke Power Company, gave a slide presentation on fossil fuel combustion and PCB burn. Mr. Roche explained how complete combustion of the fuel depended on the temperature, residence time, and the amount of oxygen present. He also explained the 100 MW pulverized coalfired boiler used at Duke Power's Riverbend Steam Station where waste PCB-contaminated transformer mineral oil was incinerated. A copy of Mr. Roche's presentation is attached in Appendix 2:1.

c. Also in February, Dr. Pete Andrews of the Institute of Environmental Studies explained what the Institute had been doing specifically in the areas of hazardous waste.

d. The Technical Committee heard a presentation by Jim Gilpin with Associated Technologies, Inc. summarizing the current processes available for Waste Volume Reduction (available upon request).

2. State Government Organization, Policy, Legislation and Funding

The Waste Management Board has received information, held discussions and made recommendations concerning the need for changes in State Government in regard to organization, policy, legislation or funding.

a. The Waste Management Board has discussed whether or not to recommend to the General Assembly the removal of G.S. 130-166.21D from the environmental legislation passed in North Carolina since 1973. The Board asked the state departments to look at EPA changes and what actions may be necessary for the General Assembly to take in 1983.

b. The Waste Management Board discussed an issue paper concerning sufficient funds to allow for a reasonable regulatory program to protect the health, safety, and the environment of North Carolinians and North Carolina's natural resources.

c. The Waste Management Board has discussed the state's need for funding to be able to monitor and clean up old waste sites that are determined to be a threat to North Carolina and its people.

d. The Waste Management Board has discussed the question of what to do in emergency or spill situations, i.e., where do we dispose of these, usually toxic, wastes?

e. The Waste Management Board has been asked to consider the current loop-hole in state law (G.S. 10F.0029) that allows municipal wastewater treatment facilities to become hazardous waste facilities, as industry processing wastes are discharged into sewer systems.

f. The Waste Management Board discussed the need for the General Assembly to authorize state agencies to require annual reports from generators, treaters, storers, and disposers.

g. The Waste Management Board has discussed the question of what wastes or waste residuals that North Carolina should not allow to be placed in burial facilities.

THREE The Board shall study and make recommendations on policy issues including but not limited to liability and financial responsibilities within the waste management area. On or before January 1, 1983, the Board shall prepare and present to the Governor and General Assembly a report concerning the desirability of establishing by statute a standard of strict liability for persons involved in storage, transporation, treatment, or disposal of hazardous or low-level radioactive waste in North Carolina.

The Waste Management Board assigned this function to its Legal Committee for study and recommendations. Several key policy questions were discussed in determining whether to support strict liability, and, if so, in what form. These policy questions included:

* For what risks or injuries will strict liability be imposed?
* How will the establishment of a standard of strict liability affect the cost of insurance for waste management facilities?
* Who or what types of activities should be subject to strict liability? Generators? Disposers? Transporters? Successors in interest? Others?
* Is strict liability to be handled through the courts or is a separate system to be established for this purpose?

The Legal Committee presented a report of its findings concerning the desirability of establishing by statute a standard of strict liability and other liability issues to the Waste Management Board on December 1, 1982. The Board voted to approve the twelve recommendations of the Legal Committee for presentation to the Governor and the General Assembly. These recommendations are presented in the "Special Report and Recommendations on Liability Issues," beginning on page 44.

FOUR The Board shall promote research and development and disseminate information on state-of-the-art means of handling and disposing of hazardous waste and low-level radioactive waste. The Board is authorized to establish a waste information exchange for the State.

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1. Hazardous Substances in North Carolina Public Schools

The Waste Management Board's Public Information Committee has met several times with the Department of Public Instruction to discuss plans for addressing the problems of storage, use and disposal of hazardous and toxic substances found in North Carolina schools.

The public schools of North Carolina have expressed a strong need for help with initiating a management system for the hazardous substances used in four major curriculum areas: a) Science Departments; b) Art Departments; c) Industrial Arts; and d) Vocational Education.

With the technical assistance of the Waste Management Board, the Department of Public Instruction proposes to identify chemical substances which should be removed from the schools, provide for the pickup and disposal of these chemicals, and provide guidelines and teacher training in the safe use of chemicals left in the schools (documentation available upon request).

2. Pollution Prevention Pays Symposium

On May 26-27, 1982, the Governor's Waste Management Board co-sponsored an educational symposium entitled "Pollution Prevention Pays" to promote the innovative new ways industries are finding to minimize or prevent the production of pollutants, while at the same time reducing production costs. Approximately 225 representatives from industry, waste management firms, government and other organizations were in attendance.

In addition, the Waste Management Board aided in the distribution of the "Pollution Prevention Pays" supplement printed in the Charlotte News and Observer. Proceedings of the symposium are now available in book form.

3. Research Projects

The Waste Management Board cooperated with the Governor's Board of Science and Technology to select the following research projects on hazardous or low-level radioactive waste.

Siting Waste Management Facilities, R. Andrews and F. Runge, University of North Carolina - Chapel Hill.

Effectiveness of "Clay Liners", J. Pierce, Duke University.

Improving Pollution Monitoring Methods, M. Kimberly, North Carolina State University.

Evaluating Potential Disposal Sites, C. W. Welby and I. J. Won, North Carolina State University.

Mercury in Wastewater Treatment Systems, J. S. Wu and H. Hilger, University of North Carolina - Charlotte.

Evaluating the Effect of Hazardous Waste Disposal Sites Upon Groundwater Aquifers, R. Janardhanam, University of North Carolina - Charlotte.

Local Emergency Response to Hazardous Materials Accidents, E. A. Holland, M. H. Ford, and E. W. Blaha, Triangle J Council of Governments.

The findings of these studies will be presented to the Waste Management Board upon their completion during 1983.

4. Workshops

The Chairman and one member of the Waste Management Board have attended a two-day workshop given by the Industrial Extension Service of North Carolina State University with support from the Solid and Hazardous Waste Branch of the Department of Human Resources entitled "Hazardous Waste Management Under RCRA". This workshop, given nine times to over 270 individuals, was found to be excellent by the attending Waste Management Board members. These and other workshops and supporting manuals developed by Industrial Extension Service have been applauded by the Governor's Waste Management Board and staff, and the Governor's Waste Management Board has encouraged participation in these educational programs. Further information regarding these programs is available in the office of the Waste Management Board.

5. Waste Information Exchange

Although the Waste Management Board is authorized to establish a waste information exchange for the state, the Board has not taken such an action during 1982. Rather, the Board has expressed the view that waste information exchange can be conducted more effectively and more appropriately by the three waste exchange programs which already operate in North Carolina. These are:

Piedmont Waste Exchange
Urban Institute
Department of Urban & Environmental Engineering
UNC at Charlotte
UNCC Station, NC 28223
(704) 597-2307
Contact: Betsy Dorn

Atlantic Coast Exchange
PES, Inc.
1905 Chapel Hill Road
Durham, NC 27707
(919) 493-3536
Contact: Doug Kent

National Waste Exchange
Route 12, Box 255A
Winston-Salem, NC 27107
(919) 769-2668
Contact: Carl Wilkins

FIVE The Board shall promote public education and public involvement in the decision-making process for the siting and permitting of proposed waste management facilities.

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1. Public Input

Public attendance and input was encouraged at meetings of the full Waste Management Board and at all committee meetings. At all meetings, guests from the general public were given ample opportunity to voice their concerns and ask questions.

To maximize citizen participation in the activities of the Waste Management Board, the following measures have been taken:

a. Members were added to the Public Information/Participation Committee from the general public;

b. Meetings were held outside of Raleigh in Tarboro and Winston-Salem; and

c. Some meetings were held in the evening.

Lists of citizens who attended the Board and committee meetings are on file in the office of the Waste Management Board.

2. Public Participation

The Waste Management Board discussed the concerns of several citizen groups in regard to public participation in the siting of hazardous waste facilities. In response to these concerns, the Waste Management Board took the following actions:

a. The Waste Management Board mailed a letter to the North Carolina Association of County Commissioners and the North Carolina League of Municipalities recommending that their constituent members establish Citizen Involvement Committees on hazardous waste in order to develop a body of local knowledge, to educate the general public on hazardous waste issues, and to prepare to participate in local hazardous waste siting issues, if required (Appendix B-1). Counties which have established citizens' committees on waste management include:

Anson
Bladen
Buncombe
Cumberland
Guilford
Lee
New Hanover

b. The Waste Management Board drafted, and adopted as working policy, Proposed Procedures for Public Participation in North Carolina's Hazardous Waste Permitting Process (available upon request from the office of the Waste Management Board). These procedures outline the type of interaction between local citizens, the permitting authority, the applicant and the Waste Management Board which the Board considers desirable. The procedures emphasize that:

(1) An informational package should be established to prepare and educate localities concerning disposal/treatment of hazardous wastes.

(2) Negotiation between local citizens and the facility developer is strongly encouraged.

(3) Local concerns and agreements with the facility developer must be given merit by the reviewing agencies.

(4) Local agreements must be incorporated into the review before a tentative decision is made by the permitting authority.

It should be noted that there is no mechanism within existing permit procedures to require adherence to these proposed procedures, and additional consideration needs to be given to methods for implementing these procedures.

c. The Public Information/Participation Committee has begun development of a public information package including:

(1) A Waste Management Board brochure (complete);

(2) A Citizen's Handbook and Decision-Making Guide on the hazardous waste facility siting process (in progress);

(3) A Citizen's Guide to negotiation in siting hazardous waste management facilities (in progress); and

(4) A North Carolina Waste Management Directory listing sources of information and technical assistance concerning hazardous and low-level radioactive wastes (in progress).

The projected completion date for these materials is February 28, 1983.

3. Public Hearings

A public hearing was held on August 26, 1982, on the proposed rules involving the procedures for the preemption process and the privilege license tax review. Comments from the public were heard at this time and written comments were accepted for two weeks following the hearing. These comments were discussed and incorporated into the procedures before final approval.

SIX The Board shall periodically evaluate and assess the type and number of hazardous waste facilities, hazardous waste landfill facilities, low-level radioactive waste facilities and low-level radioactive waste landfill facilities in existence, under construction or planned in the State and multi-State region and promote the development of additional facilities particularly retrievable above-ground storage facilities if existing or planned facilities are deemed inadequate or unavailable.

An accurate evaluation of the type and number of waste management facilities could not be done until up-to-date information was reported by the generators, treaters, storers and disposers of hazardous waste in North Carolina. As soon as these data were received from the Solid and Hazardous Waste Management Branch of the Division of Health Services, the Waste Management Board developed a Request for Proposal for an "Analysis of North Carolina's Industrial Waste." The scope of the project is outlined below.

a. Review data obtained by the Office of Solid and Hazardous Waste in its 1981 report on generators, treaters, storers and disposers of hazardous waste in North Carolina and any other pertinent data available from the state.

b. Identify hazardous waste generated in North Carolina that can be recycled or otherwise recovered through known techniques either by the generator, other industries through waste exchange or by special resource recovery facilities; estimated volumes; and recommendations as to the number and types of facilities or processes that may be needed in North Carolina to manage hazardous waste.

c. Identify the commercially operated hazardous waste treatment facilities operating in North Carolina and the types of waste that can be handled by these facilities.

d. Identify the best available technology known for the destruction or rendering to a less hazardous state the waste generated in North Carolina and subsequent disposal procedure of the residues.

e. Identify and support with conclusions of known facts those wastes treated or non-treated that should be prohibited from land disposal due to the risks they present to human health and the environment.

f. Identify and support with conclusions of known facts the most appropriate method for disposal of those wastes identified in item "e" above.

g. Identify methods of stabilizing wastes or waste residue for those wastes which have been specifically identified as safe for land disposal.

The Board has awarded a contract to Tracor Jitco, Inc. (Research Triangle Park), which will complete the analysis by February 18, 1983.

When the Board receives this report, the recommendations concerning the number and types of facilities needed will be modified as necessary. A Supplemental Report of the Board's findings and recommendations will then be submitted to the Governor and the General Assembly.

SEVEN The Board shall prepare and file jointly with the Governor and the General Assembly an annual report describing the Board's activities and setting forth its recommendations for administrative or regulatory action required to improve the State's comprehensive waste management system or remedy noted defects in the system. A special report shall be filed in January of 1983 which shall include an evaluation on the possible need to organize State agencies more efficiently to improve overall performance of waste management functions. The report should give consideration to the advantages and disadvantages of consolidating or centralizing administration of programs that are now in separate agencies.

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1. Annual Report

This document constitutes the first Annual Report of the Governor's Waste Management Board. The report was drafted by staff members, approved by the Board on January 5, 1983, and presented to the Governor and the General Assembly in mid-January.

The Annual Report of the Governor's Waste Management Board describes the activities of the Board since its creation on June 26, 1981, when the North Carolina Waste Management Act was ratified. The Annual Report also sets forth recommendations for administrative or regulatory action required to improve the State's comprehensive waste management system or remedy noted defects in the system. These recommendations are summarized within the letter of transmittal to Governor Hunt and discussed in detail in Chapter II.

2. Special Report on State Agency Reorganization

The Waste Management Board gave careful consideration to the many advantages and disadvantages of consolidating or centralizing administration of programs that are now in separate agencies. The Waste Management Board decided not to recommend any major reorganization of State agencies at this time. Rather, an issue paper recommending the establishment of a Coordinating Committee was developed by staff and circulated to the Secretaries of the relevant state agencies for comment This issue paper was discussed at length and revised to reflect the needs of all parties involved.

The Special Report mandated in the North Carolina Waste Management Act was approved by the Waste Management Board at its meeting on October 27, 1982, and submitted to the Governor and the General Assembly in January, 1983 as Chapter II.A. of this report. The Waste Management Board sincerely believes that the actions recommended within the Special Report, if implemented, would greatly increase efficiency within State agencies and improve the overall performance of waste management functions in state government.

EIGHT The Board shall each year recommend to the Governor a recipient for a "Governor's Award of Excellence" which the Governor shall award for outstanding achievement by an industry or company in the area of hazardous waste or low-level radioactive waste management.

The process and criteria for the selection of nominees and award recipients was developed by the Public Information/Participation Committee with assistance from the Technical Committee, individuals in private industry, and interested citizens.

Nomination forms for the 1982 awards were developed and mailed out to the following groups:

Chair, Boards of County Commissioners
State Agencies
Consulting Firms
Trade Associations
North Carolina Economic Development Contact List
Local Citizens Groups
Chambers of Commerce
Generators of Hazardous Waste
Generators of Low-Level Radioactive Waste

The Piedmont Waste Exchange graciously volunteered to help in the distribution of over 600 forms as an insert in its newsletter, "The Waste Watcher."

Nominations were also solicited through press releases to business editors of local newspapers and trade magazines and a radio show describing the awards program which aired on 75 stations in North Carolina.

Nominations will first be screened by the Technical Committee, and then reviewed by a panel of judges comprised of:

1 member from the Waste Management Board
1 member in the "public eye," i.e., an elected official

1 member from the university system
2 representatives of industry

The panel of judges for 1982 was:

Dr. Russell Christman, Chairman
Department of Environmental Science & Engineering
School of Public Health
Chapel Hill, NC

Robert Thompson, Physical Scientist
Environmental Sciences, Northrop Services
Research Triangle Park, NC

Jane Sharp, President
Conservation Council of NC
Chapel Hill, NC

Bill Holman, Member
Governor's Waste Management Board
Raleigh, NC

Winslow Hartford, Retired Chemist
Allied Chemical
Professor, Belmont Abbey
Charlotte, NC

Nominees will be judged on the basis of the following criteria:

* Environmental benefits
* Economic returns
* Technological achievement
* Superior management and engineering
* Commitment by company
* Leadership
* Creativity

The final recommendation for the award recipient will be made to the Governor in January, and an awards ceremony will be held in February of each year.

NINE The Board shall promote and participate in discussion with other states concerning development of regional hazardous waste and low-level radioactive waste management agreements.

In December 1980, Congress enacted the Low-Level Radioactive Waste Policy Act of 1980. This legislation declares as a matter of national policy that each state is responsible for providing for the availability of capacity for the disposal of low-level radioactive waste generated within its own borders. Congress also declared that such waste could be most safely and efficiently managed on a regional basis, and it authorized the states to enter into compacts with other states to provide for the establishment and operation of regional disposal facilities for low-level radioactive waste. Constitutionally, a compact is defined as an interstate agreement that requires Congressional approval. This legislation also allows interstate agreements to restrict importation of waste from other regions.

Soon thereafter, representatives of the Governor of North Carolina met with representatives of the governors of other southeastern states to frame an interstate compact for the management of low-level radioactive waste. These negotiators were assisted by the legal staff of the Southern States Energy Board. Meetings were held at 60 day intervals during 1981. In 1982, eight state legislatures considered drafts of compacts and two clusters of states were identified for membership eligibility in the compacts. North Carolina was named as a prospective member of each group and its representatives participated in the exploratory talks conducted in each of these regions.

Essentially, the North Carolina negotiating position included the following elements. The interstate agreements should contain a regional plan for the comprehensive management of all types of low-level radioactive waste, and a regional commission should have the authority to administer the management plan. The entire program should be funded from user fees, collected by the operators of regional facilities, except that an eligible state might be required to pay an entry fee as a condition for membership. The compact commission should have the power to regulate the importation and exportation of wastes as a means of managing known volumes and classes of waste material. Each member state should expect to contribute something to the regional program. There should be no exemptions or special inducements granted for membership, and no special requirements imposed on any state as a condition of membership.

In the summer of 1982, seven governors and their representatives met in Hilton Head Island, South Carolina. They reviewed the work of their representatives and the results of the negotiations since 1980. They agreed to work for the adoption of an interstate compact with the existing facility at Barnwell, South Carolina serving as the first regional disposal facility. Subsequently in meetings at Birmingham and Atlanta, it was agreed that, in addition to the host state of South Carolina, the eligible states would include Alabama, Florida, Georgia, Mississippi, North Carolina, Tennessee and Virginia. Most of North Carolina's other negotiating points were also accepted.

In 1983, the legislatures of each of the eight eligible states will meet in regular session, and the proposed compact will be submitted to them for approval. Although the content of the draft compact has been accepted by the gubernational negotiators, the actual text of the agreement has not been finalized in the appropriate legal form. It is expected that, with the help of the staff of the Southern States Energy Board, the negotiators will agree on the proper form of the document and the sequence for its submission to the various legislatures.

At this time, only one regional compact (Northwest) has been submitted to the Congress for approval. It is possible that the southeastern states' agreement could be submitted as early as mid-1983.

North Carolina's representatives to the 1982 negotiations were Jack Cozort, the Governor's Legal Advisor; Captain William Briner, member of Governor's Waste Management Board; and Robert Jansen, Governor's Senior Policy Advisor. Captain Briner and Mr. Jansen reported to the Waste Management Board throughout the year concerning the progress of the negotiations.

TEN The Board shall assist localities in which facilities are proposed in collecting and receiving information relating to the suitability of the proposed site. At the request of a local government in which facilities are proposed, the Board shall direct the appropriate agencies of State government to develop such relevant data as that locality shall reasonably request.

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1. Inquiries from Citizens

On several occasions, local citizens from counties in which facilities were proposed have attended meetings of the Waste Management Board and its committees. These citizens have been given the opportunity to ask questions and present their views. Citizens' questions have been answered during the meetings whenever possible; other requested information has been furnished as quickly as possible after the meeting.

2. Meeting with Citizens' Group

A member of the Waste Management Board, Mrs. Virginia Newell, and two members of the Department of Human Resources staff met with approximately 100 Greensboro citizens on the evening of January 28, 1982. The meeting was held at the request of citizens concerned about a waste treatment facility which was being proposed for the Pomona section of Greensboro. Mrs. Newell, Bryant Haskins, and Bill Meyer shared information about the permitting process and heard the concerns of the citizens.

3. Local Briefings

In addition, the Waste Management Board plans to conduct "local briefings" in localities in which facilities are proposed. The "local briefings" will be for the exchange of information only, and not to be conducted as a meeting to weigh the merits or impacts of the proposed facility. The Waste Management Board would present information on the

State's permitting process for hazardous waste management facilities, and might assist the locality in developing a negotiation process and plan of action. The Board would also present information concerning the licensing program for all radioactive material users in North Carolina.

4. Information Package

The Public Information/Participation Committee has begun developing a printed informational package for local citizens. This package is to include a Citizen's Handbook, structured to guide and help all participants in the hazardous waste facility siting process address and make decisions as the process unfolds. It will indicate available sources of assistance and information, raise pertinent questions to be answered, and pinpoint specific requirements of participants at each phase of the process. The Waste Management Board plans to print and make available approximately 250 copies of the Citizen's Handbook.

A North Carolina Waste Management Directory and a bibliography are also being developed by the Public Information/Participation Committee and staff. These materials will not be used for mass mailing, but rather they will be mailed in response to individual requests for information.

ELEVEN The Board shall, in accordance with the procedures set forth in G.S. 130-166.17B and G.S. 104E-6.2, make requisite findings and submit its recommendation to the Governor concerning the exercise of the State's preemptive powers.

Under the Waste Management Act of 1981, the Governor's Waste Management Board may recommend to the Governor that the State act to preempt local ordinances that prohibit or prevent establishment or operation of a hazardous waste facility or a low-level radioactive waste facility. G.S. 130-166.17B(c) states that the Board can recommend preemption only if it finds:

1. The facility is necessary to establish adequate capability for management of waste in North Carolina and serves the interest of the citizens of the state as a whole.
2. All legally required state and federal permits have been issued.
3. Local citizens have had adequate opportunity to participate in the decision making process.
4. The construction and operation of the facility will not pose an unreasonable health or environmental risk to the surrounding locality and that the facility developer or operator has taken or consented to take any reasonable measures to avoid or manage forseeable risks and to comply to maximum feasible extent with any applicable ordinance(s).

The Governor can make a final decision only after making the above four findings.

The Legal Committee of the Waste Management Board identified several experts who could be helpful with this function. Three experts agreed to assist the Legal Committee: Bill Campbell, Institute of Government; Don Liner, Institute of Government; and Richard "Pete" Andrews, UNC Institute for Environmental Studies.

Mr. Liner and Mr. Andrews prepared a position paper on the question of how to decide what the Board will consider in determining whether or not there is a "need" for a facility in North Carolina if the Board is petitioned to review an ordinance which prohibits the facility. This issue paper is available from the Waste Management Board's office upon request.

The Committee decided not to try to define "need", but rather for the Board to make this determination on a case-by-case basis considering the circumstances of each case at the time it is presented.

Thus, the Legal Committee agreed to proceed with establishing rules of procedure without defining the "need". On May 26, 1982, the Board voted unanimously to accept the proposed rules and send them to public hearings.

A public hearing was advertised according to the North Carolina Administrative Procedures Act and held at the August 27 Board meeting. The hearing was held open for written comments for 14 days. Comments were received from the following individuals and organizations:

1. William C. Capel, Jr., Attorney for County of Anson;
2. James E. Wall, Jr., Chairman, Anson County Hazardous Waste Management Board;
3. Catherine M. Cameron, Chairperson, Chatham County Chapter, Tri-County Alliance;
4. Jerry T. Roberts, Secretary-Treasurer, North Carolina Textile Manufacturers Association, Inc.;

5. Daniel Read, Acting Executive Coordinator, Conservation Council of North Carolina;

6. Angela Skelton, Associate Director, North Carolina Petroleum Council.

These comments were summarized and presented to the Board, and several of the comments received were incorporated into the rules of procedure. The final form of the rules of procedure for the limited preemption process were adopted by the Board on October 27, 1982 and can be found in Appendix B-7.

TWELVE The Board shall, in accordance with the procedures set forth in G.S. 160-A-211.1 and G.S. 153A-152.1, review upon appeal specific privilege license tax rates which localities may apply to waste management facilities in their jurisdiction.

The Waste Management Act of 1981 authorizes municipalities and counties to levy a privilege license tax on hazardous waste and low-level radioactive waste facilities located in their jurisdiction. (G.S. 160A-211.1 and G.S. 153A-152.1). The locality is authorized to levy a privilege license tax in an amount designed to compensate for the additional costs incurred by having a facility in its jurisdiction.

The Governor's Waste Management Board is authorized to hear appeals from any person or firm taxed under this authority, thus acting as an administrative review body for the purpose of determining the reasonableness of any such tax. The statute does not set out the specifics of this appeal process, leaving it for the Board to establish by regulation.

Two outside experts, Bill Campbell of the Institute of Government and Richard "Pete" Andrews of the UNC Institute for Environmental Studies, agreed to assist the Legal Committee with this function. The Legal Committee decided that it would be unwise to try to define what factors the Board would use in determining whether or not a privilege license tax is reasonable because to do so might limit improperly the factors that localities consider. Rather, the Committee decided that the Board should make this finding on a case-by-case basis.

A public hearing was advertised according to the North Carolina Administrative Procedures Act and held at the August 27 Board meeting. The hearing was held open for written comments for 14 days. Comments were received from the following individuals and organizations:

1. William C. Capel, Jr., Attorney for County of Anson;
2. James E. Wall, Jr., Chairman, Anson County Hazardous Waste Management Board;
3. Catherine M. Cameron, Chairperson, Chatham County Chapter, Tri-County Alliance;
4. Jerry T. Roberts, Secretary-Treasurer, North Carolina Textile Manufacturers Association, Inc.;
5. Daniel Read, Acting Executive Coordinator, Conservation Council of North Carolina.

The comments were summarized and presented to the Board and several of the comments were incorporated into the rules of procedure.

The Rules of Procedure for the Privilege License Tax Review process were adopted in final form by the Board on October 27, 1982, and can be found in Appendix B-11.

THIRTEEN The Board may insure its members against personal liability for any actions they might take pursuant to the exercise of the functions and powers of the Board.

A memorandum was presented to the Board from the Attorney General setting out the potential for liability on the part of Board members and the statutory responsibilities and policies of the Attorney General in representing them if they should be sued. A copy of the memorandum may be obtained from the Governor's Waste Management Board's office.

FOURTEEN The Board may adopt, modify, or revoke any rules necessary to carry out the functions and powers as set forth in this part.

The Board adopted rules of procedure to be used by the Governor's Waste Management Board in:

1. Reviewing petitions for limited preemption of a local ordinance pursuant to the authority set forth in NCGS 130-166.17B and 104E-62.;
2. Reviewing upon appeal specific privilege license tax rates which localities may apply to waste management facilities in their jurisdiction.

These rules of procedure may be found in Appendix B-7 and B-11. The development of these rules is described on pages 27-31.

FIFTEEN The Board shall have any and all powers necessary or incidental to the exercise of the functions and powers enumerated herein.

This is a mandated power, and not a function, of the Waste Management Board. Therefore, there are no activities to report for this item. To date, the Waste Management Board has indeed had the powers necessary to fulfill its mandated functions.

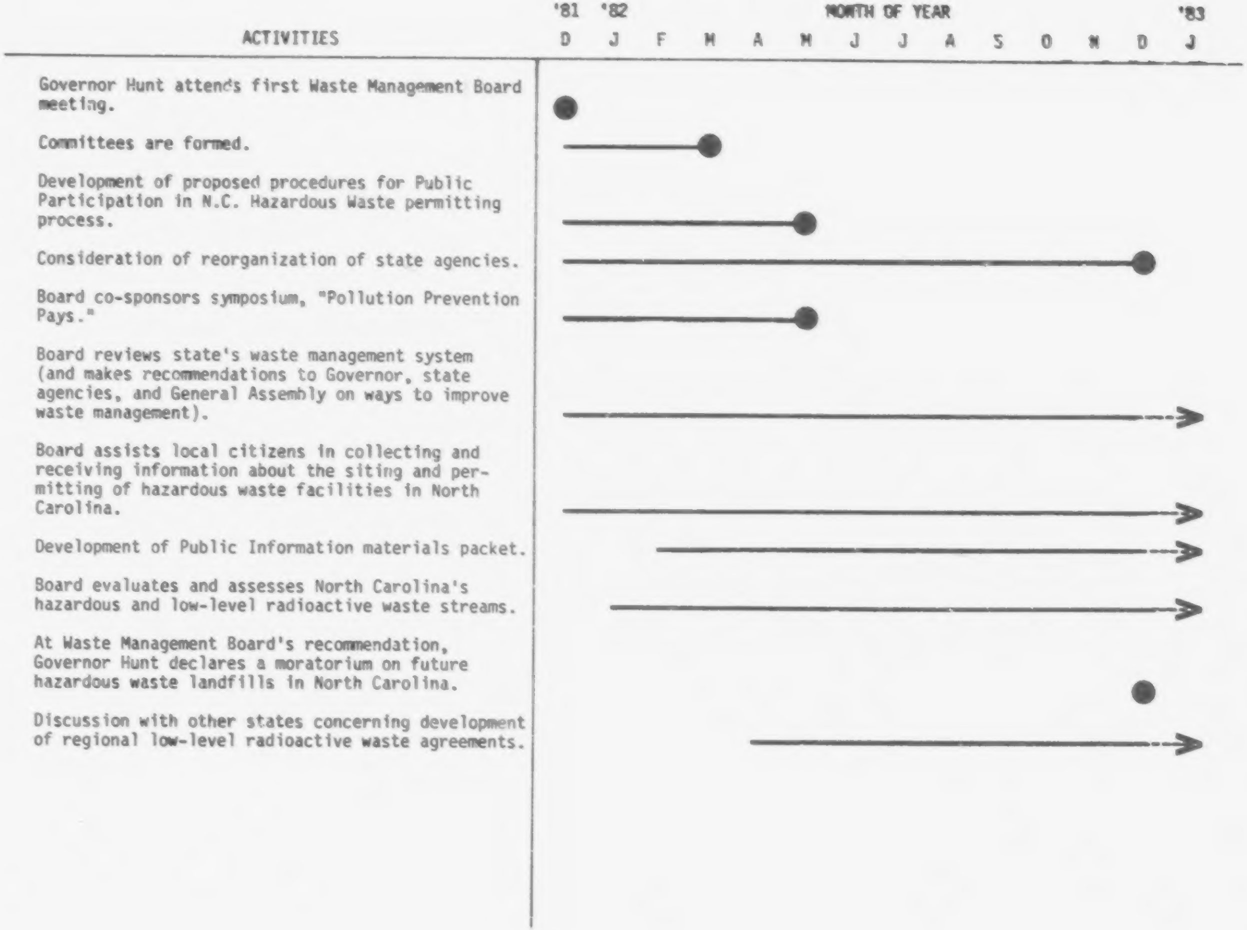
SIXTEEN The Board shall study the development of retrievable, above-ground storage facilities for hazardous wastes.

The major thrust of the Waste Management Board's efforts throughout 1982 has been to study the existing waste management practices and to develop a waste management program for North Carolina. The Waste Management Board feels very strongly that this program must emphasize waste elimination, waste reduction, and waste recycling, thereby ultimately diminishing the volume of waste which requires disposal.

It is because of this preventive focus that the Waste Management Board has not yet made an in-depth study of the development of retrievable, above-ground storage facilities for hazardous wastes. Once the "Analysis of North Carolina's Industrial Waste" is complete, the Board will be better able to determine the volume of hazardous waste which would still require disposal after all known methods of waste prevention or recycling were implemented in North Carolina. The Waste Management Board will then study the development of retrievable, above-ground storage facilities for hazardous waste, and will file a Supplemental Report of its findings and recommendations with the Governor and the General Assembly.

CHAPTER I: ACTIVITIES DURING 1982 CATEGORIZED BY MANDATED FUNCTIONS AND POWERS

PART B: TIME LINE OF ACTIVITIES



ACTIVITIES

Governor Hunt attends first Waste Management Board meeting.

Committees are formed.

Development of proposed procedures for Public Participation in N.C. Hazardous Waste permitting process.

Consideration of reorganization of state agencies.

Board co-sponsors symposium, "Pollution Prevention Pays."

Board reviews state's waste management system (and makes recommendations to Governor, state agencies, and General Assembly on ways to improve waste management).

Board assists local citizens in collecting and receiving information about the siting and permitting of hazardous waste facilities in North Carolina.

Development of Public Information materials packet.

Board evaluates and assesses North Carolina's hazardous and low-level radioactive waste streams.

At Waste Management Board's recommendation, Governor Hunt declares a moratorium on future hazardous waste landfills in North Carolina.

Discussion with other states concerning development of regional low-level radioactive waste agreements.

Development of rules for Limited Preemption Process.

Rules for Limited Preemption Process filed, go into effect.

Development of rules for Privilege License Tax.

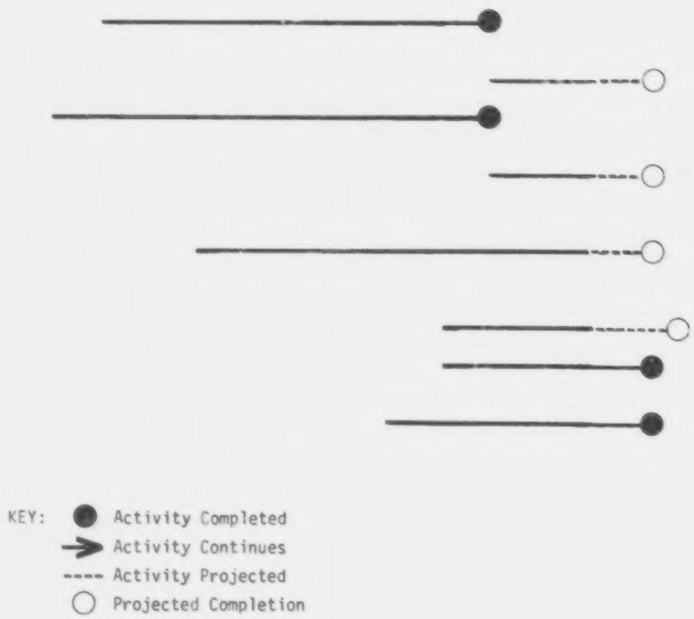
Rules for Privilege License Tax filed, go into effect.

Board studies desirability of establishing by statute a standard of strict liability; report submitted to Governor and General Assembly.

Development of Governor's Award of Excellence selection process; awards recipient recommended to Governor.

Development of Annual Report.

Development of Special Report on reorganization of state agencies.



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CHAPTER II: RECOMMENDATIONS FOR ADMINISTRATIVE OR REGULATORY ACTION

PART A: SPECIAL REPORT ON REORGANIZATION OF STATE AGENCIES

1. Introduction

The establishment of the Governor's Waste Management Board was significant in that the General Assembly, acting on recommendations from the Governor's Task Force on Waste Management, recognized the desirability of having the waste problem addressed by a board comprised of North Carolina citizens and state agencies involved in the regulatory program.

While the Board does not have statutory authority to promulgate rules and regulations on hazardous and low-level radioactive waste, the Board is charged with the legal responsibility of reviewing the state's comprehensive waste management system and make recommendations to the Governor, state agencies, and the General Assembly on ways to improve waste management. The Board is also to study and make recommendations on policy issues.

Where changes may be found necessary, the Board will recommend those changes to the affected regulatory commission, i.e., Environmental Management Commission, Commission for Health Services, Radiation Protection Commission, North Carolina Pesticides Board and any others that may exist or be formed.

In order to carry out the responsibilities as outlined above, the Governor's Waste Management Board at its October 27, 1982, meeting adopted unanimously the following policy statement and method of carrying it forth.

2. Policy Statement

a. The Board should request and receive from any state agencies, for its review and comments, proposed changes in state laws or rules of procedure that impact on the management of hazardous and low-level radioactive waste.

b. The Board should review requests for state funds for the management of North Carolina's program.

c. The Board should be an advocate for state agencies before the Advisory Budget Commission and the General Assembly when appropriate, to insure that adequate state funds are available for the management of the program.

d. The Board should be a review agency for requests for funds by departments of State Government from sources other than the state for any program purported to support North Carolina's Waste Management Program.

e. The Board should coordinate the dissemination of information on hazardous waste research that is being conducted by educational institutions in North Carolina.

3. Mechanism

The mechanism for carrying out this newly established Board policy will be the formation of a Waste Management Coordinating Committee under the Governor's Waste Management Board. The formation of this committee will allow an open forum in which problems and concerns on managing hazardous and low level radioactive waste could be addressed on a consistent basis.

4. Committee Composition

The Membership of the Committee will consist of one member from the Commission for Health Services, Environmental Management Commission, Radiation Protection Commission, N.C. Pesticide Board, Secretaries of the Department of Human Resources, Natural Resources and Community Development, the Commissioner of Agriculture and the Governor's Waste Management Board or their designees.

The Secretary of the Department of Human Resources or his/her designee will serve as Chairman of the Coordinating Committee. Support staff will be furnished by the three departments. The staff of the Governor's Waste Management Board will coordinate the work for the committee and see that the tasks are completed.

5. Committee Functions

The functions of this committee will be to: 1) look at long range planning; 2) consider all requests for funds and personnel as they affect waste management; 3) consider and comment on grant applications that affect waste management; 4) consider and make recommendations on administrative or regulatory changes that may be necessary for a more cohesive program; 5) consider proposed regulatory changes recommended by the various commissions as to their interdepartmental impacts; and, 6) any other matters that the Governor's Waste Management Board may ask the committee to consider.

6. Govenor's Waste Management Board Action

All recommendations from this committee will go to the Governor's Waste Management Board for review and final recommendation. The Coordinating Committee will be advisory in nature and will not have statutory authority for promulgating rules and regulations outside of those granted to the individual commissions by law. The meetings of the Coordinating Committee should be scheduled in such a manner as to allow any recommendations that may come from this committee to be presented to the full commissions represented by the committee. The Governor's Waste Management Board will not take any action on recommendations from the Coordinating Committee until comments have been received from the affected commissions and agencies. Those recommendations on changes that may affect city and county governments should be referred to the appropriate organizations to allow for dissemination of this information and the receipt of input on the issue.

7. Conclusion

With the formation of the Committee, the objectives of the General Assembly's intent to have a closely coordinated waste management program can be carried out. This action can be achieved without massive reorganization of departments, which would be extremely costly and could cause delay in implementing the waste management program. The mechanism does exist for bringing the formation of this committee about. Refining the existing system should produce the better product for North Carolina.

The Governor's Waste Management Board recommends this Coordinating Committee in order to enhance and strengthen the present coordination of waste management activities between state agencies and departments.

If the Governor and General Assembly feel that a study of reorganization of all environmental programs be done, funds should be appropriated to the Board for this study or the General Assembly should consider other means of achieving this task.

CHAPTER II: RECOMMENDATIONS FOR ADMINISTRATIVE OR REGULATORY ACTION

PART B: SPECIAL REPORT AND RECOMMENDATIONS ON LIABILITY ISSUES

1. Introduction and Acknowledgements

The Waste Management Act of 1981 created the Governor's Waste Management Board to perform a number of functions relating to the safe management and disposal of hazardous wastes and low-level radioactive wastes in North Carolina. Among these mandated functions is the following:

> The Board shall study and make recommendations on policy issues including but not limited to liability and financial responsibilities within the waste management area. On or before January 1, 1983, the Board shall prepare and present to the Governor and General Assembly a report concerning the desirability of establishing by statute a standard of strict liability for persons involved in storage, transportation, treatment, or disposal of hazardous or low-level radioactive waste in North Carolina.

To carry out this mandate, the Waste Management Board referred the matter to its Legal Committee for study and recommendations. The Legal Committee is chaired by Charles Holt, former State Representative from Cumberland County. In addition to Representative Holt, the Committee is constituted of the two Board members who are members of the legal profession; Heman Clark, former Superior Court Judge and presently Secretary of the North Carolina Department of Crime Control and Public Safety, and William Graham, former member of the North Carolina Court of Appeals and presently Senior Vice President and General Counsel for Carolina Power and Light. The fourth member of the Committee is Melvin Hearn, Deputy Secretary of the North Carolina Department of Agriculture. Glenn Dunn, Attorney with the Solid and Hazardous Waste Management Branch of the North Carolina Department of Human Resources was the Staff Coordinator for the Committee's study.

The composition of the Committee brought an excellent combination of viewpoints to bear on a subject which demands a blend of policy considerations and technical understanding of a complex area of the law. The Committee requested and received valuable assistance from Robert Byrd, Professor of Torts and former Dean of the School of Law at the University of North Carolina at Chapel Hill.

In addition to Professor Byrd's assistance, the Committee benefitted greatly from the viewpoints and expertise of other persons or organizations. The Committee was fortunate to have had available to it since September 1982, a very thorough Report to Congress entitled Injuries and Damages from Hazardous Wastes - Analysis and Improvements of Legal Remedies. This Report is the culmination of a year-long study conducted by a carefully chosen group of lawyers with a variety of viewpoints and considerable expertise. One member of the group that produced the study was George C. Freeman, Jr., of Hunton and Williams, a law firm in Richmond, Virginia, with an extensive environmental law practice. Mr. Freeman, with considerable assistance from Alfred Light, also of the firm, donated much time and expertise to the efforts of the Legal Committee and attended several meetings.

The American Insurance Association, a professional association representing a substantial number of insurance companies, was represented at practically all committee meetings and contributed to consideration of the possible effects of various liability reforms on the insurance industry. The North Carolina Petroleum Council and North Carolina Textile's manufacturers were represented from time to time at the meetings.

With the aid of the above-mentioned persons and groups, the Committee reached its recommendations[1] and presented them to the Waste Management Board at a public meeting held in Raleigh on December 1, 1982. Comments were received at that meeting from the following: John Runkle, Conservation Council of North Carolina; Dan Stroh, North Carolina Sierra Club; Wes Hart, North Carolina CATCH; and Cathy Markatos, Tri-County Alliance.

After considerable discussion, the Board accepted the recommendations set forth in the remainder of this report for presentation to the Governor and General Assembly. These recommendations were made only in regard to hazardous waste. The Governor's Waste Management Board will soon file a Supplemental Report addressing whether or not strict liability should apply to low-level radioactive waste.

2. Recommendations

a. Application of Strict Liability for Injuries Caused by Hazardous Waste

RECOMMENDATION 1 -- The Board recommends that strict liability be applied by statute to any generator, transporter, treater, storer, or disposer of hazardous waste in control of the waste at the time it causes injury to a claimant, and that strict liability on the part of a generator continue while the waste is transported and until it is accepted by a properly licensed treatment, storage, or disposal facility.

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[1] The Committee's discussion of the liability question, including more thorough descriptions of the issues considered and the reasons for the recommendations, is available in a separate report that may be obtained from the office of the Governor's Waste Management Board.

Imposition of strict liability will help ensure that one of the barriers to meritorious claims, particularly smaller claims, is removed. Strict liability should also cause handlers of hazardous waste to use the greatest possible care to employ practices that minimize the risk of injury.

Transporters are not required under RCRA to carry a certain minimum amount of liability insurance. For this reason, the likelihood that large claims for damages caused by waste in transport could go uncompensated because the transporter has inadequate assets is a serious problem. Continuing generator liability through the transport stage helps overcome this problem. On the other hand, under the recommended approach, a generator's strict liability does not continue indefinitely because it is terminated when the waste is accepted at a permitted and regulated treatment, storage, or disposal facility. Unlike transporters, facility operators are required under RCRA to carry liability insurance, and it is therefore less likely that they will be unable to compensate for damages.

The recommended apportionment of damages where strict liability is applied would be as follows: a person injured by a waste under control of a generator would seek recovery only from the generator under a standard of strict liability; a person injured by waste in the transport stage could seek recovery from the transporter in control of the waste at the time it caused the injury, from the generator, or from both because they would be jointly and severally liable under a standard of strict liability; a person injured by waste at a treatment, storage, or disposal facility could seek recovery only against the owner or operator of the facility under a standard of strict liability.

This approach can be implemented by providing by statute that the person in control of a waste shall be strictly liable for damages caused by that waste; and furthermore a generator of a hazardous waste shall be strictly liable for any damages caused by that waste, except that it shall be an absolute defense if the generator can show that the waste has been properly delivered to and accepted by a treatment, storage and disposal facility properly permitted to receive the waste.

This recommendation blends well with the regulatory program which, among other things, requires that a generator prepare and keep a written "manifest" for any waste shipped by him. The statute could provide that the manifest gives rise to a presumption that the waste was shipped to and accepted by a permitted facility. This presumption would encourage generators to comply with the manifest system.

RECOMMENDATION 2 -- The Board recommends that strict liability apply only to active facilities and ongoing activities.

The existing statutes requiring responsible parties to clean up inactive facilities should be given a chance to succeed in minimizing potential injuries. It is at best harsh, and at worst unconstitutional, to apply statutorily a standard of strict liability for injuries caused by past acts. In such cases the appropriate common law standard should apply.

RECOMMENDATION 3 -- The Board recommends that strict liability apply only for injuries resulting from risks associated with the characteristics that make the waste hazardous.

There is no justification for applying strict liability for injuries caused by the normal activities of a hazardous waste handler if those injuries are not caused by the waste itself. It is, after all, the hazardous nature of the waste that justifies application of strict liability.

RECOMMENDATION 4 -- The Board recommends that a handler of hazardous waste be held strictly liable for all injuries caused by the waste, regardless of whether he knew at the time of injury that the waste could cause that particular type of injury.

This recommendation is consistent with the goal of emphasizing compensation regardless of fault. The Board emphasizes that this does not mean that strict liability will be applied retroactively in the case of a waste that was not considered hazardous at the time of the act that caused the injury. It only applies to an unknown effect from a waste known to be hazardous at the time it causes the injury.

RECOMMENDATION 5 -- The Board recommends that strict liability apply to successors in interest as well as to the generator, transporter, treater, storer or disposer in control of the waste at the time it causes the injury.

This approach would help avoid cases where an injured person is unable to recover damages because the company that created the hazard has passed out of existence or sold a site with a latent hazardous waste problem to another person. In such cases, the new owner may be the only person able to compensate the injured person. Owners of facilities are required to record on the deed that the site has been used as a hazardous waste facility. Therefore, subsequent purchasers should be on notice and have full opportunity to assess the risks they may be acquiring.

RECOMMENDATION 6 -- The Board recommends that punitive damages not be allowed for claims where strict liability is applied.

This recommendation is consistent with the goal of compensating claimants for injuries without consideration of fault on the part of the defendant, and may help avoid extremely large recoveries based on punitive damages that render a defendant financially unable to compensate other injured persons. Sufficient punitive remedies are provided by statute in the form of criminal and administrative penalties for hazardous waste handlers that violate regulations.

RECOMMENDATION 7 -- The Board recommends that although strict liability is imposed, contributory negligence of a claimant will bar any recovery by that claimant.

An injured person should not be able to recover, even where strict liability is imposed, if his own negligent acts are an essential cause of the injury.

b. Statute of Limitations

RECOMMENDATION 8 -- The Board recommends that the statute of limitations begin to run only when the injury becomes apparent or ought to become apparent to an injured person, and that the ten-year statute of repose not apply.

The purpose of statutes of limitations are practical ones: (1) to ensure that legal actions are not initiated so long after the time of the acts alleged to have caused the damage that the facts necessary to litigating the matter have "grown cold" (i.e., are no longer capable of accurate determination), and (2) to ensure that persons are not subjected indefinitely to the threat of liability for past actions.

When strict liability is applied, the first reason becomes less important because there is no need to recall facts relevant to whether the defendant's acts were negligent. The main issue of fact becomes whether the hazardous waste under the control of the defendant caused the injury, an issue based more on scientific data than on facts relating to the defendant's actions. Furthermore, statutes of limitations have no deterrent value and in the case of injuries with long latency periods, they provide a shield for waste handlers.

Commencement of the running of the statute of limitations can be an unfair barrier to recovery for injuries caused by hazardous wastes. Many of the effects of chemical wastes may not manifest themselves for many years (i.e., have long latency periods). This same problem exists in relation to many other types of injuries in our society today. The fairness of statutes of limitation that run from the time of the act of the defendant has been challenged and most states have statutes that run from the time of "discovery" of the injury by the injured party.

North Carolina's statute [N.C.G.S. 1-52(16)] presently provides that an action must be brought within 3 years "from the time that the injury or damage becomes apparent or ought reasonably to have become apparent to the claimant," but then provides an absolute bar, known as a statute of repose, against any action brought "more than 10 years from the last act or omission of the defendant giving rise to the cause of action." Thus, in North Carolina an injured party has three years after the discovery of the injury in which to initiate an action for recovery; however, no action may be brought after ten years under any circumstances.

The goal of facilitating compensation of injured persons outweighs the practical advantages of putting potential legal actions to rest after a certain period of time. This is especially important in cases where the condition causing the damage may develop very slowly (for example, a buried toxic waste may take many years to reach adjacent water supplies) and where the injury from the condition may not manifest itself for many more years.

c. Causation and Related Evidentiary Requirements

RECOMMENDATION 9 -- The Board recommends that there be no attempt to ease the plaintiff's burden of proving that the injury or damage was caused by the hazardous waste handling activity of the defendant.

As the law presently stands, a plaintiff must prove in each case that there is a causal connection between the disease or injury complained of and the hazardous waste related activity of the defendant. Proof of this causal connection is usually difficult in these cases for several reasons.

First, a plaintiff must show the nature of the exposure - whether by inhalation, ingestion or other contact - in order to show the duration, frequency and intensity of exposure. If exposure is adequately proven, proof of causation will usually require large amounts of sophisticated and thus very expensive, medical and scientific testimony to demonstrate the epidemiologic or statistical correlation between the hazardous waste(s) in question and the injury or disease.

Additional problems of proof stem from the fact that there is often a long latency period before effects of exposure become obvious, and during that period the injured party may have been exposed to a wide variety of substances from other sources. Furthermore, a facility alleged to cause the injury may have handled many types of waste which may interact to cause effects that none of them would cause alone, making it more difficult to determine the exact cause of the injury.

Finally, medical science has simply not been able to determine the effects of many chemicals already on the market and, of course, even less will be known at any given time concerning the effects of new chemicals that enter the market constantly.

The difficulties in proving causation have been eased in Workers Compensation Laws by a "rebuttable presumption" approach. Under this approach, if the plaintiff can prove that he suffers from certain types of injuries and that he has had a certain amount of exposure to the substance in question that is known to cause those injuries, the burden shifts to the defendant to prove that the substances within his control did not cause the injury.

This "rebuttable presumption" approach is considered fair because a substantial body of information exits concerning certain substances (for example, asbestos) to show a strong statistical correlation between exposure to the substance and the subsequent development of certain diseases [for example, in the case of asbestos, mesothelioma (lung cancer)]. This is particularly valid and useful in worker's compensation cases because employees are exposed day-in and day-out to a known set of substances.

However, in the case of injuries allegedly stemming from hazardous wastes, the plaintiff will less often be subject to constant exposure over a long period of time and the exposure is likely to be to a greater variety of substances or, for that matter, many unknown substances. This should become even more the case now that strict regulatory programs are in effect which tend to isolate the waste in closely controlled facilities. Thus a presumption of causation in the case of alleged injuries from hazardous waste is probably not as valid or fair as in workplace injury cases. Even in the workplace injury context, however, there have been abuses. For example, Congress in 1981, felt it necessary to abolish several statutory "rebuttable presumptions" in the Black Lung Benefits program because many successful claimants under the program did not have adequate medical evidence of black lung disease.

There is at present insufficient indication that there will be a large number of cases alleging injuries from similar types of wastes, which is the circumstance that justifies a presumption of causation. At present, it seems likely that claims for injuries will involve a wide variety of wastes, and that causation must be verified on a case-by-case basis.

d. Administrative Mechanisms for Determining Liability and Compensating Injured Persons

RECOMMENDATION 10 - The Board does not recommend at this time that an administrative agency be created to adjudicate strict liability in hazardous waste cases.

There are two key factors to consider in deciding whether an agency rather than the courts should adjudicate liability. (1) Will there be enough claims to justify establishing a separate system? and (2) Are the cases likely to be so complicated that special judges or panels are needed to resolve them?

Concerning the first question, there is little actual proof yet that there will be a great increase in toxic tort cases, although it is logical to assume that there will be at least some increase due to the proliferation of chemicals in the last 20-25 years and the typically long time it takes to discover an effect. Furthermore, the implementation of a very stringent regulatory program should reduce the potential for injuries. At this point it seems premature to assume that the case load will justify a separate system.

Concerning the second question, the complexity of these cases will certainly be reduced if strict liability is applied, although the remaining issues of causation are highly technical and the use of administrative law judges with particular expertise might promote more consistent decisions.

The Board concluded that it is premature at this time to assume that the volume or complexity of hazardous waste liability cases will be so great that the general court system cannot handle them adequately.

RECOMMENDATION 11 - The Board does not recommend at this time that a fund be established for compensating injured claimants.

The advantage of a fund is that it would ensure that the money is available to compensate a person with a meritorious claim, that the compensation could be immediate and that identification of a specific responsible party would not be necessary. However, the fact that most of the facilities are required to have liability insurance for precisely this purpose certainly reduces the need for a fund. Furthermore, use of a fund entails some administrative costs that will increase the "transactional" cost that we are trying to avoid.

If compensation is made from a fund rather than by the responsible party, then compensation is divorced from responsibility and has no deterrence value unless the agency administering the fund is empowered to seek recovery from the reponsible person(s). Such a process has potential to increase even further the "transactional" costs. A fund would appear more appropriate in the abandoned or inactive site context, where the identity of a potentially responsible party may be unknown This is the area Congress will be studying closely in connection with the report required by Section 301(e) of the federal Superfund law. California has adopted an "orphan site" fund to compensate those injured by abandoned sites, but that state is apparently the only one which has found such a system necessary.

Furthermore, any attempt to establish a fund would entail difficult problems of deciding who should provide the money for the fund, whereas direct compensation by the responsible defendant places financial responsibility where it should be. Mandatory liability insurance requirements should be adequate to ensure that the defendant will be able to provide compensation in most cases.

CHAPTER II: RECOMMENDATIONS FOR ADMINISTRATIVE OR REGULATORY ACTION

PART C: OTHER RECOMMENDATIONS

In addition to the recommendations outlined within the Special Report on Reorganization of State Agencies and the Special Report on Strict Liability, the Waste Management Board offers the following recommendations for administrative or regulatory action.

RECOMMENDATION 1 -- The Governor's Waste Management Board requests that the General Assembly give to the Solid and Hazardous Waste Management Branch the authority to require annual reports from all generators, transporters, storers and disposers of hazardous waste (1000 kilograms or more) in North Carolina.

On March 15, 1982, the Department of Human Resources, Division of Health Services, Solid and Hazardous Waste Management Branch was notified that the Environmental Protection Agency had postponed its requirement that major generators of hazardous waste file annual reports. Once the Environmental Protection Agency postponed this requirement, the Solid and Hazardous Waste Management Branch could not by North Carolina law require the annual report.

The Governor's Waste Management Board finds the data collected from these annual reports to be most valuable. It is the opinion of the Board that the State should continue to require the annual report for some period of time in order to track its program of managing hazardous and low-level radioactive waste.

RECOMMENDATION 2 -- The Governor's Waste Management Board recommends that sufficient funds be appropriated by the General Assembly for a reasonable regulatory program to protect the health, safety, and environment of the people of North Carolina.

The acceptability of a waste management plan by the people of North Carolina will be primarily based on their beliefs in the state and federal agencies' abilities to protect their health, safety, and their environment. With the continued cutbacks and elimination of federal funds, North Carolina must assume more and more of the financial support of the regulatory and response programs. Statutes for protecting the public health and environment without sufficient personnel and funds to support their mandates will be unacceptable. North Carolina must begin now to look at its budgets for regulatory programs with long range planning in mind to be sure that the program's integrity is maintained.

RECOMMENDATION 3 -- The Governor's Waste Management Board recommends that adequate funding be appropriated by the General Assembly to monitor and clean up old waste sites.

The State regulates, through its permitting procedures, those industries which could produce health threatening situations through accidental discharge of hazardous materials or waste. Even under the most stringent controls and safety measures, accidents will happen and the State must be in a position to respond. A concern voiced often by the public is "if something does happen, what will the State do?" Establishment of guilt before responding to a health threatening situation and searching for funds to clean up the environment is of little comfort to those who are threatened.

The "Oil and Other Hazardous Substances Pollution Fund," General Statute 143-215.87, 215.88, 215.89, 215.90, appears to be the mechanism already in place to house funds for use by North Carolina in responding to clean up. The fund currently has a balance of approximately $33,000. At present, this is the only available source of money with which the State may respond to the need for removal of oil and other hazardous substances and for restoration of areas polluted by discharges of such substances. The fund is not adequate for contingencies which are likely in North Carolina.

If the State enters into a cooperative agreement with EPA to utilize federal Superfund monies for emergency response activities, it will be required to maintain the Pollution Protection Fund as a form of match requirement. If Superfund monies are not available, the State must still be in a financial position to immediately respond to a clean-up and then seek reimbursement, through court action if necessary, from the guilty parties. Money could also be used from this fund to clean up several of the orphan sites that may need some remedial action. G.S. 143-215.87 may require some amendments to be certain that the law allows for the Oil and Other Hazardous Substances Pollution Protection Fund to be spent for cleaning up orphan sites as well as future spills.

APPENDIX A: GLOSSARY

CORROSIVE WASTES	Substances that eat away materials and living tissues by chemical action. They are of particular concern to persons involved in waste hauling and disposal because they can corrode containers and cause leakage. Examples: alkaline cleaners, acid liquids used in etching, and wastes from battery production.
HAZARDOUS WASTES	The Resource Conservation and Recovery Act of 1976, under which hazardous substances are regulated, defines a hazardous waste as "a solid waste or combination of solid wastes, which because of its quantity, concentration or physical, chemical or infectious characteristics may: -- cause or significantly contribute to an increase in mortality or an increase in serious irreversible, or incapacitating reversible illness. -- pose a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported, or disposed of, or otherwise managed." These waste materials can be either solids, liquids, sludges, or contained gases. A hazardous waste has at least one of four characteristics: ignitability, corrosivity, reactivity, or toxicity.
IGNITABLE WASTES	Substances which catch fire easily and as a result normally are segregated from other waste materials. Fires involving hazardous substances not only are dangerous because they generate heat and sometimes toxic smoke, but also because they can spread harmful particles over large areas. Examples: discarded organic solvents, such as toluene and benzene, oils, plasticizers, some pesticides and paint and varnish removers.

LOW-LEVEL RADIOACTIVE WASTES	The Nuclear Regulatory Commission, the federal government agency which regulates the use of commercial radioactive material, defines low-level radioactive wastes as any commercial radioactive waste other than the products from reprocessing used nuclear power plant fuel rods, the by-products of the mining and milling of uranium ore, or certain long-lived radioactive materials which require extremely long periods of isolation from the environment. As their name indicates, low-level radioactive wastes generally contain very low levels of radioactivity. They usually consist of everyday items which have become slightly radioactive through exposure to a radioactive element. Though they present a very small hazard when compared with high-level wastes, low-level radioactive wastes must still be handled carefully and disposed according to strict regulations. Examples: paper, plastic, glassware, disposable syringes, clothing or gloves from medical or research laboratories; or filters, filter sludges, mechanical equipment, piping and liquid wastes from nuclear power plants or industrial operations.
REACTIVE WASTES	Substances which may during routine management react spontaneously, react vigorously with air or water, be unstable to shock or heat, generate toxic gases, or explode. Examples: obsolete munitions and manufacturing wastes from the explosives and chemical industries.
TOXIC WASTES	Any substance (solid, liquid, or gas) that are poisonous to living beings. If they are improperly handled, these wastes or their by-products may be released into the environment and harm human health, particularly through the contamination of ground water. Not all hazardous wastes are toxic, but all toxic wastes are potentially hazardous. Examples: pesticides, arsenic, cadmium, and their salts.

STATE OF NORTH CAROLINA
OFFICE OF THE GOVERNOR
RALEIGH 27611

JAMES B. HUNT, JR.
GOVERNOR

WASTE MANAGEMENT BOARD
March 15, 1982

Mr. Grover Lancaster, Jr., President
NC Association of County Commissioners
Route 1
P.O. Box 412
Vanceboro, North Carolina 28586

Dear Grover:

Thank you for taking time from your busy schedule to meet with the Governor's Waste Management Board on March 3. We sincerely appreciate your offers for help and assistance to the Board as we begin to put together a Waste Management Program in North Carolina.

One of the most important roles of this Board is to insure that correct and proper information is received by the local units of government and by the people who live in our state. Therefore, one of the first suggestions that we would like to discuss with your respective organizations is how you may help encourage the formation of Citizen Involvement Committees on waste management on the local level. Meaningful citizen involvement and participation is critical if an effective waste management system is to be developed in North Carolina.

Waste management facilities, in particular, have met with much suspicion and resistance from citizens due, at least in part, to poor communications between the facility developer, state regulatory agencies, and local citizens. These same problems may occur with existing facilities if proper education and information is not disseminated to local governing bodies and to your citizens who live near existing manufacturing facilities who have a waste stream that may contain some hazardous waste, or an existing company that is a transporter, storer, or processor of hazardous waste. Past experience in other states indicates that early and thorough public participation and education must and should be encouraged.

The Governor's Waste Management Board recommends that local Citizen Involvement Committees on waste management be established. These committees would serve as a forum for exchange of information and opinions between state regulatory agencies and the involved localities. This should provide state agencies with a means for understanding and addressing local concerns. This approach should help avoid misinformation and distrust by educating the citizenry about the rules and regulations that govern the operation of existing facilities and of any new ones that may be proposed.

The formation of Citizen Involvement Committees on waste management is of course optional. Nevertheless, widely based local participation is vital. The Governor's Waste Management Board recommends that committees be organized, and put into operation prior to the permitting of existing or new facilities so that the community will have a good grass roots understanding of what is happening within their own areas and why.

Listed below is a suggested organizational chart for the local citizen committee.

I. Organization

  A. Citizens Organizations
  B. County Government
  C. Municipal Government
  D. Local Industry and Commerce
  E. Local Health and Environmental Agencies or Groups
  F. Local Education and Research Institutions
  G. Local Transportation and Shipping Interest
  H. Agricultural Interest

Nominations should be allowed by anyone from the groups or agencies to be represented. Appointment of the committee should be made by the county commissioners after receipt of these nominations with one commissioner representing county government. Functions of the committee could be, but are not limited to, the following:

1. To meet with the state and private industry to determine the nature and scope of operations of existing facilities and any proposed new facilities that may be under consideration.

2. To participate in planning public meetings, distribution of information and such other steps that should be taken to fully inform all the people of the community about what currently exists or is being proposed, including the advantages and disadvantages.

3. To take steps in conjunction with state and local government and private industry to insure that response is made to questions and concerns of the citizens of the affected areas in so far as available knowledge and understanding permit.

4. To recommend to state and local governments and private industry those measures that should or could be taken to make the location of an existing or new facility acceptable to the citizens.

There may already be in existance, community organizations whose role could be expanded to become a Citizen Involvement Committee on waste management.

The members and staff of the Governor's Waste Management Board stand ready to assist you.

Sincerely,

Buck

Buck O'Shields, Chairman
Governor's Waste Management Board

BO/ad

cc: Ron Aycock
  All Board Members

NORTH CAROLINA ASSOCIATION OF COUNTY COMMISSIONERS

MEMORANDUM

TO: Chairmen and Managers

FROM: Grover Lancaster, Jr.
President

DATE: March 17, 1982

RE: Meeting - Governor's Waste Management Board

On March 3, I met with the Governor's Waste Management Board to discuss with them ways that our Association could be of assistance.

The Governor's Waste Management Board feels that the creation of local Citizen Involvement Committees on waste management will be of great importance in establishing an effective program in North Carolina.

Attached is a letter I received from Buck O'Shields, Chairman of the Governor's Waste Management Board, and I want to share it with you. If you have any questions, please feel free to call Buck at the Waste Management Board Office in Raleigh. It is located on the fifth floor of the Albemarle Building, and the phone number is (919) 733-9020.

ALBERT COATES LOCAL GOVERNMENT CENTER
215 N DAWSON ST • P O BOX 1488 • RALEIGH, NORTH CAROLINA 27602 • TELEPHONE [illegible]

STATE OF NORTH CAROLINA
OFFICE OF THE GOVERNOR
RALEIGH 27611

JAMES B. HUNT, JR.
GOVERNOR

WASTE MANAGEMENT BOARD
March 15, 1982

Mayor Hugh Currin, President
NC League of Municipalities
P. O. Box 147
Oxford, North Carolina 27565

Dear Hugh:

Thank you for taking time from your busy schedule to meet with the Governor's Waste Management Board on March 3. We sincerely appreciate your offers for help and assistance to the Board as we begin to put together a Waste Management Program in North Carolina.

One of the most important roles of this Board is to insure that correct and proper information is received by the local units of government and by the people who live in our state. Therefore, one of the first suggestions that we would like to discuss with your respective organizations is how you may help encourage the formation of Citizen Involvement Committees on waste management on the local level. Meaningful citizen involvement and participation is critical if an effective waste management system is to be developed in North Carolina.

Waste Management facilities, in particular, have met with much suspicion and resistance from citizens due, at least in part, to poor communications between the facility developer, state regulatory agencies, and local citizens. These same problems may occur with existing facilities if proper education and information is not disseminated to local governing bodies and to your citizens who live near existing manufacturing facilities who have a waste stream that may contain some hazardous waste, or an existing company that is a transporter, storer, or processor of hazardous waste. Past experience in other states indicates that early and thorough public participation and education must and should be encouraged.

The Governor's Waste Management Board recommends that local Citizen Involvement Committees on Waste Management be established. These committees would serve as a forum for exchange of information and opinions between state regulatory agencies and the involved localities. This should provide state agencies with a means for understanding and addressing local concerns. This approach should help avoid misinformation and distrust by educating the citizenry about the rules and regulations that govern the operation of existing facilities and of any new ones that may be proposed.

The formation of Citizen Involvement Committees on waste management is of course optional. Nevertheless, widely based local participation is vital. The Governor's Waste Management Board recommends that committees be organized, and put into operation prior to the permitting of existing or new facilities so that the community will have a good grass roots understanding of what is happening within their own areas and why.

Listed below is a suggested organizational chart for the local citizen committee.

I. Organization

- A. Citizens Organizations
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1. To meet with the state and private industry to determine the nature and scope of operations of existing facilities and any proposed new facilities that may be under consideration.

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4. To recommend to state and local governments and private industry those measures that should or could be taken to make the location of an existing or new facility acceptable to the citizens.

There may already be in existance, community organizations whose role could be expanded to become a Citizen Involvement Committee on Waste Management.

The members and staff of the Governor's Waste Management Board stand ready to assist you.

Sincerely,

Buck

Buck O'Shields, Chairman
Governor's Waste Management Board

BO/ad

cc: Leigh Wilson
All Board Members

From League of Municipalities
Magazine

MAY 21 1982

Waste Management Board Recommends Local Citizen Involvement

At the recommendation of its Public Participation Subcommittee, chaired by Winston-Salem Alderwoman Virginia Newell, the Governor's Waste Management Board recently adopted a policy statement calling for the establishment of local Citizen Involvement Committees on Waste Management.

The Governor's Waste Management Board, in dealing with the issue of providing for effective citizen involvement in local policy decisions on hazardous waste management, feels that one of its most important roles is to insure that correct and proper information is received by local units of government and by the people who live in our State.

Representatives of the League of Municipalities and the Association of County Commissioners met with the Waste Management Board at its March 3 meeting to discuss this matter prior to the Board action calling for establishment of Citizen Involvement Committees (CIC). Both the League and the Association offered their assistance to the Waste Management Board in public information and education efforts, but urged that actions recommended at the local level be on a voluntary basis.

The Waste Management Board feels that waste management facilities have met with much suspicion and resistance from citizens due, at least in part, to poor communications between the facility developer, state regulatory agencies, and local citizens. These same problems may occur with existing facilities if proper education and information is not disseminated to local governing bodies and to citizens. Past experience in other states indicates that early and thorough public participation and education must and should be encouraged.

The local Citizen Involvement Committees on Waste Management could serve as a forum for exchange of information and opinions between State regulatory agencies and involved localities. This could provide State agencies with an added opportunity for understanding and addressing local concerns. This approach should also help avoid misinformation and distrust by educating the citizenry about the rules and regulations that govern the operation of existing facilities and of any new ones that may be proposed. The Waste Management Board recommends that these committees be organized and put into operation prior to the permitting of existing or new facilities so that the community will have good grass roots understanding what is happening within their own areas and why.

The following interest areas should probably be represented on local CIC's: Citizen Organizations, County Government, Municipal Government, Local Industry and Commerce, Local Health and Environmental Agencies or Groups, Local Education and Research Institutions, Local Transportation and Shipping Interests, and Local Agricultural Interests. The Waste Management Board suggests that county commissioners appoint these local CIC's, although larger or smaller geographic areas could be covered by one CIC.

The functions of local CIC's could include: (1) meeting with State officials and private industry to determine the nature and scope of operations of existing facilities and any proposed new facilities that may be under consideration, (2) to participate in planning public meetings, distribution of information, and other such steps that should be taken to fully inform all the people of the community about what currently exists or is being proposed, including the advantages and disadvantages, (3) to take steps in conjunction with State and local government and private industry to insure that response is made to questions and concerns of the citizens of the affected areas insofar as available knowledge and understanding permit, and (4) to recommend to State and local governments and private industry those measures that should or could be taken to make the location of an existing or new facility acceptable to the citizens.

The Waste Management Board also noted that existing community organizations or groups may want to expand their existing role to become a Citizen Involvement Committee on Waste Management.

Southern City

10 NCAC 48A .0101 through .0111 have been adopted as follows:

SECTION .0100 - RULES OF PROCEDURE FOR IMPLEMENTING THE LIMITED PREEMPTION PROCESS

.0101 PURPOSE

The purpose of these rules is to establish procedures to be used by the Governor's Waste Management Board in reviewing petitions for limited preemption of a local ordinance pursuant to the authority set forth in NCGS 130-166.17B and 104E-6.2.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0102 DEFINITIONS

As used in these rules

(a) "Board" shall mean the Governor's Waste Management Board.

(b) "Chairman" shall mean the Chairman of the Governor's Waste Management Board.

(c) "Developer" means, in the case of a proposed facility, a person who has initiated and paid for plans, investigations, interests in land or made other expenditures preliminary to initiating construction of a facility; or, in the case of a facility in the process of construction, the person with overall responsibility for construction of the facility.

(d) "Facility" shall mean any low-level radioactive waste facility as defined in NCGS 104E-5(9b) and any hazardous waste facility as defined in NCGS 130-166.16(5).

(e) "Operator" means the person responsible for the overall operation of a facility.

(f) "Party" means the person who submits a petition and the city or county that adopted the ordinance(s) that is the subject of the petition.

(g) "Person" means an individual, corporation, company, association, partnership, unit of local government, state agency, federal agency, or other legal entity.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0103 STANDING TO PETITION THE BOARD

The developer or operator of a facility that would be prevented from construction or operation by a county, municipal or other local ordinance(s) may petition the Board to review the local ordinance(s) according to these procedures. The facility may be proposed, under construction, or completed at the time of the appeal. In order for a facility to be "proposed," it must be shown that the developer has a real interest in construction or operation of a facility in the jurisdiction to which the ordinance(s) in question applied. Such a real interest shall be shown by:

(a) Possession of printed plans for a facility at a specific site in the jurisdiction, and;

(b) Significant expenditures of money for technical investigation of a site or sites in the jurisdiction, and;

(c) Possession of land, options to purchase land, or some other contractual agreement evidencing an interest in land for a potential site.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0104 GROUNDS FOR PETITION

(a) The grounds for petitioning the Board to review a local ordinance(s) are that a facility would be prevented from construction or operation by the ordinance(s) in question. In order to show that a facility would be prevented from construction or operation, the petitioner must show that:

(1) The facility has been denied, pursuant to the ordinance, any legally required permit, license, certification or other form of permission to initiate or continue construction or operation of the facility; provided, however, that the petitioner must show that he/she has exhausted any appeal procedures provided by the ordinance(s), or that despite his/her good faith efforts to appeal as provided by such ordinance(s), the county has been responsible for such unreasonable delay as to effectively constitute denial, or;

(2) Any legally required permit, license, cetficiation or other form of permission to initiate or continue construction or operation of the facility has been granted with conditions that in effect prevent construction or operation, or;

(3) Any legally required permit, license, certification or other form of permission to initiate or continue construction or operation of the facility has been withheld for an unreasonable time so as to constitute denial, or;

(4) The ordinance(s) in question as written and adopted would necessarily prevent the facility from initiating construction or from operating.

(b) For the purposes of this rule, an ordinance may be considered to prevent construction or operation if compliance with it is so economically burdensome beyond what is necessary to protect the public, health and safety as to make it clearly impracticable for the facility to compete with similar facilities.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0105 NOTICE OF PETITION

A notice of petition shall be in writing, shall describe the petitioner's interest in the matter, and shall set forth clearly the grounds for the petition. The notice of petition shall be mailed to the Governor's Waste Management Board, Department of Human Resources, P.O. Box 2091, Raleigh, NC 27602.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0106 RESPONSE TO NOTICE OF PETITION

(a) A notice of petition to the Board will be acknowledged in writing. A copy of the letter of acknowledgement will also be sent to the Chairman of the Board of Commissioners or the mayor of the city that adopted the ordinance(s) in question.

(b) Along with the acknowledgement of the petition the Board shall request from the petitioner such information and arguments relevant to the petitioner's standing to petition as defined in .0103 of these rules and to the grounds for appeal set forth in .0104 of these rules as it deems necessary to complete the petition. The Board shall specify a date before the public hearing concerning the matter by which the requested information and arguments must be received by the Board. The Board, at its discretion, may refuse the petition as incomplete if it finds that the petitioner has failed to comply substantially with the request for information relating to standing and grounds for the appeal, or to show cause why compliance is unduly burdensome.

(c) The Board may also request from the petitioner or the city or county any information and arguments relevant to the findings set forth in .0109 that the petitioner or the city or county desires the Board to consider in making its recommendations to the Governor. The requested information concerning the findings is not mandatory and no party shall be prejudiced for failing to provide such information.

Both the petitioner and the city or county shall be notified of any request made pursuant to this paragraph.

(d) Nothing in this rule shall prevent the petitioner from modifying or providing additional information and arguments at the hearing or in any other appropriate manner before the Governor's final decision is made.

(e) Upon receiving the information and arguments requested from the petitioner, the Board shall transmit it to the city or county and shall inform the city or county that an answer may be filed at anytime before the final decision of the Governor is made.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0107 PUBLIC HEARING TO BE HELD

(a) When a petition is filed with the Governor's Waste Management Board, the Board shall hold a public hearing to consider the petition. The hearing shall be held in the affected locality in accordance with G.S. 150A, Article 2, within a reasonable time but not more than 45 days time requirement for good cause.

(b) The Board shall publish notice of the hearing twice a week for two successive weeks in a newspaper of general circulation in the county where the proposed site is located. The final notice shall appear at least 15 days but not more than 25 days before the hearing date. The notice shall include:

(1) A reference to the statutory authority under which the petition is filed and the hearing is being held;
(2) The time and place of the public hearing and a statement of the manner in which data, views and arguments may be submitted by any interested person to the Board either at the hearing or at other times;
(3) A summary statement of the subject matter of the hearing.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0108 CONDUCT OF THE HEARING

(a) Any interested person may appear before the Board at the hearing to offer testimony. In addition to testimony before the Board, any interested person may submit written material to the Board for its consideration.

(b) The Board may, in its discretion, place reasonable time limits on the testimony of each interested person who appears before the Board at the hearing.

(c) Members of the Board may ask questions of any person who appears before the Board at the hearing.

(d) The Board shall hold the record open to receive written comments from interested persons for 15 days after termination of the hearing.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0109 THE BOARD's RECOMMENDATIONS TO THE GOVERNOR

(a) No later than 60 days after the hearing, the Board shall present its written recommendations to the Governor to approve or disapprove the facility.

Before recommending that the Governor approve the facility, the Board must make the following findings:

(1) That the proposed facility is needed in order to establish adequate capability for the management of hazardous waste generated in North Carolina and therefore serves the interests of the citizens of the State as a whole;
(2) That all legally required State and federal permits or approvals have been issued by the appropriate State and federal agencies;
(3) That local citizens and elected officials have had adequate opportunity to participate in the siting process;
(4) That the construction and operation of the facility will not pose an unreasonable health or environmental risk to the surrounding locality and that the facility developer or operator has taken or consented to take any reasonable measures to avoid or manage foreseeable risks and to comply to the maximum feasible extent with any applicable ordinance(s).

(b) The Board's recommendation shall include a complete transcript of the hearing, all written material presented to the Board regarding the site location and the four specific findings set forth in (a) above, and any minority positions on the recommendation and the four specific findings.

(c) The Board's decision as to its recommendation shall be made by concurrence of a majority of the Board members. To be eligible to vote, a Board member shall be present at the time the vote is taken.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0110 GOVERNOR'S DECISION

(a) The Governor's decision shall be issued by transmittal to the petitioner and town or county within a reasonable time following receipt of the Board's recommendation.

(b) The Governor's decision shall be in writing and shall identify the material submitted to him by the Board plus any additional materials used in arriving at his decision. The decision shall affirm or disaffirm the findings of the Board and may set forth additional findings. The decision shall state whether the Governor approves or disapproves the establishment or operation of the facility. If the Governor makes the four findings set forth in .0109 (a) of these rules, he shall approve the facility. If the Governor does not make all four findings, he shall disapprove the facility.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

.0111 APPEAL OF THE GOVERNOR'S DECISION

(a) The decision of the Governor shall be final unless a party shall file pursuant to G.S. 7A-29 a written appeal within 30 days of such decision.

(b) The record on appeal shall include all materials and information submitted to or considered by the Governor in accordance with G.S. 130-166.17B (c) and these rules. The scope of judicial review shall be limited to questions of abuse of discretion.

History Note: Statutory Authority G.S. 143B-216.13 (11) & (14);
Eff. January 1, 1983.

10 NCAC 48B .0101 through .0112 have been adopted as follows:

SECTION .0100 - RULES OF PROCEDURE FOR IMPLEMENTING PRIVILEGE LICENSE TAX APPEAL

.0101 PURPOSE

The purpose of this Section is to set forth the rules of practice for appeals to the Governor's Waste Management Board by any person or firm upon which a privilege license tax is levied pursuant to G.S. 160A-211.1 or G.S. 153A-152.1.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0102 DEFINITIONS

As used in these rules

(a) "Hearing" shall mean a contested case hearing as provided in G.S. 150A-23(a).

(b) "Board" shall mean the Governor's Waste Management Board.

(c) "Chairman" shall mean the Chairman of the Governor's Waste Management Board.

(d) "Facility" shall mean any low-level radioactive waste facility as defined in NCGS 104E-5(9b) and any hazardous waste facility as defined in NCGS 130-166.16(5).

(e) "Hearing Panel" shall mean any group of members of the Board appointed by the Chairman and delegated by him the authority to hear an appeal.

(f) "Hearing Officer" shall mean any person to whom the Chairman delegates authority to hear an appeal.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0103 WHO MAY APPEAL

(a) Any person or firm operating a facility taxed pursuant to G.S. 160A-211.1 or G.S. 153A-152.1 may appeal the tax rate to the Board, but shall pay the tax when due, subject to a refund when the appeal is resolved by the Board or in the Courts.

(b) No person shall appeal to the Board pursuant to these procedures unless any appeal procedure provided by local ordinance has been exhausted, or a good faith effort has been made to appeal, but the local government has failed to take timely action such that the operation or construction of the facility may be prohibited.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0104 GROUNDS FOR APPEAL

Grounds for appealing the tax rate shall be that the amount of the tax does not meet the requirements of 160A-211.1(b) in case of a city tax or G.S. 153A-152.1 in the case of a county tax.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0105 NOTICE OF APPEAL

Appeals of a tax rate shall be filed no later than 30 days after the levy of the tax in the case of a facility subject to the tax at the time of the levy, or no later than 30 days after notice that the tax is due in the case of a facility not subject to the tax at the time of the levy. An appeal shall be in writing and shall be mailed to the Governor's Waste Management Board, Department of Human Resources, P.O. Box 2091, Raleigh, NC 27602. The appellant shall cause a copy of the notice of appeal to be served upon the city or town levying the tax in question.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0106 RESPONSE TO NOTICE OF APPEAL

(a) Notice of appeal to the Board will be acknowledged in writing and a form requesting information necessary to perfect the appeal will be mailed to the appellant along with the acknowledgement. A copy of the letter of acknowledgement will also be sent to the Chairman of the Board of Commissioners and the attorney for the county, in the case of an appeal of a county tax, or the Mayor and attorney for the city in the case of an appeal of a city tax.

(b) Unless an extension is requested and granted, the requested information necessary to perfect the appeal must be returned within 30 days of the date the appellant receives the letter of acknowledgement or the appeal will be dismissed. The applicant shall send a copy of the requested information to the city or county attorney at the same time it is returned to the Board.

(c) The acknowledgement will also transmit a date for hearing the appeal to be set by the Chairman. The date of the hearing shall be within 90 days of receipt of the notice of appeal, except that at the discretion of the Chairman, the date may be extended for good cause.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0107 FILING OF AN ANSWER

(a) The city or county that levied the tax being appealed shall file an answer within 20 days from receipt of the completed application, and shall send a copy of the answer to the appellant at the same time it is filed with the Board.

(b) The city or county's failure to an answer shall not constitute waiver of the city or county's rights nor an admission of the appellant's allegations.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0108 DOCUMENTS TO BE SUBMITTED BEFORE HEARING

(a) At least 10 days prior to the date of the hearing, each party to the appeal shall furnish to the Board a copy of all documents to be introduced at the hearing, including maps, pictures, facility descriptions and briefs, and shall also submit the names of any witnesses that they plan to call to testify at the hearing. This requirement may be modified by the Board if it is shown that compliance would cause unduc hardship on one or both of the parties, provided that any such modification and the reasons for it be set out in writing.

(b) In the absence of an agreement to the contrary, a copy of each such document and a list of the names of witnesses shall also be furnished or made available to the opposing party at the same time.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0109 WHO SHALL CONDUCT A HEARING

The Chairman shall determine for each panel whether the matter should be heard by the full Board, a panel of Board members, or a hearing officer, and shall notify the parties of the determination. The Chairman is hereby authorized to appoint Board members to a panel or a hearing officer and to delegate to the panel or hearing officer authority to hear an appeal for the Board.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0110 RULES FOR CONDUCTING THE HEARING

The following rules of the Department of Human Resources for contested cases set forth in 10NCAC .01B.0200 are hereby adopted, except that when used for the purpose of appeals of privilege license taxes under this section, the term "hearing office" shall mean the Office of the Governor's Waste Management Board and the term "Presiding Officer" shall mean the hearing officer or head of a hearing panel who has been appointed by the Chairman.

(a) 10NCAC .01B.0207 Waiver of Hearing
(b) 10NCAC .01B.0208 Intervention
(c) 10NCAC .01B.0209 Written Answer
(d) 10NCAC .01B.0210 Venue
(e) 10NCAC .01B.0211 Discovery
(f) 10NCAC .01B.0212 Pre-Hearing Conference
(g) 10NCAC .01B.0213 Subpoenas
(h) 10NCAC .01B.0214 Consolidation
(i) 10NCAC .01B.0215 Stipulations
(k) 10NCAC .01B.0217 Failure of Party to Attend Hearing
(l) 10NCAC .01B.0218 Oath
(m) 10NCAC .01B.0219 Conduct of Hearing
(n) 10NCAC .01B.0221 Record
(o) 10NCAC .01B.0222 Transcript

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January, 1983

.0111 PROPOSAL FOR DECISION

(a) If the appeal is heard by a hearing officer or a panel composed of less than a majority of the Board members, the decision shall not be made until a proposal for decision is served on the parties, and an opportunity is given to each party to file exceptions and proposed findings of fact and to present oral and written arguments to the Board.

(b) A proposal for decision shall contain proposed findings of fact and proposed conclusions of law, and shall be prepared by a person who conducted the hearing unless he becomes unavailable to the Board. If no such person is available, the proposal may be prepared by one who has read the record, unless demeanor of witnesses is a factor. If demeanor is a factor, the portions of the hearing involving demeanor shall be held again, or the case shall be dismissed without prejudice.

(c) The parties, by written stipulation or at the hearing, may waive compliance with this rule.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

.0112 FINAL DECISION

(a) No final decisions as to whether the amount of the tax meets the requirements of G.S. 160A-211.1(b) or G.S. 153A-152.1 shall be made unless a majority of the Board members is eligible to vote.

(b) To be eligible to vote, a member shall:

(1) be present at the hearing, or, if the appeal was heard by a panel or hearing officer, at the oral presentation by the parties and the hearing officer or hearing panel; and

(2) be present at the time the vote is taken.

(c) If the entire Board hears an appeal, a final decision may be made at the close of the hearing or the Board may defer the decision until some future date to be determined at the discretion of the Board. If the final decision is deferred, the Board may require each party to prepare and submit to it a proposal for decision.

(d) A final decision shall be made by concurrence of a majority of the Board members eligible to vote.

History Note: Statutory Authority G.S. 143B-216.13(12) and (14)
Eff. January 1, 1983

ACKNOWLEDGEMENTS

The Governor's Waste Management Board wishes to express its appreciation to the members of the two Technical Advisory Committees who contributed their time and expertise providing valuable background information to the Technical Committee.

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The Waste Management Board wishes to express its special thanks to individuals who were not members of the Board who volunteered their time to serve as advisory members of the Public Information/Participation Committee. Their efforts were extremely valuable in developing ways to educate and involve citizens in the formulation of an acceptable waste management program for North Carolina.

Jean Vollrath-Vaughn
June Kimmel
Russ Edmonston
Karen Fox

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Department of Administration:	Secretary Jane Patterson
Department of Commerce:	Secretary D. M. Faircloth
Department of Agriculture:	Commissioner James A. Graham
Department of Crime Control and Public Safety:	Secretary Heman Clark
Department of Public Education:	State Superintendent Craig Phillips
Department of Transportation:	Secretary William Roberson, Jr.

North Carolina State University

East Carolina University

University of North Carolina - Chapel Hill

Duke University

Robert Jansen, Senior Policy Advisor, Office of the Governor

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Dr. Don Huisingh

Sam Johnson, Attorney

Jerry Roberts, North Carolina Textile Manufacturers Association

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Conservation Council of North Carolina
Tri-County Alliance
North Carolina CATCH
Anson County CACTUS
Anson County Waste Management Board
League of Women Voters

The Waste Management Board would also like to express their appreciation to the many members of the public and news media who attended meetings and volunteered their opinions regarding matters facing the Board. These expressions of opinion continue to reinforce in the minds of the Waste Management Board members their responsibility to serve the interest of the general public.

A very special thank you to the staff of the Governor's Waste Management Board for their dedication and professionalism in carrying out their assigned tasks on behalf of the Board and in the preparation of this report.

Angie Woodall, Administrative Assistant to the Chairman/Director
Kathy Visocki, Community Relations Coordinator
Doris Wilson, Secretary
Cecil Cross, Summer Intern
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H

GENERAL ASSEMBLY OF NORTH CAROLINA
SESSION 1983

2

HOUSE BILL 738
Committee Substitute Favorable 7/8/83

Short Title: Strict Liab. Hazardous Wastes. (Public)

Sponsors: Representative

Referred to: Water & Air Resources.

April 7, 1983

A BILL TO BE ENTITLED

AN ACT TO PROVIDE FOR STRICT LIABILITY FOR DAMAGES RESULTING FROM HAZARDOUS WASTES IN NORTH CAROLINA.

The General Assembly of North Carolina enacts:

Section 1. Short title. This act may be referred to as the Hazardous Waste Strict Liability Act of 1983.

Sec. 2. Purpose. The purpose of this act is to provide for a workable strict liability system for hazardous wastes in North Carolina as recommended in the 1983 Report of the Governor's Waste Management Board.

Sec. 3. Creation of a strict liability system for hazardous wastes. Chapter 130 of the General Statutes as it appears in Volume 3B of the General Statutes (and the Cumulative Supplement thereto) is amended:

(1) By adding the following at the end of G.S. 130-166.16:

"(23) 'Claimant' means a person damaged by a hazardous waste occurrence.

"(24) 'Hazardous waste occurrence' means any sudden or non-sudden occurrence in which damages result from any quality or characteristic of a solid waste, as defined in G.S. 130-166.16(16), which quality or characteristic causes the waste to be a hazardous waste under this Article and the rules and regulations adopted pursuant to it.

"(25) 'Strictly liable' means liable without regard to the defendant's negligence or fault. The defendant's foreseeability of danger or risk arising from or injury or other consequences caused by a hazardous quality or characteristic of the waste either at or prior to the occurrence shall not be relevant to a determination of liability.

"(26) 'Caused by' means caused in fact. If the evidence offered by any party tends to show that the damage of which the claimant complains was caused by both a hazardous waste occurrence and other causes, liability under G.S. 130-166.21G shall be limited to damages attributable to the hazardous waste occurrence."

(2) By adding a new section G.S. 130-166.21G to read as follows:

"§ 130-166.21G. Hazardous waste liability.--(a) This section applies only to hazardous waste occurrences caused wholly or partially by a defendant's operations or activities occurring after the effective date of this act. For the purpose of this section, waste polychlorinated biphenyls shall be considered a hazardous waste. Furthermore, for the purposes of this section, governmental immunity from strict liability for bodily injury or property damage caused by a hazardous waste occurrence is hereby waived, but only to the extent that damages do not exceed the amount of damages authorized by the North Carolina Tort Claims Act, G.S. 143-291. Any agency of the State of North Carolina that generates, transports, treats, stores, or disposes of hazardous waste is hereby authorized and empowered to procure proper insurance against such liability..

(b)

(1) Subject only to the defenses set forth in subsections (c) and (e) of this section, the generator, transporter, treater, storer or disposer in control of a hazardous waste at the time of a hazardous waste occurrence shall be strictly liable for bodily injury or property damage caused by the occurrence.

(2) Subject only to the defenses set forth in subsections (c) and (e) of this section, the generator of a hazardous waste, whether or not in control of the hazardous waste at the time of an occurrence, shall be strictly liable for bodily injury or property damage caused by hazardous waste occurrences arising from the hazardous waste generated. Under this subdivision, the generator shall be jointly and severally liable together with the transporter or any other person in control of the hazardous waste at the time of the occurrence. For purposes of this subsection, any person that accepts a hazardous waste from a transporter for storage and causes the hazardous waste to be again

transported shall be considered a generator.

(c) There shall be no liability under subsection (b) for a person otherwise liable who can establish by a preponderance of the evidence:

(1) that the claimant had knowledge of the danger and voluntarily and unreasonably encountered that danger; or

(2) that the hazardous waste occurrence was caused solely by any one of the following:

(i) an act of God;

(ii) an act of war or sabotage; or

(iii) an intentional act or omission of a third party (but this defense shall not be available if the act or ommission is that of an employee or agent of the defendant, or if the act or omission occurs in connection with a contractual relationship with the defendant).

(d) Nothing in this section shall bar or otherwise affect the transfer of liability to the Post Closure Liability Fund, under Section 232 of the Federal Comprehensive Environmental Response, Compensation and Liability Act of 1980.

(e)

(1) Subject to the provisions of subsection (i), any defendant who proves that hazardous waste was transferred to and accepted by a hazardous waste facility duly permitted by appropriate regulatory agencies of which he is not the beneficial owner or

operator and that such transfer and acceptance is in compliance with applicable federal and State requirements existing at the time of the transfer and acceptance shall not be liable under subsection (b) for bodily injury or property damage caused by a hazardous waste occurrence after such transfer and acceptance.

(2) When signatures are admitted and established, production of a copy of a hazardous waste manifest entitles a defendant, who according to the manifest generated or transported the waste to a hazardous waste facility, to the defense under this subsection unless the claimant establishes that the waste was not in fact transferred to and accepted by the hazardous waste facility prior to the hazardous waste occurrence.

(3) Each signature on a hazardous waste manifest is presumed to be genuine or authorized unless the party opposing its admission offers prima facie evidence that it is not genuine or authorized.

(f) No punitive or exemplary damages shall be awarded in any action under subsection (b).

(g) Nothing in this section shall deprive a claimant from electing to pursue any other cause of action based upon a hazardous waste occurrence that may exist under statutory or common law, or to deprive a person liable under subsection (b) of any right of contribution or indemnity he may have under law in existence at the time of the occurrence, nor shall anything in this section restrict any right which any person (or class of persons) may have under any statute or common law to seek enforcement of any standard or requirement or to seek the imposition by any State and federal authorities of civil and criminal sanctions. If the principal action was decided on the basis of strict liability, the principles of strict liability shall also apply in any action for contribution or indemnification arising out of the same hazardous waste occurrence.

(h) If any provision of this section or its application to any person or circumstances is held invalid by any court of competent jurisdiction, the invalidity will not affect other provisions or applications that can be given effect without the invalid provision or application; and to this end the provisions of this act are severable."

Sec. 4. G.S. 1-52(16) is amended by changing the second sentence to read as follows:

"Provided that no cause of action shall accrue more than 10 years from the last act or omission of the defendant giving rise to the cause of action; except that for any cause of action arising out of a hazardous waste occurrence as defined in G.S. 130-166.16(24) no cause of action based on strict liability shall accrue more than 30 years from the last act or omission of the defendant giving rise to the cause of action."

Sec. 5. This act shall become effective October 1, 1983.

CALIFORNIA

y References
yds ⟸67.
S Records §§ 35, 41.

55. Alcohol and drug abuse records; communicable disease carriers

Nothing in this chapter shall require a health care provider to permit inspection or provide
of alcohol and drug abuse records where, or in a manner, prohibited by Section 408 of the
d Drug Abuse Office and Treatment Act of 1972 (Public Law 92-255) or Section 338 of the
d Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment, and Rehabilitation Act
70 (Public Law 91-616), or by regulations adopted pursuant thereto. Alcohol and drug abuse
is subject to such federal laws shall also be subject to the provisions of this chapter, to the extent
such federal laws do not prohibit disclosure of the records. All other alcohol and drug abuse
ls shall be fully subject to the provisions of this chapter.

Nothing in this chapter shall require a health care provider to permit inspection or provide
of records or portions of records where or in a manner prohibited by existing law respecting the
lentiality of information regarding communicable disease carriers.

d by Stats.1982, c. 15, p. —, § 1.)

y References
ords ⟸54.
S. Records § 36.

56. Summary of record; preparation; time of delivery; contents; fee

A health care provider may prepare a summary of the record, according to the requirements of
ection, for inspection and copying by a patient. If the health care provider chooses to prepare
a summary of the record rather than allowing access to the entire record, he or she shall make
a summary of the record available to the patient within 10 working days from the date of the
nt's request. However, if more time is needed because the record is of extraordinary length or
se the patient was discharged from a licensed health facility within the last 10 days, the health
provider shall notify the patient of this fact and the date that the summary will be completed,
n no case shall more than 30 days elapse between the request by the patient and the delivery of
immary. In preparing the summary of the record the health care provider shall not be obligated
lude information which is not contained in the original record.

A health care provider may confer with the patient in an attempt to clarify the patient's
se and goal in obtaining his or her record. If as a consequence the patient requests information
only certain injuries, illnesses, or episodes, nothing in this subdivision shall require the provider
pare the summary required by this subdivision for other than the injuries, illnesses, or episodes
quested by the patient. The summary shall contain for each injury, illness, or episode any
nation included in the record relative to the following:

Chief complaint or complaints including pertinent history.

Findings from consultations and referrals to other health care providers.

Diagnosis, where determined.

Treatment plan and regimen including medications prescribed.

Progress of the treatment.

Prognosis including significant continuing problems or conditions.

Pertinent reports of diagnostic procedures and tests and all discharge summaries.

Objective findings from the most recent physical examination, such as blood pressure, weight,
ctual values from routine laboratory tests.

Nothing in this section is to be construed as requiring any medical records to be written or
tained in any manner not otherwise required by law.

The summary shall contain a list of all current medications prescribed, including dosage, and any
ivities or allergies to medications recorded by the provider.

The provisions of subdivision (c) of Section 25252 shall be applicable whether or not the health
provider elects to prepare a summary of the record.

(f) The health care provider may charge no more than a reasonable fee based on actual time and cost for the preparation of the summary. The cost shall be based on a computation of the actual time spent preparing the summary for availability to the patient or the patient's representative. It is the intent of the Legislature that summaries of the records be made available at the lowest possible cost to the patient.

(Added by Stats.1982, c. 15, p. —, § 1.)

Library References

Records ⇐54.
C.J.S. Records § 36.

§ 25257. Law governing; Confidentiality of Medical Information Act; Insurance Information and Privacy Protection Act; Information Practices Act of 1977

Except as otherwise provided by law, nothing in this chapter shall be construed to grant greater access to individual patient records by any person, firm, association, organization, partnership, business trust, company, corporation, or municipal or other public corporation, or government officer or agency. Therefore, the provisions of this chapter do not do any of the following:

(a) Relieve employers of the requirements of the Confidentiality of Medical Information Act (Part 2.6 (commencing with Section 56) of Division 1 of the Civil Code).

(b) Relieve any person subject to the provisions of the Insurance Information and Privacy Protection Act (Article 6.6 (commencing with Section 791) of Chapter 1 of Part 2 of Division 1 of the Insurance Code) from the requirements of that act.

(c) Relieve government agencies of the requirements of the Information Practices Act of 1977 (Title 1.8 (commencing with Section 1798) of Part 4 of Division 3 of the Civil Code).

(Added by Stats.1982, c. 15, p. —, § 1.)

Library References

Records ⇐54.
C.J.S. Records § 36.

§ 25258. Law governing; Information Practices Act of 1977

The provisions of the Information Practices Act of 1977 (Title 1.8 (commencing with Section 1798) of Part 4 of Division 3 of the Civil Code) shall prevail over the provisions of this chapter with respect to records maintained by a state agency.

(Added by Stats.1982, c. 15, p. —, § 1.)

CHAPTER 6.8. HAZARDOUS SUBSTANCE ACCOUNT

Article		Section
1.	Short Title and Legislative Intent	25300
2.	Definitions	25310
3.	Hazardous Substance Account	25330
4.	Tax	25340
5.	Uses of the State Account	25350
6.	Recovery Actions	25360
~~7.~~	~~Compensation~~	~~25370~~
8.	Miscellaneous	25395

Chapter 6.8 was added by Stats.1981, c. 756, p. —, § 2, urgency eff. Sept. 25, 1981.

Repeal

Chapter 6.8, added by Stats.1981, c. 756, p. —, § 2, is repealed July 1, 1991, under the terms of § 25395.

Asterisks * * * indicate deletions by amendment

ARTICLE 1. SHORT TITLE AND LEGISLATIVE INTENT

Sec.
25300. Short title.
25301. Legislative intent.

Article 1 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

Cross References

Hazardous substance as governed by definitions contained in this chapter, see Revenue and Taxation Code § 43002.

Law Review Commentaries

Hazardous substance account, hazardous Substance Tax Law. (1982) 13 Pacific L.J. 699.

§ 25300. Short title

This chapter shall be known and may be cited as the Carpenter-Presley-Tanner Hazardous Substance Account Act.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25301. Legislative intent

It is the intent of the Legislature to:

(a) Establish a program to provide for response authority for releases of hazardous substances, including spills and hazardous waste disposal sites that pose a threat to the public health or the environment.

(b) Compensate persons, under certain circumstances, for out-of-pocket medical expenses and lost wages or business income resulting from injuries proximately caused by exposure to release of hazardous substances.

(c) Make available adequate funds in order to permit the State of California to assure payment of its 10 percent share of the costs mandated pursuant to Section 104(c)(3) of the federal act (42 U.S.C. 9604(c)(3)).

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References

Health and Environment ⇐25.5(2).

C.J.S. Health and Environment §§ 61 et seq., 91 et seq., 106 et seq., 115 et seq., 125 et seq., 133 et seq.

ARTICLE 2. DEFINITIONS

Sec.
25310. Application; definitions in federal act.
25312. Department.
25313. Director.
25315. Federal act.
25316. Hazardous substance; inclusions.
25317. Hazardous substance; exclusions.
25320. Release; inclusions.
25321. Release; exclusions.
25322. Remedy or remedial action.
25323. Remove or removal.
25324. State account.
25325. Federally permitted release.
25326. Release authorized or permitted pursuant to state law.

Article 2 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

Underline indicates changes or additions by amendment

Cross References

Hazardous substance tax, governed by definitions contained in this chapter, see Revenue and Taxation Code § 43002.

§ 25310. Application; definitions in federal act

The definitions set forth in this article shall govern the interpretation of this chapter. Unless the context requires otherwise and except as provided in this article, the definitions contained in Section 101 of the federal act (42 U.S.C. 9601) shall apply to the terms used in this chapter.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25312. Department

"Department" means the State Department of Health Services.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25313. Director

"Director" means the Director of Health Services.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25315. Federal act

"Federal act" means the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, P.L. 96–510 (42 U.S.C. 9601, et seq.).

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25316. Hazardous substance; inclusions

"Hazardous substance" means:

(a) Any substance designated pursuant to Section 1321(b)(2)(A) of Title 33 of the United States Code.

(b) Any element, compound, mixture, solution, or substance designated pursuant to Section 102 of the federal act (42 U.S.C. 9602).

(c) Any hazardous waste having the characteristics identified under or listed pursuant to Section 6921 of Title 42 of the United States Code, but not including any waste the regulation of which under the Solid Waste Disposal Act has been suspended by act of Congress.

(d) Any toxic pollutant listed under Section 1317(a) of Title 33 of the United States Code.

(e) Any hazardous air pollutant listed under Section 7412 of Title 42 of the United States Code.

(f) Any imminently hazardous chemical substance or mixture with respect to which the Administrator of the United States Environment Protection Agency has taken action pursuant to Section 2606 of Title 15 of the United States Code.

(g) Any hazardous waste or extremely hazardous waste as defined by Sections 25117 and 25115, respectively, unless expressly excluded.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25317. Hazardous substance; exclusions

"Hazardous substance" does not include:

(a) Petroleum, including crude oil or any fraction thereof which is not otherwise specifically listed or designated as a hazardous substance in subdivisions (a) to (f), inclusive, of Section 25316, and natural gas, natural gas liquids, liquefied natural gas, or synthetic gas usable for fuel (or mixtures of natural gas and such synthetic gas), or the ash produced by a resource recovery facility utilizing a municipal solid waste stream.

(b) Nontoxic, nonflammable, noncorrosive stormwater run-off drained from underground vaults, chambers, or manholes into gutters or stormsewers.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25320. Release; inclusions

"Release" means any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25321. Release; exclusions

"Release" does not include any of the following:

(a) Any release which results in exposure to persons solely within a workplace, with respect to a claim such exposed persons may assert against their employer.

(b) Emissions from the engine exhaust of a motor vehicle, rolling stock, aircraft, vessel, or pipeline pumping station engine.

(c) Release of source, byproduct, or special nuclear material from a nuclear incident, as those terms are defined in the Atomic Energy Act of 1954 (42 U.S.C. 2011, et seq.), if such release is subject to requirements with respect to financial protection established by the Nuclear Regulatory Commission under Section 2210 of Title 42 of the United States Code or, for the purposes of Section 104 of the federal act (42 U.S.C. 9604) or any other response action, any release of source byproduct, or special nuclear material from any processing site designated under Section 7912(a)(1) or 7942(a) of Title 42 of the United States Code, which sections are a part of the Uranium Mill Tailings Radiation Control Act of 1978.

(d) The normal application of fertilizer, plant growth regulants, and pesticides.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25322. Remedy or remedial action

"Remedy" or "remedial action" includes those actions which are consistent with a permanent remedy, that are taken instead of, or in addition to, removal actions in the event of a release or threatened release of a hazardous substance into the environment, as further defined by Section 101(24) of the federal act (42 U.S.C. 9601(24)), except that any reference in Section 101(24) of the federal act (42 U.S.C. 9601(24)) to the President, relating to determinations regarding the relocation of residents, businesses, and community facilities shall, for the purposes of this chapter, be deemed to be a reference to the Governor and any other reference in that section to the President shall, for the purposes of this chapter, be deemed a reference to the Governor, or the director, if designated by the Governor.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25323. Remove or removal

"Remove" or "removal" includes the cleanup or removal of released hazardous substances from the environment or the taking of such other actions as may be necessary to prevent, minimize, or mitigate damage which may otherwise result from a release or threatened release, as further defined by Section 101(23) of the federal act (42 U.S.C. 9601(23)).

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25324. State account

"State account" means the Hazardous Substance Account established pursuant to Section 25330.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25325. Federally permitted release

"Federally permitted release" shall have the same meaning as that term is defined in Section 101(10) of the federal act (42 U.S.C. 9601(10)).

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25326. Release authorized or permitted pursuant to state law

"A release authorized or permitted pursuant to state law" means any release into the environment which is authorized by statute, ordinance, regulation, or rule of any state, regional, or local agency or government or by any specific permit, license, or similar authorization from any such agency, including one of the foregoing which recognizes a standard industry practice, including variances obtained from any such agency which allow operations for facilities during a period of time when releases from such facilities do not conform with relevant statutes, ordinances, regulations, or rules. The terms includes a federally permitted release, as defined by Section 25325, and releases which are in accordance with any court order or consent decree.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

ARTICLE 3. HAZARDOUS SUBSTANCE ACCOUNT

Sec.

25330. Establishment; administration; deposits.
25331. Actions in name of account.
25332. Appropriation; loan to account.
25333. Appropriation; loan to account; repayment of principal plus interest.

Article 3 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

§ 25330. Establishment; administration; deposits

There is in the General Fund a Hazardous Substance Account which shall be administered by the director. In addition to any other moneys appropriated by the Legislature to the account, the following amounts shall be deposited in the account:

(a) All moneys recovered by the state account pursuant to Section 25360.

(b) Any moneys recovered by the State Board of Control pursuant to Section 25380.

(c) Any fines or civil penalties collected pursuant to Section 25343.

(d) The taxes collected pursuant to Article 4 (commencing with Section 25340).

(e) Any interest earned upon money deposited in the state account.

(f) Any moneys received from the federal government pursuant to the federal act.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References

States ⟹127.
C.J.S. States § 228.

§ 25331. Actions in name of account

The state account may sue and be sued in its own name.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References

States ⟹190; 191(1).
C.J.S. States §§ 297, 298, 314.

§ 25332. Appropriation; loan to account

The sum of two million dollars ($2,000,000) is hereby appropriated from the contingency reserve for economic uncertainties, which reserve is continued in existence by Section 12.30 of the Budget Act of 1981 (Ch. 99, Stats. 1981), as a loan to the state account for expenditures therefrom authorized by this chapter.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25333. Appropriation; loan to account; repayment of principal plus interest

The principal amount of the loan specified in Section 25332 shall be repaid in annual amounts of not less than four hundred thousand dollars ($400,000), plus interest, with the total loan to be repaid on or before January 1, 1986. The loan shall be repaid with interest charged at the rate for the Pooled

Money Investment Account in the General Fund for the 1981 82 fiscal year. The Director of Finance may prescribe additional terms and conditions for this loan.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Maximum amount of tax to be collected after deduction of payments specified in this section, see § 25347.

ARTICLE 4. TAX

Sec.	
25340.	Board.
25341.	Dispose or disposal.
25342.	Annual disposal report; submission; contents.
25342.1.	Total amount of hazardous waste [New].
25343.	Annual disposal report; failure to file; civil and criminal penalties.
25344.	Report to governor; amount of wastes; amount of taxes collected; amount of taxes subject to rebate.
25345.	Imposition of tax; formula; assessment notice; payment; subsequent removal of waste to another place; exception.
25345.3.	State or local agencies; removal or release of hazardous waste [New].
25345.5.	Exemptions from recordkeeping requirements and tax; voluntary hazardous waste collection program, definitions; regulation of hazardous waste collections [New].
25346.	Deposit in state account.
25347.	Maximum tax.
25347.2.	State account rebate; computation; credit or payment; appropriation [New].
25348.	Enforcement; rules and regulations.

Article 4 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

Cross References

Collections pursuant to this article to be deposited in hazardous substance account, see § 25330.

§ 25340. Board

"Board," as used in this article, means the State Board of Equalization.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25341. Dispose or disposal

(a) "Dispose" or "disposal," as used in this article, means to abandon, deposit, inter, or otherwise discard waste into or on land as a final action after use has been achieved and no intended beneficial use or reuse can be demonstrated by any person.

(b) "Dispose" or "disposal" shall not include any injection of fluids or other materials for the purpose of stimulating or treating wells for the production of crude oil, natural gas, water or geothermal energy or for the purpose of secondary, tertiary, or other enhanced recovery of crude oil or natural gas, or fluids or other materials which are brought to the surface in conjunction with the production of geothermal resources, crude oil or natural gas and which are reinjected.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25342. Annual disposal report; submission; contents

On or before March 1, 1982, and March 1 of each year thereafter, every person who submitted for disposal offsite, or who disposed of onsite, hazardous waste or extremely hazardous waste in the state during the preceding calendar year shall report to the board the following information:

(a) The total amount of hazardous or extremely hazardous waste, as defined by Sections 25117 and 25115, respectively, the federal regulation of which has been suspended under the Solid Waste Disposal Act (42 U.S.C. 6901) by act of Congress, which that person has disposed of, or submitted for disposal, in the state, plus the total amount of hazardous or extremely hazardous waste and material,

* * * which that person has transferred to a surface impoundment in the state for purposes of reducing the water content of the waste and material by evaporation, exclusive of that waste specified in subdivision (d).

(b) The total amount of hazardous waste disposed of, or submitted for disposal, in the state by that person, exclusive of that waste specified in subdivisions (a), (c), and (d).

(c) The total amount of extremely hazardous waste disposed of, or submitted for disposal, in the state by that person, exclusive of that waste specified in subdivisions (a), (b), and (d).

(d) The total amount of hazardous or extremely hazardous waste that person has disposed of, or submitted for disposal, in the state from the extraction, beneficiation, and processing of ores and minerals, including phosphate rock and overburden from mining of uranium ore.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981. Amended by Stats.1982, c. 496, p. ——, § 13, urgency, eff. July 12, 1982; Stats.1982, c. 1244, p. ——, § 4, urgency, eff. Sept. 22, 1982.)

1982 Legislation.

Legislative intent concerning Stats.1982, c. 1244, see note under § 25174.

Cross References

Effect of amendment of section by two or more acts at the same session of the legislature, see Government Code § 9605.

Imposition of tax, formula, see § 25345.

Inapplicability to local health officers or county agricultural commissioners conducting voluntary hazardous waste collection programs, see § 25345.5.

Report to governor certifying amount of wastes specified pursuant to provisions of this article, see § 25344.

Library References

Health and Environment ⟹25.5(5).

C.J.S. Health and Environment §§ 91 et seq., 106 et seq., 131.

§ 25342.1. Total amount of hazardous waste

The total amount of hazardous waste reported pursuant to Section 25342 shall be the total weight, measured in tons, of all components of the waste in the form in which the waste existed at the time of disposal, submission for disposal, or transferral to a surface impoundment, as specified in subdivision (a) of Section 25342.

(Added by Stats.1982, c. 1244, p. ——, § 5, urgency, eff. Sept. 22, 1982.)

1982 Legislation.

Legislative intent concerning Stats.1982, c. 1244, see note under § 25174.

Library References

Health and Environment ⟹25.5(5).

C.J.S. Health and Environment §§ 91 et seq., 106 et seq., 131.

§ 25343. Annual disposal report; failure to file; civil and criminal penalties

Any person who fails to file the report required by Section 25342 shall be liable for a civil penalty not to exceed five hundred dollars ($500) for each day the violation continues. In addition, any person who knowingly fails to file such report shall, upon conviction, be punished by a fine of not more than twenty-five thousand dollars ($25,000) for each day the violation continues, or by imprisonment in the county jail for a period not to exceed one year, or both.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References

Health and Environment ⟹38.

C.J.S. Health and Environment §§ 49, 50, 134, 139, 151 to 154, 156.

§ 25344. Report to governor; amount of wastes; amount of taxes to be collected; amount of taxes subject to rebate

On April 15th of each year, the board shall certify to the Governor:

(a) The total amount of waste and material specified by all persons pursuant to subdivision (a) of Section 25342.

(b) The total amount of waste specified by all persons pursuant to subdivision (b) of Section 25342.

(c) The total amount of waste specified by all persons pursuant to subdivision (c) of Section 25342.

(d) The total amount of waste specified by all persons pursuant to subdivision (d) of Section 25342.

(e) The total amount of taxes to be collected pursuant to this chapter.

(f) The total amount of taxes in the state account subject to the rebate specified in Section 25347.2.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981. Amended by Stats.1982, c. 49, § 14, urgency, eff. July 12, 1982; Stats.1982, c. 1244, p. ——, § 6, urgency, eff. Sept. 22, 1982.)

1982 Legislation.

Legislative intent concerning Stats.1982, c. 1244; see note under § 25174.

Cross References

Effect of amendment of section by two or more acts at the same session of the legislature, see Government Code § 9605.

Library References

Health and Environment ⟹25.5(9).
C.J.S. Health and Environment §§ 65, 66, 103, 107, 140 et seq.

§ 25345. Imposition of tax; formula; assessment notice; payment; subsequent removal of waste to another place; exception

(a) There is hereby imposed a tax, payable annually, and to be collected by the board, upon each ton or fraction thereof, of waste and material specified in Section 25342, according to the formula specified herein. The board shall set the base tax rate upon the waste and material specified in Section 25342 in accordance with the following formula:

$$\text{Base tax rate} = \frac{(\$10{,}000{,}000 - M)}{(0.15W_1 + W_2 + 2W_3 + 0.01W_4)}$$

where:

(1) "M" is the existing unobligated balance in the fund * * * (M shall equal zero (0) for * * * June 30, 1982). As used in this article, "unobligated balance" means all unencumbered funds in the account as of June 30, as estimated on March 1 of each year, less:

(A) Any funds in the reserve account for emergencies established by Section 25354.

(B) Any remaining principal of the loan authorized by Section 25332.

(C) Any interest due on any remaining principal of the loan authorized by Section 25332.

(D) Any funds paid as taxes for the following fiscal year.

(E) Any funds received from the federal government pursuant to the federal act.

The unobligated balance shall not be less than zero (0).

(F) Any funds in the state account subject to the rebate specified in Section 25347.2.

(2) W_1 is the total amount in tons of waste and material specified in subdivision (a) of Section 25344.

(3) W_2 is the total amount in tons of waste specified in subdivision (b) of Section 25344.

(4) W_3 is the total amount in tons of waste specified in subdivision (c) of Section 25344.

(5) W_4 is the total amount in tons of waste specified in subdivision (d) of Section 25344.

(b) On or before May 1 of each year, using the formula specified in subdivision (a), the board shall determine the base tax rate for the current year.

(c) The tax imposed upon each person subject to Section 25342 shall be assessed according to the following formula:

$$\text{Tax} = (\text{base tax rate}) \times (0.15\,P_1 + P_2 + 2P_3 + 0.01P_4)$$

where:

(1) "Base tax rate" equals the figure determined pursuant to subdivision (a).

(2) P_1 is the amount in tons of waste and material specified by the person pursuant to subdivision (a) of Section 25342.

(3) P_2 is the amount in tons of waste specified by the person pursuant to subdivision (b) of Section 25342.

(4) P_3 is the total amount in tons of waste specified by the person pursuant to subdivision (c) of Section 25342.

(5) P_4 is the total amount in tons of waste specified by the person pursuant to subdivision (d) of Section 25342.

(d) On or before May 1 of each year, the board shall send to each person subject to Section 25342 a notice of assessment * * *.

(e) On or before July 1 of each year, each person subject to Section 25342 shall pay to the board the amount of the assessment as stated in the notice. The tax under this chapter shall not be imposed again on hazardous waste or extremely hazardous waste if the waste, or any portion thereof, is removed subsequently to another place for disposal * * * and if the person has paid the amount of assessment for the waste in accordance with this section before the removal thereof.

(f) Notwithstanding any other provision of this section, hazardous wastes disposed of into an injection well during the 1981 calendar year shall be subject to a tax rate that does not exceed 26.4 percent of the base tax rate, as determined in subdivision (a), per ton or fraction thereof. For purposes of this section, the following definitions apply:

(1) "Injection well" means a bored, drilled or driven shaft, whose depth is greater than the largest surface dimension, and which is used to inject hazardous waste into an underground formation. Injection well does not mean any well located in Class I or a Class II-1 disposal site, as defined in Sections 2510 and 2511 of Title 23 of the California Administrative Code, on the effective date of this section.

(2) "Formation" means a body of rock characterized by a degree of lithologic homogenity which is prevailingly, but not necessarily, tabular and is mapable on the earth's surface or traceable in the subsurface.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981. Amended by Stats.1982, c. 327, p. ——, § 118, urgency, eff. June 30, 1982; Stats.1982, c. 496, p. ——, § 15, urgency, eff. July 12, 1982; Stats.1982, c. 1205, p. ——, § 1; Stats.1982, c. 1244, p. ——, § 7, urgency, eff. Sept. 22, 1982.)

Repeal

Tax imposed under § 25345, added by Stats.1981, c. 756, p. ——, § 2, is repealed July 1, 1991, under the terms of § 25395.

1982 Legislation.

Legislative intent concerning Stats.1982, c. 1244, see note under § 25174.

Cross References

Authority to collect tax imposed pursuant to this section, see Revenue and Taxation Code § 43052.

Effect of amendment of section by two or more acts at the same session of the legislature, see Government Code § 9605.

Library References

Taxation ⚷1231.
C.J.S. Licenses §§ 26 to 31.

§ 25345.3. State or local agencies; removal or release of hazardous waste

(a) Any state or local agency, or its contractor, which removes or remedies a release, by another person, of hazardous waste or extremely hazardous waste, shall not be subject to the requirements of Sections 25342 and 25345. This subdivision shall be applicable to all acts performed by a state or local agency, or its contractor, on or after September 25, 1981.

(b) The person responsible for a release of hazardous or extremely hazardous waste, which has been removed or remedied by a state or local agency, or its contractor, shall submit to the board the information required by Section 25342 for the hazardous or extremely hazardous waste which was released and shall also pay the tax provided in Section 25345.

(Added by Stats.1982, c. 496, p. ——, § 16, urgency, eff. July 12, 1982.)

§ 25345.5. Exemptions from recordkeeping requirements and tax; voluntary hazardous waste collection program, definition; regulation of hazardous waste collections

Neither the recordkeeping requirements of Section 25342 nor the tax provided in Section 25345 shall apply to local health officers or county agricultural commissioners if the local health officers or county agricultural commissioners are conducting voluntary hazardous waste collection programs and if the total quantities collected are limited to 1,000 kg. or less per month from all sources; provided, however, that they comply with all requirements of Chapter 6.5 (commencing with Section 25100) and regulations adopted pursuant thereto. For the purposes of this section, "voluntary hazardous waste collection program" means a program in which small quantities of hazardous wastes are received from nonindustrial sources, stored, and ultimately transferred to a licensed hazardous waste disposal site. Any hazardous waste collections in excess of 1,000 kg. per month shall be subject to all requirements of this chapter and Chapter 6.5 (commencing with Section 25100) and regulations adopted pursuant thereto, including, but not limited to, those providing for payment of taxes and recordkeeping.

(Added by Stats.1982, c. 1205, p. ——, § 2.)

Library References
Health and Environment ⟹25.5(5).
C.J.S. Health and Environment §§ 91 et seq., 106 et seq., 131.

§ 25346. Deposit in state account

The taxes collected pursuant to this article shall be deposited in the state account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References
Taxation ⟹1344.
C.J.S. Licenses § 56.

§ 25347. Maximum tax

* * * (a) In no event shall the total taxes collected in any one fiscal year pursuant to this chapter exceed * * * ten million dollars ($10,000,000), less the * * * unobligated balance in the state account * * * as defined in paragraph (1) of subdivision (a) of Section 25345.

(b) This section shall not be construed to prevent the board from collecting any taxes which are not paid when due, or from issuing any determination or redetermination pursuant to Sections 43201 and 43304 of the Revenue and Taxation Code. Any such determination of taxes shall be at the tax rates established pursuant to Section 25345 of this code for the period for which the determination is issued.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981. Amended by Stats.1982, c. 327, p. ——, § 119, urgency, eff. June 30, 1982; Stats.1982, c. 496, p. ——, § 17, urgency, eff. July 12, 1982; Stats.1982, c. 1244, p. ——, § 8, urgency, eff. Sept. 22, 1982.)

1982 Legislation.

Legislative intent concerning Stats.1982, c. 1244, see note under § 25174.

Cross References

Effect of amendment of section by two or more acts at the same session of the legislature, see Government Code § 9605.

§ 25347.2. State account rebate; computation; credit or payment; appropriation

(a) "State account rebate" means the total taxes, interest and penalties for any preceding calendar year which have been collected during the previous 12-month period, pursuant to Section 25345 and Part 22 (commencing with Section 43001) of Division 2 of the Revenue and Taxation Code, less the total of:

(1) Ten million dollars ($10,000,000), or the amount appropriated for the current fiscal year by the Legislature for the purposes of this chapter, whichever is the lesser amount.

(2) The total amount of refunds of tax, interest and penalties paid during the previous 12-month period.

(3) The total amount of claims for refund filed and pending, to the extent that these claims relate to tax, interest and penalties collected during the previous 12-month period.

(b) "Previous 12-month period" means the period from March 1 of the preceding year to March 1 of the current year.

(c) On or before June 1 of each year the State Board of Equalization shall compute the amount of the state account rebate for the previous 12-month period. The state account rebate shall be credited or paid to each person who paid taxes for the preceding calendar year pursuant to Section 25345 and Part 22 (commencing with Section 43001) of Division 2 of the Revenue and Taxation Code on a pro rata basis in proportion to the amount of taxes, interest and penalties paid by that person for the preceding year.

(d) The amount of any credit or payment for the state account rebate shall be accompanied by a brief explanation or statement of account and may be reflected in the notice of assessment for the current year.

(e) There is hereby continuously appropriated from the Hazardous Substance Account in the General Fund, to the State Board of Equalization, sufficient funds to pay the state account rebates authorized by this section.

(Added by Stats.1982, c. 1244, p. ——, § 9, urgency, eff. Sept. 22, 1982.)

1982 Legislation

Legislative intent concerning Stats.1982, c. 1244, see note under § 25174.

Library References

Health and Environment 🔑25.5(5).
C.J.S. Health and Environment §§ 91 et seq., 106 et seq., 131.

§ 25348. Enforcement; rules and regulations

The board shall enforce the provisions of this article and may prescribe, adopt, and enforce rules and regulations relating to the administration and enforcement of this article.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

ARTICLE 5. USES OF THE STATE ACCOUNT

Sec.

25350. Response actions qualifying for appropriation and expenditure; duplication of federal response action prohibited.
25351. Administrative costs and expenses; state share mandated by federal act; equipment; removal costs; studies; use of federal funds; rules and regulations.
25352. Appropriation on specific site basis; restoration of natural resources; assessment of long-term and short-term injury; unavoidable injury excluded; recovery of expenditures.
25353. Remedial action where governmental entity liable for costs; occurrences prior to effective date of this chapter.
25354. Reserve account for emergencies; continuous appropriation; uses; contracts; reports; maximum deposit and appropriation; reversion and deposit of unencumbered balance.
25355. Sites requiring remedial action; coordination of state response actions; responsible party; when director to initiate removal or remedial action.
25356. Sites for remedial action; criteria for selection and priority ranking; publication and revision; expenditure of funds [New].
25356.5. Report to Legislative; sites upon which funds are proposed to be expended; expenditure of funds [New].
25357. Expenditures exceeding amount in state account.
25358. Federal funds; cooperative agreements.

Article 5 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

§ 25350. Response actions qualifying for appropriation and expenditure; duplication of federal response action prohibited

For response actions taken pursuant to the federal act, only those costs for actions which are consistent with the priorities, guidelines, criteria, and regulations contained in the national contingency plan, as revised and republished pursuant to Section 105 of the federal act (42 U.S.C. 9605), shall qualify for appropriation by the Legislature and expenditure by the director pursuant to Sections 25351, 25352, and 25354. For response actions not taken pursuant to the federal act or for response actions taken which are not specifically addressed by the priorities, guidelines, criteria, and regulations contained in the national contingency plan, as revised and republished, the costs thereof shall also qualify for appropriation by the Legislature and expenditure by the department pursuant to Sections 25351, 25352, and 25354 provided they are, to the maximum extent possible, consistent with the priorities, guidelines, criteria, and regulations contained in the national contingency plan for similar releases, situations, or events. No response actions taken pursuant to this chapter by the department or regional or local agencies shall duplicate federal response actions.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25351. Administrative costs and expenses; state share mandated by federal act; equipment; removal costs; studies; use of federal funds; rules and regulations

Consistent with the requirements of Section 114(c) of the federal act (42 U.S.C. 9614(c)), moneys in the state account may be expended by the director, upon appropriation by the Legislature, for the following purposes:

(a) The costs and expenses reasonably necessary for, and incidental to, the administration of this apter by the department.

(b) The state share mandated pursuant to Section 104(c)(3) of the federal act (42 U.S.C. 9604(c)(3)).

(c) The purchase by the state, or by any local agency with the prior approval of the director, of zardous substance response equipment and other preparations for response to a release of hazardous bstances. However, all equipment shall be purchased in a cost-effective manner after consideration the adequacy of existing equipment owned by the state or the local agency, and the availability of uipment owned by private contractors.

(d) For reimbursement of all costs of removal and remedial action incurred by the state, or by any al agency with the approval of the director, in response to a release or threatened release of a zardous substance to the extent the costs are not reimbursed by the federal act.

(e) All costs incurred in cooperation with the Agency for Toxic Substances and Disease Registry tablished pursuant to Section 104(i) of the federal act (42 U.S.C. 9604(i)) and all costs of health fects studies undertaken regarding specific sites or specific substances at specific sites. Funds propriated for this purpose shall not exceed five hundred thousand dollars ($500,000) in any single cal year. However, such actions shall not duplicate reasonably available federal actions and studies.

(f) All federal funds deposited in the state account shall be expended for the purposes for which ey were provided to the state.

(g) The department shall adopt any regulations necessary to carry out its responsibilities pursuant this chapter, including, but not limited to, regulations governing the expenditure of, and accounting ocedures for, moneys allocated to state, regional, and local agencies pursuant to this chapter.

dded by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

brary References
States ⟜123.
C.J.S. States § 226.

25352. Appropriation on specific site basis; restoration of natural resources; assessment of long-term and short-term injury; unavoidable injury excluded; recovery of expenditures

Money deposited in the state account may also be appropriated by the Legislature to the partment on a specific site basis for the following purposes:

(a) For all costs incurred in restoring, rehabilitating, replacing, or acquiring the equivalent of, any atural resource injured, degraded, destroyed, or lost as a result of any release of a hazardous bstance, to the extent the costs are not reimbursed pursuant to the federal act and taking into count processes of natural rehabilitation, restoration, and replacement.

(b) For all costs incurred in assessing short-term and long-term injury to, degradation or destruction of, or any loss of any natural resource resulting from a release of a hazardous substance, to the tent that the costs are not reimbursed pursuant to the federal act. No costs may be incurred for ay release of a hazardous substance from any facility or project pursuant to subdivisions (a) or (b) for jury, degradation, destruction, or loss of any natural resource where such injury, degradation, struction, or loss was specifically identified as an irreversible and irretrievable commitment of atural resources in an environmental impact statement prepared under the authority of the National vironmental Policy Act (42 U.S.C. 4341, et seq.), or was identified as a significant environmental ffect to such natural resources which cannot be avoided in an environmental impact report prepared ursuant to the provisions of the California Environmental Quality Act, Division 13 (commencing with ection 21000) of the Public Resources Code, and a decision to grant a permit, license, or similar uthorization for any facility or project is based upon a consideration of such significant environmental effects to such natural resources, and the facility or project was otherwise operating within the erms of its permit, license, or similar authorization at the time of release.

(c) Notwithstanding the provisions of Section 25355, the Governor, or the authorized representative f the state, shall act on behalf of the public as trustee of such natural resources to recover costs xpended pursuant to subdivision (a) or (b).

Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References
States ⇐131.
C.J.S. States §§ 230, 234 to 239.

§ 25353. Remedial action where governmental entity liable for costs; occurrences prior to effective date of this chapter

(a) No money shall be available for remedial action with respect to facilities, owned or operated by a government entity at the time of disposal to the extent that the governmental entity would otherwise be liable for such costs.

(b) No money may be used for the purposes specified in Section 25352 where the injury, degradation, destruction, or loss to natural resources, or the release of a hazardous substance from which such damages to natural resources resulted, has occurred prior to the effective date of this chapter.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25354. Reserve account for emergencies; continuous appropriation; uses; contracts; reports; maximum deposit and appropriation; reversion and deposit of unencumbered balance

(a) There is hereby continuously appropriated from the state account to the department * * *, the sum of one million dollars ($1,000,000) for each fiscal year as a reserve account for emergencies. The department may expend moneys available in the reserve account for the purpose of taking immediate corrective action necessary to remedy or prevent an imminent substantial danger to the public health or the environment caused by the release or threatened release of a hazardous substance.

(b) Notwithstanding any other provision of law, the department may enter into written contracts for corrective action taken or to be taken pursuant to subdivision (a), and may enter into oral contracts, not to exceed two thousand dollars ($2,000) in obligation, when in the judgment of the department immediate corrective action is necessary to remedy or prevent an imminent substantial danger to the public health, domestic livestock, wildlife, or the environment. Such contracts, whether written or oral, may include provisions for the rental of tools or equipment, either with or without operators furnished, and for the furnishing of labor and materials necessary to accomplish the work. Any such contract by the department shall be exempt from approval by the Department of General Services pursuant to the provisions of Section 14780 of the Government Code.

(c) Any disbursement made pursuant to subdivision (a) shall be reported to the Director of Finance within 72 hours of commencement of the corrective action taken.

(d) Once the appropriation made pursuant to subdivision (a) is fully expended, the director shall file a report with the Legislature if it is in session, or if it is not in session, with the Committee on Rules of the Assembly and the Senate as to the moneys expended pursuant to this section. The Legislature may appropriate moneys from the state account, in addition to those appropriated pursuant to subdivision (a), to the department for the purpose of taking corrective action pursuant to subdivision (a).

(e) Except as provided in subdivision (d), in no event shall the amount deposited in the reserve account and appropriated pursuant to this section exceed one million dollars ($1,000,000) in any fiscal year. On June 30 of each year the unencumbered balance of the reserve shall revert to and be deposited in the state account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981. Amended by Stats.1982, c. 327, p. ——, § 120, urgency, eff. June 30, 1982.)

Library References
States ⇐131.
C.J.S. States §§ 230, 234 to 239.

§ 25355. Sites requiring remedial action; coordination of state response actions; responsible party; when director to initiate removal or remedial action

(a) The Governor shall be responsible for the coordination of all state response actions for sites identified in Section 25356 in order to assure the maximum use of available federal funds.

(b) The director shall not initiate removal or remedial action pursuant to this chapter unless the director determines that such actions will not be done properly or in a timely fashion by any responsible party.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Asterisks * * * indicate deletions by amendment

Library References

States ⊂⊃41
C.J.S. States §§ 88 to 90, 130, 131.

§ 25356. Sites for remedial action; criteria for selection and priority ranking; publication and revision; expenditure of funds

(a) Except as provided in Section 25356.5, on or before April 1, 1983, the department shall adopt by regulation the criteria for the selection and for the priority ranking of sites for remedial action under this chapter. The criteria shall take into account the pertinent factors relating to the public health and the environment, which shall include, but are not limited to, potential hazards to public health and environment, the risk of fire or explosion, toxic hazards, and the criteria established pursuant to Section 105(8) of the federal act (42 U.S.C. 9605(8)).[1]

(b) The department shall publish and revise at least annually a priority ranking of sites. The list shall be transmitted to the Legislature on or before January 10 of each year. The development and publication of the priority ranking of sites shall be subject to the regulations adopted under subdivision (a).

(c) The department's development and publication of the priority ranking of sites shall not be subject to the provisions of Chapter 3.5 (commencing with Section 11340) of Part 1 of Division 3 of Title 2 of the Government Code.

(d) Funds appropriated to the department for remedial action shall be expended in conformance with the priority ranking of sites. Funds may be expended on more than one site on the list at any one time.

(Added by Stats.1982, c. 327, p. ——, § 122, urgency, eff. June 30, 1982; Stats.1982, c. 496, p. ——, § 16.7, urgency, eff. July 12, 1982.)

[1] 42 U.S.C.A. § 9605(8).

1982 Legislation.

The addition by Stats.1982, c. 327, § 122 read as follows:

"(a) On or before April 1, 1983, the department shall adopt by regulation the criteria for the selection and for the priority ranking of sites for remedial action under this chapter. The criteria shall take into account pertinent factors relating to the public health and the environment. These factors shall include, but are not limited to, potential hazards to public health and environment, the risk of fire or explosion, toxic hazards, and the criteria established pursuant to Section 105(8) of the federal act (42 U.S.C. 9605(8)).

"(b) The department shall publish and revise at least annually a priority ranking of sites. The list shall be transmitted to the Legislature on or before January 10 of each year. The development and publication of the priority ranking of sites shall be subject to the regulations required by subdivision (a) when they are adopted, but shall not be subject to the provisions of Chapter 3.5 (commencing with Section 11340) of Part 1 of Division 3 of Title 2 of the Government Code.

"(c) Funds appropriated to the department for remedial action shall be expended in conformance with the priority ranking of sites. Funds may be expended on more than one site on the list at any one time."

Former § 25356, added by Stats.1981, c. 756, p. ——, § 2, relating to similar subject matter, was repealed by Stats.1982, c. 327, p. ——, § 121, urgency, eff. June 30, 1982; Stats.1982, c. 496, p. ——, § 16.3, urgency, eff. July 12, 1982.

Derivation: Former § 25356, added by Stats.1981, c. 756, p. ——, § 2.

§ 25356.5. Report to Legislature; sites upon which funds are proposed to be expended; expenditure of funds

On or before July 1, 1982, the department shall provide a report to the Legislature which lists the sites upon which funds are proposed to be expended during the 1982–83 fiscal year and the criteria and rationale upon which the list is based. Funds appropriated to the department from the account for remedial action for fiscal year 1982–83 shall be expended by the department only on those sites listed in this report.

(Added by Stats.1982, c. 327, p. ——, § 123, urgency, eff. June 30, 1982; Stats.1982, c. 496, p. ——, § 16.9, urgency, eff. July 12, 1982.)

1982 Legislation.

The addition of this section by Stats.1982, c. 327, § 123 and c. 496, § 16.9, was identical, except the beginning of the first sentence in c. 327, § 123, read: "Notwithstanding the provisions of Section 25356 on or before July 1, 1982, * * *".

§ 25357. Expenditures exceeding amount in state account

Expenditures from the state account shall not be made in excess of the total amount of money in the state account at any one time. Expenditures in excess of such amount may be made only when additional money is collected or otherwise added to the state account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25358. Federal funds; cooperative agreements

The state shall actively seek to obtain all federal funds to which it is entitled under the federal act and shall take all actions necessary to enter into contractual or cooperative agreements under Sections 104(c)(3) and 104(d)(1) of the federal act (42 U.S.C. 9604(c)(3) and 42 U.S.C. 9604(d)(1)).

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

ARTICLE 6. RECOVERY ACTIONS

Sec.
25360. Amounts to be recovered; deposit in state account.
25361. Parties; state account to be a party.
25362. Parties; joinder of persons liable.
25363. Finding of liability; apportionment of amounts to be recovered; portion payable by state account.
25364. Agreements ineffective to transfer liability.
25365. Judgment not to bar future action.
25366. Construction of chapter.

Article 6 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

Cross References

Subrogation by state to rights of claimant upon payment from compensation account, attorney general to commence action pursuant to this article, see § 25380.

§ 25360. Amounts to be recovered; deposit in state account

Any costs incurred and payable from the state account shall be recovered by the department from the liable person or persons. In addition, such person or persons shall be liable to the department for administrative costs in an amount equal to 10 percent of the reasonable cost actually incurred, or five hundred dollars ($500), whichever is greater. The amount of cost determined pursuant to this section shall be recoverable in a civil action. Nothing in this section shall deprive a party of any defense he or she may have. Moneys recovered by the department pursuant to this section shall be deposited in the state account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Chapter not to affect a person's obligations or liability under other law, including common law, except for this section, see § 25366.

Recoveries pursuant to this section to be deposited in hazardous substance account, see § 25330.

§ 25361. Parties; state account to be a party

The state account shall be a party in any action for recovery of costs or expenditures under this chapter.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Chapter not to affect a person's obligations or liability under other law, including common law, except for this section, see § 25366.

§ 25362. Parties; joinder of persons liable

Upon motion and sufficient showing by any party, the court shall join to the action any person who may be liable for costs or expenditures of the type recoverable under this chapter.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Chapter not to affect a person's obligations or liability under other law, including common law, except for this section, see § 25366.

§ 25363. Finding of liability; apportionment of amounts to be recovered; portion payable by state account

(a) Any party found liable for any costs or expenditures recoverable under this chapter who establishes by a preponderance of the evidence that only a portion of such costs or expenditures are attributable to his or her actions, shall be required to pay only for such portion.

(b) If the trier of fact finds the evidence insufficient to establish each party's portion of costs or expenditures under subdivision (a), the court shall apportion such costs or expenditures, to the extent practicable, according to equitable principles, among the defendants.

(c) The state account shall pay any portion of the judgment in excess of the aggregate amount of costs or expenditures apportioned under subdivisions (a) and (b).

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Chapter not to affect a person's obligations or liability under other law, including common law, except for this section, see § 25366.

§ 25364. Agreements ineffective to transfer liability

No indemnification, hold harmless, conveyance, or similar agreement shall be effective to transfer any liability for cost or expenditures recoverable under this chapter. This section shall not bar any agreement to insure, hold harmless, or indemnify a party to such agreement for any costs or expenditures under this chapter.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25365. Judgment not to bar future action

The entry of judgment against any party to the action shall not be deemed to bar any future action by the state account against any person who is later discovered to be potentialy liable for costs and expenditures paid by the state account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25366. Construction of chapter

(a) This chapter shall not be construed as imposing any new liability associated with acts that occurred on or before January 1, 1982, if the acts were not in violation of existing state and federal laws, or regulations, at the time they occurred.

(b) Nothing in this chapter shall be construed as authorizing recovery for response costs or damages resulting from any release authorized or permitted pursuant to state law or a federally permitted release.

(c) Except as provided in Sections 25360, 25361, 25362, and 25363, nothing in this chapter shall affect or modify in any way the obligations or liability of any person under any other provision of state or federal law, including common law, for damages, injury, or loss resulting from a release of any hazardous substance or for removal or remedial action or the costs of removal or remedial action of such hazardous substance.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

ARTICLE 7. COMPENSATION

Sec.
25370. Board.
25371. Establishment of hazardous substance compensation account.
25372. Application for compensation; conditions.
25373. Claims; forms and procedures; minimum requirements; giving false information; penalty.
25374. Decisions; time; final agency action for purposes of judicial review.
25375. Compensable losses; exclusion.

Sec.
25376. Limitations.
25377. Claim against board not condition precedent to other remedy.
25378. Compensation pursuant to this arti [illegible] to preclude indemnification or reimbursement from other sources and vice versa.
25379. Compensation, decision, or settlement; admissibility as evidence.
25380. Subrogation by state.
25381. Rules and regulations; payment of claims; appropriations.
25382. Administrative expenses.

Article 7 was added by Stats.1981, c. 756, p. —, § 2, urgency, eff. Sept. 25, 1981

§ 25370. Board

"Board," as used in this article, means the State Board of Control.

(Added by Stats.1981, c. 756, p. —, § 2, urgency, eff. Sept. 25, 1981.)

§ 25371. Establishment of hazardous substance compensation account

There is hereby established within the state account, a Hazardous Substance Compensation Account.

(Added by Stats.1981, c. 756, p. —, § 2, urgency, eff. Sept. 25, 1981.)

Library References

States ⟹127.
C.J.S. States § 228.

§ 25372. Application for compensation; conditions

Any person may apply to the board, pursuant to Section 25373, for compensation of a loss caused by the release, in California, of a hazardous substance if any of the following conditions are met:

(a) The source of the release of the hazardous substance, or the identity of the party liable for damages in connection therewith, or responsible for the costs of removal of the hazardous substance, is unknown or cannot, with reasonable diligence, be determined.

(b) The loss was not compensable pursuant to law, including Chapter 6.5 (commencing with Sectio 25100), because there is no liable party or the judgment could not be satisfied, in whole or part against the party determined to be liable for the release of the hazardous substance.

(Added by Stats.1981, c. 756, p. —, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Authority to revise rules and regulations to determine the claimant has exercised reasonable diligence under this section, see § 25381.

Library References

Health and Environment ⟹25.5(1).
C.J.S. Health and Environment §§ 61 et seq., 91 seq., 106 et seq., 115 et seq., 125 et seq., 133 et se

§ 25373. Claims; forms and procedures; minimum requirements; giving false information; pena ty

The board shall prescribe appropriate forms and procedures for claims filed pursuant to this artic which shall include, as a minimum, all of the following:

(a) A provision requiring the claimant to make a sworn verification of the claim to the best of his her knowledge.

(b) A full description, supported by appropriate evidence from government agencies of the rele of the hazardous substance claimed to be the cause of the physical injury or illness or loss of incor

(c) Certification by the claimant of dates and places of residence for the five years preceding date of the claim.

(d) Certification of the medical history of the claimant for the five years preceding the date of claim, along with certification of the alleged physical injury or illness and expenses for such physi injury or illness. Such certification shall be made by hospitals, physicians, or other qualified med authorities.

(e) The claimant's income as reported on the claimant's federal income tax return for the preceding three years in order to compute lost wages or income.

(f) Any person who knowingly gives, or causes to be given, any false information as a part of any such claim shall be guilty of a misdemeanor and shall, upon conviction, be fined up to five thousand dollars ($5,000), or imprisoned for not more than one year, or both.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References

Authority to revise rules and regulations to determine that claimant has exercised reasonable diligence under this section, see § 25381.

§ 25374. Decisions; time; final agency action for purposes of judicial review

All decisions rendered by the board shall be in writing, with notification to all appropriate parties, and shall be rendered within 90 days of submission of a claim to the board unless all the parties to the claim agree in writing to an extension of time. Such decision shall be considered a final agency action for the purposes of judicial review of such decision by any party to the proceedings resulting in such decision.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25375. Compensable losses; exclusion

The following losses shall be compensable pursuant to this article, provided that the board has found that the claimant has established, by the weight of the evidence, that such losses were proximately caused by the designated release:

(a) One hundred percent of uninsured, out-of-pocket medical expenses, for up to three years from the onset of treatment.

(b) Eighty percent of any uninsured, actual lost wages, or business income in lieu of wages, caused by injury to the claimant or the claimant's property, not to exceed fifteen thousand dollars ($15,000) per year for three years.

No money shall be used for the payment of any claim authorized by this chapter, where such claim is the result of long-term exposure to ambient concentrations of air pollutants.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25376. Limitations

No claim may be presented to the board pursuant to this article later than three years from the date of discovery of the loss or from January 1, 1982, whichever is later.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25377. Claim against board not condition precedent to other remedy

Nothing in this article shall require, or be deemed to require, pursuit of any claim against the board as a condition precedent to any other remedy.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25378. Compensation pursuant to this article to preclude indemnification or reimbursement from other sources and vice versa

Compensation of any loss pursuant to this article shall preclude indemnification or reimbursement from any other source for the identical loss, and indemnification or reimbursement from any other source shall preclude compensation pursuant to this article.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

§ 25379. Compensation, decision, or settlement; admissibility as evidence

No compensation or decision under this chapter shall be admissible as evidence of any issue of fact or law in any proceeding brought under any other section of this division or under any other provision of law, including common law. In addition, no settlement reached pursuant to this chapter shall be admissible as evidence in any such proceeding.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Underline indicates changes or additions by amendment

§ 25380. Subrogation by state

Compensation of any loss pursuant to this article shall be subject to the state's acquiring, by subrogation, all rights of the claimant to recover such loss from the party determined to be liable therefor. Upon the request of the board, the Attorney General shall commence an action to recover any amount paid in compensation for any loss pursuant to this article against any party who is liable to the claimant for any loss compensable pursuant to this article in accordance with the procedures set forth in Sections 25360 to 25364, inclusive. Moneys recovered pursuant to this section shall be deposited in the state account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Cross References
Recoveries pursuant to this section to be deposited in hazardous substance account, see § 25330.

§ 25381. Rules and regulations; payment of claims; appropriations

The board shall adopt, and revise when appropriate, all rules and regulations necessary to implement the provisions of this article, including methods that provide for establishing that a claimant has exercised reasonable diligence in satisfying the conditions specified in Sections 25372 and 25373 and regulations that specify the proof necessary to establish a loss compensable pursuant to this article.

(a) Claims approved by the board under the provisions of this article shall be paid from the Hazardous Substance Compensation Account.

(b) The Legislature may, commencing July 1, 1982, appropriate up to two million dollars ($2,000,000) annually from the state account to the Hazardous Substance Compensation Account to be used as a revolving fund by the board for the payment of awards pursuant to this article. However, the total amount in the Hazardous Substance Compensation Account shall not exceed two million dollars ($2,000,000) in any fiscal year.

(c) Claims against or presented to the board shall not be paid in excess of the amount of money available in the Hazardous Substance Compensation Account. Such claims shall be paid only when additional money is collected, appropriated, or otherwise added to that account.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

Library References
Health and Environment ⇐25.5(9).

C.J.S. Health and Environment §§ 65, 66, 103, 107, 140 et seq.

§ 25382. Administrative expenses

The board may expend from the Hazardous Substance Compensation Account such sums of money as are reasonably necessary to administer and carry out the provisions of this article.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

ARTICLE 8. MISCELLANEOUS

Sec.
25395. Duration of chapter; duration of hazardous substance tax.

Article 8 was added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.

§ 25395. Duration of chapter; duration of hazardous substance tax

This chapter and the tax imposed by Section 25345 shall remain in effect only until July 1, 1991, an as of such date is repealed, unless a later enacted statute, which is chaptered before July 1, 1991 deletes or extends such date.

(Added by Stats.1981, c. 756, p. ——, § 2, urgency, eff. Sept. 25, 1981.)

A BILL

TO AMEND CHAPTER 56 OF TITLE 44, CODE OF LAWS OF SOUTH CAROLINA, 1976, AS AMENDED, RELATING TO HAZARDOUS WASTE MANAGEMENT, SO AS TO DELETE THE EXCLUSION FOR SOURCE, SPECIAL NUCLEAR, OR BY-PRODUCT MATERIAL AS DEFINED BY THE ATOMIC ENERGY ACT OF 1954 AND TO AMEND THE USE OF THE HAZARDOUS WASTE CONTINGENCY FUND TO INCLUDE REMEDY FOR PROBLEMS AT UNCONTROLLED HAZARDOUS WASTE SITES.

Be it enacted by the General Assembly of South Carolina:

SECTION 1: Subsection (6) of Section 44-56-20 of the 1976 Code is amended to read as follows:

"(6) "Hazardous waste" means any waste, or combination of wastes, of a solid, liquid, contained gaseous, or semisolid form which because of its quantity, concentration, or physical, chemical, or infectious characteristics may in the judgment of the Department:

a. cause, or significantly contribute to an increase in mortality or an increase in serious irreversible, or incapacitating reversible illness; or

b. pose a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported, or disposed of, or otherwise managed. Such wastes may include, but are not limited to, those which are toxic, corrosive, flammable, irritants, strong sensitizers, persistent in nature, assimilated or concentrated in tissue, or which generate pressure through decomposition, heat or other means. The term does not include solid or dissolved materials in domestic sewage, or solid dissolved materials in irrigation return flows, or industrial discharges which are point sources subject to NPDES permits under the Federal Water Pollution Control Act or the Pollution Control Act of South Carolina or materials covered under Article 2 of Chapter 7 of Title 13 of the 1976 Code as amended."

SECTION 2: Section 44-56-20 of the 1976 Code is amended by adding the following new subsection:

"(13) "Uncontrolled hazardous waste site" means any site where hazardous wastes or other hazardous substances have been released, abandoned, or otherwise improperly managed so that governmental response action is deemed necessary to remedy actual or potential damages to public health or welfare or the environment.

For the purposes of this item the term Hazardous Waste does not include petroleum, including crude oil or fraction thereof; natural gas; natural gas liquids; liquified natural gas; synthetic gas usable for fuel; or mixtures of natural gas and such synthetic gas."

SECTION 3: Section 44-56-60 of the 1976 Code is amended to read as follows:

"Section 44-56-60. Requirements for treatment, storage and disposal facilities.

A. No person shall construct, substantially alter, or operate any hazardous waste treatment, storage or disposal facility or site, nor shall any person store, treat or dispose of any hazardous waste without first obtaining a permit from the department for such facility, site or activity.

B. Any person who:

1. Owns or operates a facility required to have a permit under this section which facility is in existence on the effective date of this chapter; and

2. Has complied with the requirements of Section 44-56-120; and

3. Has made an application for a permit under this section;

shall be deemed to have been issued such permit until such time as final administrative disposition of each application is made by the department, unless final administrative disposition of each application has not been made because of the failure of the applicant to furnish information reasonably required or requested in order to process the application.

C. Before issuance of a permit, the Department shall require:

1. Evidence of liability insurance for sudden and nonsudden accidental occurrences in such amount as the Department may determine necessary for the protection of the public health and safety of the environment; and.

2. Evidence of financial assurance in such form and amount as the Department may determine to be necessary to ensure that, upon abandonment, cessation or interruption of the operation of a facility or site, all appropriate measures are taken to prevent present and future damages to the public health and safety and to the environment.

D. No person who owns or operates a treatment, storage or disposal facility shall accept a hazardous waste which is generated in another state and is banned or prohibited in any manner by any statute, regulation or administrative decision of that state. Written documentation demonstrating compliance with this requirement shall be submitted to the Department for all hazardous wastes imported into this state prior to their transport.

SECTION 4: Section 44-56-90 of the 1976 Code is amended to read as follows:

"Section 44-56-90. Right of Entry.

A. For the purpose of enforcing this chapter or any regulations authorized pursuant thereto, any authorized representative or employee of the department may, upon presentation of appropriate credentials, at any reasonable time:

1. Enter any place where hazardous wastes are generated, stored, treated or disposed of;

2. Inspect and copy any records, reports, information, or test results relating to the purpose of this chapter; and

3. Inspect and obtain samples from any person of any wastes including samples from any vehicles in which wastes are being transported, as well as samples of any containers or labels. The Department shall provide a sample of equal volume or weight to the owner, operator or agent in charge upon request. The Department shall also provide the owner, operator, or agent in charge a copy of the results of any analyses of such samples.

B. For the purpose of implementing necessary governmental response actions as provided in Section 44-56-180, the Department or its authorized representative may, at any time, enter the premises of any publicly or privately owned property which it has determined to be an uncontrolled hazardous waste site. The owner or operator of such site shall cooperate fully with the department when such governmental response actions are taken."

SECTION 5: Section 44-56-160 of the 1976 Code, as amended by Act No. 517 Part II of 1980, is further amended to read as follows:

"Section 44-56-160. Hazardous Waste Contingency Fund.

The Department of Health and Environmental Control is hereby authorized and directed to establish a Hazardous Waste Contingency Fund to ensure the availability of funds for contingencies arising from permitted hazardous waste landfills and to defray the costs of governmental response actions at uncontrolled hazardous waste sites. The Contingency Fund shall be financed through the imposition of fees provided in Section

44-56-170 and such annual appropriations as may be provided by the General Assembly. Provided, that an amount equal to $1.50 per ton for wastes reported under Section 44-56-170(A) and $2.00 per ton for wastes reported under Section 44-56-170(C) shall be held separate and distinct with the Fund for the purpose of mitigating any contingencies arising from the operation of permitted land disposal facilities in this State. Any interest accruing from the management of the funds held pursuant to this section shall be credited to the Contingency Fund."

SECTION 6: Section 44-56-170 of the 1976 Code, as amended by Act No. 517 Part II of 1980, is further amended to read as follows:

"Section 44-56-170. Reporting requirements and payment of fees into the Hazardous Waste Contingency Fund:

A. There is hereby imposed a fee of $5.00 per ton of hazardous wastes which are generated and disposed of in this state by landfilling or other means of land disposal.

B. Each generator shall, no later than thirty (30) days after the end of each calendar quarter, submit a report to the Department containing the following information:

1. the types and quantities of hazardous wastes generated;

2. the types and quantities of such wastes stored, treated and disposed of by landfilling or other means of land disposal; and

3. a check made payable to the department for the amount of fee imposed on such wastes by the provisions of paragraph A and D.

C. Each owner/operator of a hazardous waste land disposal facility shall submit a similar report to the one described in B above for all hazardous wastes generated outside of the State and received at the facility during the quarter except that the fee imposed shall be the same as that fee charged on out of state waste in the state from which the hazardous waste originated but no less than $7.50 per ton."

D. Each owner or operator of a facility permitted under the provision of Section 44-56-60 shall no later than 30 days of the end of each calendar quarter, submit a report containing types and quantities of wastes stored and a check made payable to the Department in an amount equal to $1.00 per ton for each ton of hazardous wastes stored at the end of the reporting period.

SECTION 7: Section 44-56-180 of the 1976 Code, as amended by Act No. 517 Part II of 1980, is further amended to read as follows:

"Section 44-56-180. Administration of Hazardous Waste Contingency Fund.

A. In determining the use of the fund for a particular governmental response action, the Department shall consider the relative risk of danger to public health or welfare or the environment and the hazard potential of the substances involved including potential for fire, explosions, release of harmful air contaminants, direct human contact, contamination of surface water or groundwater including those used for drinking water supplies, and damages to sensitive ecosystems. In taking a Governmental response action and coincident to the use of the fund, the Department shall initiate the appropriate administrative action to exhaust any applicable liability insurance or other financial assurance mechanisms which have been provided by the responsible party as required pursuant to Section 44-56-60 including where appropriate funds available through P.L.96-510. The Department shall annually make a report to the General Assembly on the activities and response actions tha have been carried out under the auspices of the Contingency Fund..

B. When the amount of monies collected and paid into the fund reaches in the aggregate 7.5 million dollars, the Department shall provide a report to the presiding officers of both Houses of the General Assembly outlining the accomplishments of cleanup activities at uncontrolled hazardous waste sites and the need to continue or discontinue the collection of fees for such purposes. The General Assembly shall, within ninety (90) days of receipt of the report by both presiding officers, evaluate the report and take action to continue the collection of fees or take whatever action deemed necessary for this purpose. Provided, however, that if the specified amount is collected and the General Assembly is not in session the collection of fees shall continue until the report is presented at the next convening session.

SECTION 8: Section 44-56-190 of the 1976 Code, as amended by Act No. 517 Part II of 1980, is further amended to read as follows:

"Section 44-56-190. Hazardous Waste Contingency Fund; Inconsistent regulations to be revised.

The Department is directed to revise and amend the necessary provisions of R.61-79 (DHEC) which are contrary or inconsistent with the provisions of this Act."

Robert M. Adams
Senior Vice President

January 6, 1984

3M

Rep. Elliott Levitas
Chairman, Subcommittee on
Investigations and Oversight
Public Works and Transportation Committee
B-376 Rayburn Building
Washington, D.C. 20515

Dear Rep. Levitas:

This is a response to your invitation to comment as part of the November 8 and 9, 1983, hearing of the Investigations and Oversight Subcommittee concerning review of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980. We appreciate your courtesy in keeping the hearing record open and welcome the opportunity to present 3M comments on victim compensation to the Subcommittee.

In recent years, 3M has played an active role in the development of environmental policy in Minnesota, particularly in waste management; and our technical experts have been invited to participate in numerous state and federal government sponsored waste management programs. At these programs, there always seems to be considerable interest in the 3M Pollution Prevention Pays program which has been widely recognized throughout the world as an excellent method of eliminating or reducing the amount of hazardous wastes and other pollutants generated by industry. In fact, the 3P program is referenced in the 1982 annual report of the President's Council on Environmental Quality.

In the state legislative area, 3M strongly supported and lobbied for enactment of the Minnesota Waste Management Act of 1980. Among other things, this Act set up a statewide public participation process for determining the location of a land disposal site for hazardous wastes; it is one of the most comprehensive public participation programs in any state for any reason. 3M also strongly supported and lobbied for the waste site cleanup and matching fund provisions of the Minnesota Environmental Response and Liability Act of 1982 (MERLA).

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The record will show that 3M has taken a strong public stand in support of federal and state regulatory efforts to clean up existing waste sites. Industry must continue to bear its part of this effort if the task is to be successful. Similarly, however, there must be a recognition by governmental leaders and the general public that the problem being addressed is societal in nature and cannot fairly be thrust only upon industry, much less a specific segment of industry.

It is this aspect of addressing hazardous waste problems that is one of the most troublesome to 3M. While there may be some short-term political expediency in pointing to industry as the scapegoat to which is directed public hazardous waste concerns, such a policy serves only to create adversarial barriers rather than foster coordinated commitment of the public and private sectors that is necessary to fully address and resolve hazardous waste problems.

It is in this context that 3M has found it necessary to take a public stand against ill-conceived measures that conveniently, but erroneously, focus liability upon industry for society's waste problems. An outstanding example of such a measure is the Minnesota Environmental Response and Liability Act (MERLA) as it applies to personal injury and economic loss liabilities and upon which the Subcommittee has previously received testimony. Although it serves no purpose here to debate the content of this prior testimony, it is important to note that the rather extraordinary provisions of MERLA - provisions that ignore traditional principles of fundamental fairness, if not due process, in order to ensure industry vulnerability to litigation - were not predicated on a demonstrated need for such provisions. Rather, they were promulgated upon an unfortunate mixture of political expediency, anti-business sentiment and the interests of those who stand to gain in litigation representing claimants.

Consequently, there was no desire to address the very real issue of need for such extraordinary measures. But major legislation must be based upon demonstrable facts, not anecdotal scenarios that are neither real world nor demonstrable.

Clearly, this principle should apply to the Subcommittee's consideration of victim compensation legislation. Most certainly, 3M gives support to a program that compensates actual victims where the victims are otherwise without relief. But such legislation should not become reality until there is a comprehensive and factually based demonstration that there are significant health problems from hazardous wastes that current laws and procedures do not address. It simply is not sufficient to rely upon a general public or political perception that there are significant human health problems allegedly caused by exposure to hazardous wastes and that the present legal system does not adequately provide redress for such problems.

At such time as victim compensation legislation is justified in the above-referenced manner, 3M urges that it contain the following provisions:

1. That claimants be required to choose between the administrative remedy of a victim compensation scheme and a tort claim. Compensation under victim compensation legislation must be an exclusive remedy.

2. A pre-emption of other laws that will allow for a consistent and equitable national program for all claimants no matter where situated.

3. A fund whose size is predicated on a demonstrable scope of the problem.

4. A fund that is financed in a manner that correctly recognizes the problem as societal in nature and thus embraces a cost sharing by the public and private sectors with the private sector share being derived from all hazardous waste generators in a manner that reflects not only volume but also degree of toxicity and manner of disposal.

5. Application of the legislation should be solely for disposal site exposures and not occupational exposures.

6. Standardize the statute of limitation period applicable to seeking relief under the legislation. Whatever the period adopted, it should run from the date of discovery of the injury.

At 3M we are mindful of the great difficulty facing the Subcommittee in coming to grips with the victim compensation issue. Of necessity I have here attempted to offer our general comments on the issue. As your consideration progresses, we stand ready to assist you in any manner that may be appropriate.

Thank you again for your patience and the opportunity to present our views on this most important matter. May you have a prosperous New Year.

Sincerely,

c: Sante J. Esposito